



[Abstract:0059]

THE IMPORTANCE OF A DETAILED NEUROLOGICAL EXAMINATION

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A 55-year-old woman with a personal history of essential arterial hypertension, morbid obesity, and recent admission to Mental Health for major depression with psychotic symptoms (Cotard syndrome). She attended the emergency room due to lower limb weakness with inability to walk. Her family said that the weakness was intentional. In previous days, flu-like symptoms with episodes of daily low-grade fever. To the physical examination, oriented in the three spheres with little collaboration. Peripheral oxygen saturation 97% FiO₂ 21. No ophthalmoparesis. No cranial nerve alterations. Not dysarthria. Muscular balance 0/5 in lower limbs and 3/5 in upper limbs. Preserved sensitivity. Deep tendon reflexes absent in lower limbs and decreased in upper limbs bilaterally. Bilateral cutaneous-plantar flexor reflexes. Cardiac auscultation with rhythmic tones without murmurs. Respiratory auscultation with preserved vesicular murmur without superimposed noises. Lower limbs without oedema or signs of venous thrombosis. A cranial CT scan was performed, without alterations, and a lumbar puncture showed albuminocytological dissociation. She was admitted to the ward to start treatment and complete the study. An electromyographic study compatible with acute symmetrical axonal motor polyneuropathy of moderate-severe intensity was performed. Study of porphyria, heavy metals, cryoglobulins, syphilis and antiganglioside antibodies were negative. After confirming the diagnosis of axonal variant Guillain-Barré syndrome (AMAN), the administration of intravenous human immunoglobulins (IVIG) was initiated at a dose of 0.4 g/kg for 5 days with progressive improvement in the following weeks. After a month and a half, she began to walk with help.

Keywords: polyneuropathy, albuminocytological dissociation, Guillain-Barré syndrome

[Abstract:0151]

NOT EVERY STROKES ARE HEADACHES

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Cerebral venous thrombosis is less common than other types of stroke and is characterized by great clinical variability.

The most common form of presentation is a sudden onset headache or other signs of isolated intracranial hypertension. It may also present with focal neurological deficits, convulsions, or encephalopathy. The following case represents this diagnostic challenge.

An 80-year-old man with a history of atrial fibrillation and diverticular disease was taken to the emergency department after a sudden change in speech, noted by his relatives, without any other neurological deficit. Once in a hospital, the patient was monitored, hypertensive (BP 150/80 mmHg), afebrile, normoglycemic and aphasia was noted. The brain-computed tomography revealed no haemorrhagic lesion and due to a favourable time window and absence of contraindications, fibrinolysis with alteplase was performed.

The procedure was interrupted due to a worsening state of consciousness and a generalized tonic clonic crisis. A CT angiography was performed with an image suggestive of either occupying lesion or haemorrhagic transformation. Faced with new findings, the patient was transferred to our hospital centre and started thrombolysis reversal.

Once transferred, he maintained aphasia and due to the risk of hydrocephalus, he was admitted to the stroke unit for etiological study and monitoring. A CT was repeated at 24 hours with clear reabsorption of the haemorrhage and spontaneous hyperdensity in the left sigmoid sinus, suggestive of cerebral venous thrombosis. A Venous-CT was requested which confirmed the diagnostic suspicion. Therapeutic anticoagulation with LMWH was started and the patient improved.

Keywords: stroke, thrombosis, fibrinolysis, anticoagulation



Figure 1. Complementary Diagnosis Exams - Brain CT.
Extensive intraventricular and left temporal parenchymal hematic content.



Figure 2. Complementary Diagnosis Exams - Brain CT (2).
Spontaneous hyperdensity in the sigmoid sinus on the left, suggesting possible venous thrombosis with associated hemorrhagic infarction (red circle).

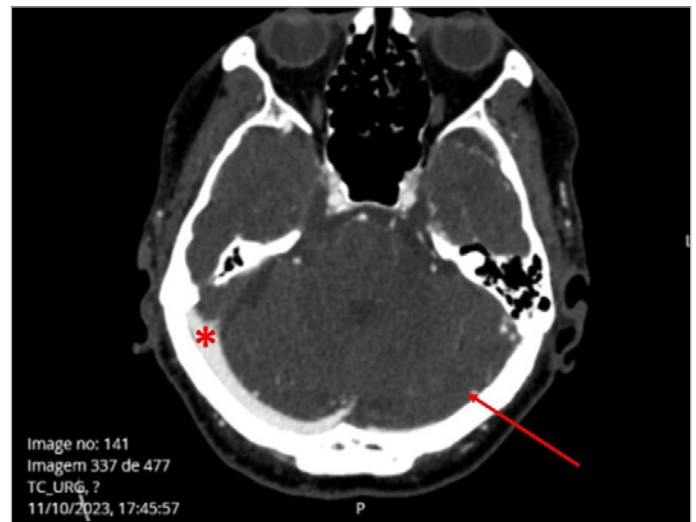


Figure 3. Complementary Diagnosis Exams - Venous - Brain CT
Thrombosis of the left sigmoid sinus (red arrow). Normal filling of the remaining venous sinuses (red star showing patency of the right sigmoid sinus).

[Abstract:0164]

ACUTE MOTOR AXONAL NEUROPATHY (AMAN), A CASE REPORT

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Case Description: Female, 79-year-old, who consulted for progressive onset weakness of the four limbs, dysphagia and hypophonia on the last three days. She associated dyspnoeic sensation and denied dysautonomia, sensory or diplopia disorders. Neurological examination revealed hypophonia, loss of four extremities and neck muscles strength with normal osteotendinous reflexes and without sensory deficit or pyramidalism.

Diagnostic Pathways: Considering the generalized motor deficit with pseudobulbar involvement, we established differential diagnosis between acute inflammatory polyneuropathies and motor plate diseases such as myasthenia gravis.

Cranial and cervical spine magnetic resonance imaging was performed ruling out the existence structural damage; cerebrospinal fluid analysis resulted normal without albuminocytological dissociation and an electromyogram showed a generalized decrease in motor amplitude potentials with normal repetitive stimulation, compatible pattern with motor axonal polyneuropathy. Finally, the autoimmunity study resulted positive for anti-ganglioside antibodies GQ1b, GT1a and GT1b with negative results for anti-acetylcholine receptor antibodies, anti-Musk and botulinum toxin.

In relation to the clinical presentation and the results obtained in the different complementary tests performed, the diagnostic conclusion was the presence of an acute motor axonal polyneuropathy (AMAN). Subsequently questioned about

possible precipitants of the clinical picture, the patient reported diarrheal syndrome two weeks earlier, without microbiological isolation.

Discussion: AMAN is a rare variant of Guillain Barré syndrome characterized by pure motor symptoms without sensory involvement, being the triggering factor in most cases an infectious process. Its course is usually more severe than other variants of acute inflammatory demyelinating polyradiculoneuropathy and immunoglobulins or plasmapheresis may be used for treatment.

Keywords: AMAN, Guillain-Barré, polyradiculoneuropathy

[Abstract:0217]

VALPROIC ACID-INDUCED HYPERAMMONAEMIA MIMICKING HYPOXIC-ISCHEMIC ENCEPHALOPATHY ON NEUROIMAGING. AN INTERESTING CASE OF PROLONGED ENCEPHALOPATHY SECONDARY TO MULTIDRUG OVERDOSE

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Introduction: Acute valproic acid poisoning may provoke hyperammonaemia and result in hyperammonaemic encephalopathy. It can manifest with severe neurological symptoms and life-threatening complications such as cerebral oedema and brain herniation, and hence, proper management is warranted. This case follows the treatment journey of a well-premorbid young lady managed for multidrug overdose with hyperammonaemia in a tertiary hospital.

Case Description: An 18-year-old student with a history of anxiety disorder was found drowsy in her hostel room after discovering her suicide post on social media. Empty pill packings of estazolam, quetiapine, valproate and sertraline were found in her room. The patient turned comatose and was intubated for airway protection and was admitted to the intensive care unit. Initial magnetic resonance imaging (MRI) demonstrated diffuse diffusion-weighted (DWI) hyperintensities over basal ganglia and cortical grey reported as hypoxic ischemic encephalopathy changes. The decision was subsequently made to initiate intravenous carnitine and haemodialysis for the uptrending levels of valproate and ammonia, and the development of new-onset cortical myoclonus. The valproate and ammonia levels gradually came down, and she eventually regained full consciousness on day eight of admission. There was no significant neurological sequela, and repeat MRI showed resolution of previous diffuse DWI changes and cortical swelling. Psychiatric consultation and follow-up was subsequently arranged for the patient on the general ward.

Discussion: This case highlights the treatment of valproic acid-induced hyperammonaemia, the limitation of neuroimaging in

differentiating hypoxic changes from hyperammonaemia, and the importance of an early diagnosis as hyperammonaemic encephalopathy is potentially reversible.

Keywords: hyperammonaemia, hypoxic-ischemic, encephalopathy, neuroimaging, valproic acid

[Abstract:0243]

EVOLUTIONARY MODE OF NEUROLOGICAL VASCULO-INFLAMMATORY PHENOTYPE OF DADA 2: ABOUT A CASE AND REVIEW OF THE LITERATURE

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Introduction: Adenosine deaminase 2 deficiency (DADA 2) is characterized by a wide spectrum of clinical manifestations.

The vasculo-inflammatory manifestations are little described and their progression mode is little known.

The objective of our work is to describe the evolutionary mode of neuro-vascular-inflammatory phenotype.

Clinical cases: The first case reported by the parents is patient F born in 1984 and died at the age of 17. In his history, we noted a stroke at the age of 5 years without after-effects associated with paroxysmal inflammatory syndrome diagnosed as Still's disease. Death occurred at the age of 17 following kidney failure.

The 2nd case, aged 25, presented with recurrent fever since the age of 2, treated as Still's disease. at the age of 18, the patient presented headache and intense dizziness. MRI showed vascular-like cerebral demyelinating lesions treated with good progress. Three months later, the patient presented with retrobulbar optic neuritis treated good response and since then he has been in complete remission on low dose of CTC with a follow-up of 7 years. The 3rd case is 17 years old, diagnosed with DADA 2 since the age of 10, presented headache and dizziness, with normal neurological exploration.

Conclusions: The neurological vasculo-inflammatory phenotype evolves differently in DADA 2 patients from the same family. The onset of ischemic neurological damage early in life is associated with a poor prognosis, unlike late inflammatory neurological damage around adolescence which tends to have a favourable outcome.

Keywords: neurological damage, DADA 2, autoinflammatory disease, stroke, retrobulbar optic neuritis, vascular cerebral demyelinating lesions

[Abstract:0271]

VERTEBROBASILAR ARTERY FENESTRATION-RELATED CEREBRAL INFARCTION WITH ATOPIC DERMATITIS

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Introduction: Vertebrobasilar artery (VBA) fenestration, a rare congenital arterial bifurcation-reconvergence, has been associated with aneurysm and ischemic stroke. Atopic dermatitis (AD) is also associated with stroke. We identified 2 cases of VBA fenestration-related cerebral infarction in young Japanese men with AD.

Case 1: A 38-year-old man with AD developed sudden lightheadedness, left upper quadrant anopia, and right lower limb ataxia (NIHSS 2). MRI/A showed acute infarction in the right cerebellar and medial occipital lobes, and left VBA fenestration on BPAS (Figure 1). Other clinical data were normal. He was treated with dual antiplatelet therapy (DAPT) and heparin anticoagulation (due to recurrence on day 21), with improvement thereafter.

Case 2: A 36-year-old man with AD and dyslipidaemia developed sudden vertigo, right nystagmus, and decreased pure tone hearing (NIHSS 0). Blood pressure was 155/117 mmHg and LDL was elevated. MRI/A showed acute right cerebellar infarction and left VBA fenestration on BPAS (Figure 2) with no other abnormalities. Patient improved on single antiplatelet therapy.

Discussion: VBA fenestration has a frequency of 0.28-5.26% in post-mortem studies and is associated with aneurysm and cerebral infarction. Abnormal shear stress and hemodynamic at bifurcation sites are thought to cause multidirectional endothelial damage, platelet aggregation, thrombogenicity, and plaque instability. Similarly, AD has been associated with thrombogenic platelet activation and decreased fibrinolysis. Our cases suggest a synergistically increased risk of cerebral infarction associated with VBA fenestration and AD-related chronic inflammation.

Conclusions: VBA fenestration should be considered in young patients with AD and cerebral infarction.

Keywords: cerebral infarction, vertebrobasilar artery fenestration, atopic dermatitis

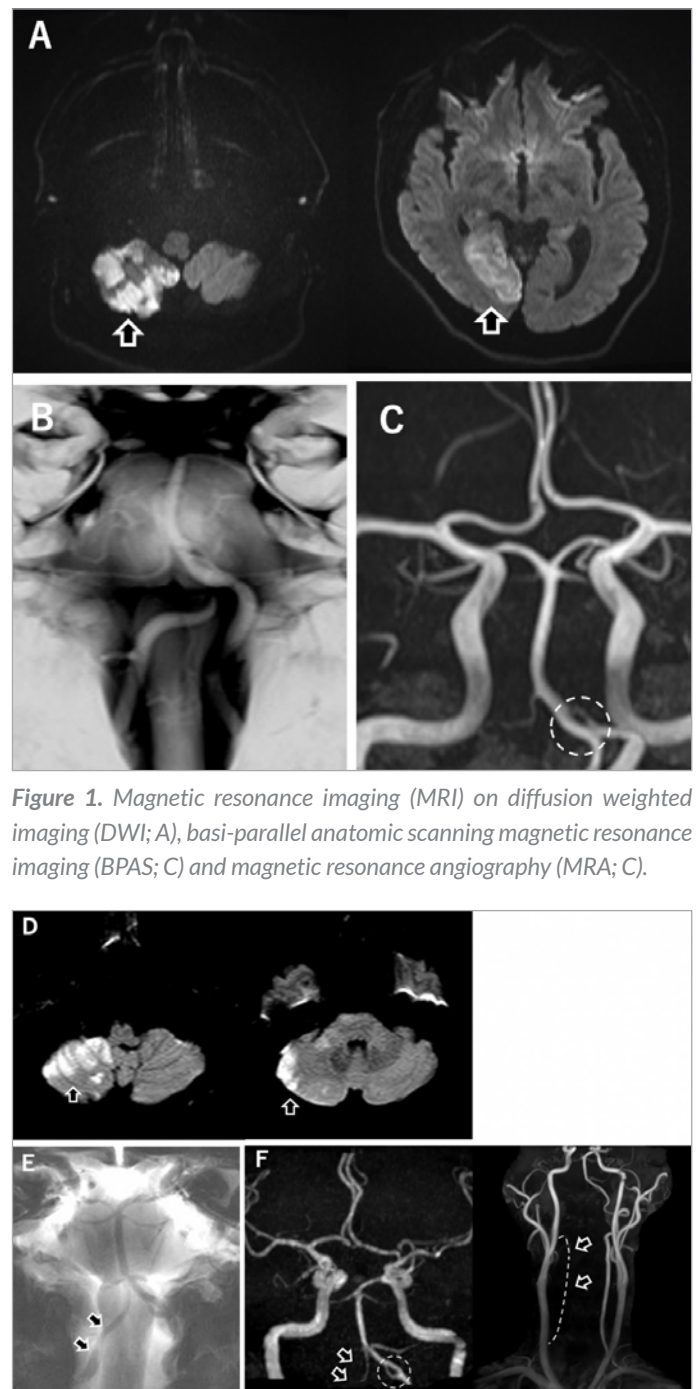


Figure 1. Magnetic resonance imaging (MRI) on diffusion weighted imaging (DWI; A), basi-parallel anatomic scanning magnetic resonance imaging (BPAS; C) and magnetic resonance angiography (MRA; C).

Figure 2. MRI on diffusion weighted imaging (DWI; D), basi-parallel anatomic scanning magnetic resonance imaging (BPAS; E) and magnetic resonance angiography (MRA; F).

[Abstract:0301]

RETROBULBAR OPTIC NEURITIS IN BEHÇET'S DISEASE: A CASE REPORT

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Introduction: Behçet's disease, a rare systemic vasculitis of unknown origin, commonly manifests with ocular involvement, yet retrobulbar optic neuritis remains an under-documented complication. This case report highlights a unique instance of retrobulbar optic neuritis associated with Behçet's disease, emphasizing diagnostic challenges and therapeutic considerations.

Case: Mrs. B.I, a 40-year-old with hypertension and diabetes, presented with a third episode of sudden vision loss and an inflammatory syndrome. Ophthalmological examination revealed bilateral visual acuity decline to 1/10, without uveitis, vasculitis, or diabetic retinopathy. Cerebral angio-MRI showed no anomalies, visual evoked potential suggested retrobulbar optic neuritis, the emergence of other clinical manifestations, such as bipolar aphthosis and inflammatory joint involvement, led to the diagnosis of Behçet's disease. Treatment involved a 3-day 500 mg/day IV bolus of corticosteroids, followed by oral administration at 1 mg/kg/day, along with methotrexate and adalimumab.

Discussion: Retrobulbar optic neuritis in Behçet's disease presents a diagnostic challenge, requiring the exclusion of other causes (multiple sclerosis, granulomatous diseases). It tends to be more severe than in alternative aetiologies, with 80% exhibiting visual acuity below 5/10. While corticosteroids, immunosuppressants, and biologic therapy are effective, delayed diagnosis may result in visual sequelae in 90% of severe cases.

Conclusions: Retrobulbar optic neuritis is a serious manifestation of Behçet's disease. It should be considered in the presence of any decline in visual acuity accompanied by a normal ophthalmological examination. Prompt and appropriate management can enhance visual prognosis. Further studies are needed to better comprehend the underlying mechanisms and to formulate more specific therapeutic strategies.

Keywords: Behçet, retrobulbar optic neuritis, vasculitis



Figure 1. Genital ulcer observed in the vulva of the patient.



Figure 2. Lingual Aphthosis. Aphthous ulcers or canker sores are a small and painful lesions on the surface of the tongue.

[Abstract:0381]

DIAGNOSTIC SIGNIFICANCE AND CLINICAL CHALLENGES OF DIFFUSE AXONAL INJURY: A CASE STUDY IN TRAUMATIC BRAIN INJURY MANAGEMENT

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Diffuse axonal injury (DAI) is a consequential outcome of traumatic brain injury, particularly in cases of severe head trauma. It arises from rapid rotational acceleration and deceleration of the cranium, resulting in extensive shearing of nerve fibers. This process induces both macroscopic and microscopic axonal damage, predominantly at the gray-white matter junction. The

subsequent damage involves axonal misalignment and, less frequently, stretching or shearing, initiating a complex cascade of pathophysiological events such as axonal depolarization, metabolic disturbances, cellular swelling, cytotoxic oedema, and apoptosis.

While the term 'diffuse' implies widespread involvement, DAI is more accurately characterized by lesions consistently located within white matter tracts, such as the corpus callosum. These specific lesions correlate with unfavourable short-term outcomes. DAI manifests a spectrum of clinical manifestations, varying from acute consciousness loss to cognitive deficits and persistent coma. Outcome prediction remains inconclusive, with MRI evidence primarily relevant to short-term in-hospital functional prognosis. Standardized treatment for DAI is currently lacking.

Illustrating these complexities, a case study involves a 23-year-old male patient who was in a high-velocity road traffic accident. Despite initially unremarkable cranial CT scans, subsequent cranial MRI, on the 7th day, revealed grade III DAI with multiple haemorrhagic foci, particularly affecting the corpus callosum and the cerebral hemispheres' gray-white transition zone. The patient exhibited progressive recovery after 12 days, successfully overcoming orotracheal intubation and engaging in communication. This case highlights the clinical challenges and diagnostic significance of DAI in the management of traumatic brain injury.

Keywords: traumatic brain injury, diffuse axonal injury, neurofilament, diffuse brain trauma, traumatic axonal injury

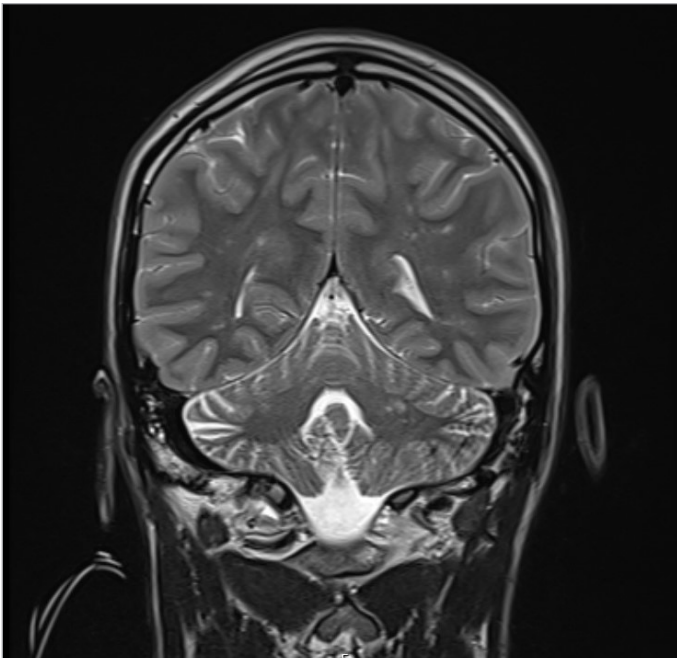


Figure 1. Image of the lesions in the MRI

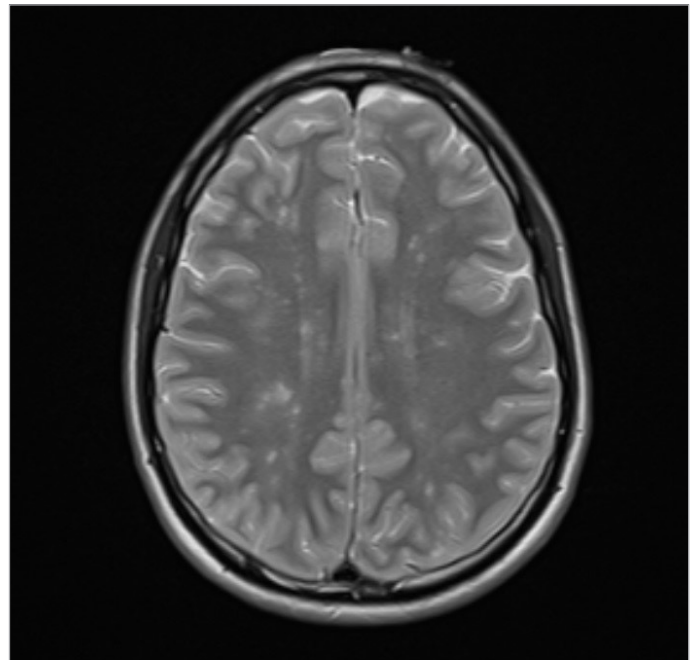


Figure 2. Image of the lesions in the MRI.

[Abstract:0428]

A CASE SERIES OF NEUROINVASIVE DISEASE CAUSED BY WEST NILE VIRUS (WMN): ADAPTING TO EVOLVING REGIONAL AND CLINICAL DEMANDS

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Purpose: The COVID-19 pandemic has highlighted the significance of quickly adjusting to outbreaks of diseases. The diagnostic difficulties posed by the nonspecific symptoms of neuroinvasive WNV are particularly pertinent in regions where it is not endemic or where it is newly emerging.

Methods: We examine the initial three cases of neuroinvasive WNV observed at our hospital during 2023. All patients had encephalitis confirmed through cerebrospinal fluid analysis (CSF). WNV was detected in all patients through serum polymerase chain reaction testing (PCR) on peripheral blood samples.

Findings: Case 1. A middle-aged man with fever, respiratory symptoms, and worsening condition leading to unconsciousness and respiratory failure. This case marked the first diagnosis of West Nile Virus in north-western Greece in 2023.

Case 2. An immunocompromised older man with IgA nephropathy experiencing fever, confusion, and declining kidney function. A delayed diagnosis of WNV was made after ruling out other potential causes related to the patient's health condition.

Case 3. An elderly woman with a history of breast cancer exhibiting prolonged fever lasting ten days and confusion. After excluding specific pathogens for encephalitis through CSF PCR testing, we

considered WNV infection as a possible cause based on current knowledge of regional prevalence.

Conclusions: In this report, we discuss our experience in adapting and responding to the 2023 outbreak of WNV encephalitis in Greece. Notably, Greece has the second-highest number of reported cases of WNV in Europe, following Italy^[1].

Reference:

[1] European Centre for Disease Prevention and Control. Available at: <https://www.ecdc.europa.eu/en/west-nile-fever/surveillance-and-disease-data/disease-data-ecdc>

Keywords: West Nile virus, neuroinvasive disease, outbreak

[Abstract:0476]

NEUROLEPTIC MALIGNANT SYNDROME - A DIAGNOSIS TO CONSIDER

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Male, 57-years-old, long-term hospitalized at the psychiatry ward for depression related with a Cotard's syndrome, treated with typical antipsychotics and electroconvulsive therapy. Weeks after admission, the patient developed an akinetic-rigid tetraparesis. Since he displayed agitation persisting for several days, the dosage of medications such as haloperidol, olanzapine and promethazine was increased. Transferred to the emergency department with fever, agitation, and dyspnoea. On admission he was feverish (39°C), tachycardic, diaphoretic, confused and in mutism, with severe muscle rigidity and dysphagia, and incontinent.

The most likely hypothesis, considering patient's history and physical examination, was an extrapyramidal syndrome with tardive dystonia secondary to neuroleptics, and posterior neuroleptic malignant syndrome (NMS) secondary to the increased dosage of antipsychotics.

Nevertheless, entities such as central nervous system infections (meningitis or encephalitis), malignant catatonia, toxic encephalopathies and nonconvulsive status epilepticus were excluded.

Blood tests revealed leucocytosis, elevated creatine phosphokinase (21977 UI/L), creatinine and transaminases, hypocalcaemia, and metabolic acidosis. Head CT scan and electroencephalogram without pathological findings. Blood cultures were negative. Psychiatric medication was discontinued, and correction of fluid deficits and electrolyte imbalances ensued. Biperiden and baclofen were initiated, and the patient was transferred to the Intensive Care Unit. Electroconvulsive therapy was continued.

This case underscores the complex management of NMS. Prompt recognition of symptoms, cessation of neuroleptic medications

and implementation of supportive care are crucial in improving patient outcomes. Additionally, a multidisciplinary approach, including close monitoring, correction of metabolic abnormalities and considering alternative therapeutic agents, remains pivotal in the management of this severe and potentially life-threatening condition.

Keywords: neuroleptic malignant syndrome, antipsychotics

[Abstract:0610]

VESTIBULAR NEURITIS: A SIMPLE YET CHALLENGING DIAGNOSIS

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Vestibular neuritis (VN) is characterised by peripheral vestibular dysfunction of acute onset. Although the aetiology remains unclear, a viral cause is suspected. The differential diagnosis with a vascular cause such as ischaemia or haemorrhage of the brainstem or cerebellum is critical since these are potentially life-threatening conditions. An acute unilateral vestibulopathy, when placed in a typical context in a healthy young person, can be a simple clinical diagnosis. However, in some cases, the presentation may not be linear making the diagnosis more challenging. We describe the case of a 72-year-old male, with high cardiovascular risk, who presented with a history of sudden vertigo, vomiting and imbalance to the right, and whose examination was not entirely consistent with a peripheral lesion - he had a multidirectional inextinguishable nystagmus and a doubtful head impulse test (HIT). The cranioencephalic computed tomography (CE-TC) performed upon admission to the emergency department and upon reassessment 15 hours later did not show any acute lesions suggestive of a cerebral vascular event. The magnetic resonance imaging (CE-MRI) performed later ruled out the possibility of a vascular event and no signs of inflammation were observed. He was then treated with steroids with rapid clinical improvement. This case report describes the diagnostic investigation and most important features of this often-challenging entity, highlighting the challenge of initial management and diagnosis in the acute setting, especially if physical examination findings are inconsistent and in the absence of imaging findings to support this hypothesis.

Keywords: vestibular neuritis, minor posterior circulation ischaemic stroke, vertigo, nystagmus, case report

[Abstract:0682]

WHAT IS THE RELATION BETWEEN SPIDERMAN AND INTERNAL MEDICINE

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Generally, patients who suffer hallucinations without evidence of cognitive deterioration, are referred to either Psychiatric or Neurologic Departments for evaluation and diagnosis.

We present two cases in which the cause of this pathology was diagnosed in a Department of Internal Medicine after a proper differential diagnosis was performed.

Case 1: A 74 years old male patient, was accompanied by a family member to the Emergency Unit, because he kept seeing children playing with Spiderman dolls in the street.

Case 2: A 79 years old female patient. She was terrified because at night she used to see, how the crucifix above her bed used to talk to her.

Discussion: The differential diagnosis of hallucinations is extensive, because they could be the manifestation of diverse underlying medical, neurologic or psychiatric disorders. Vitamin B12 deficiency include symptoms as cognitive deterioration, changes in mood, psychotic symptoms and confusional states. More specifically, B12 deficiency psychosis could manifest as suspiciousness, persecutory or religious delusions (as cases we presented here). All of them, are clinical presentations which are not very frequent and can be the only initial manifestation.

In our cases a suitable anamnesis, a physical exploration, an imagen testing together with a simple and ordinary blood analysis, showed that a severe deficit of B12 could be the cause of the symptoms of patients. Due to this, it was possible to start a replacement treatment.

Both patients recovered completely.

Conclusions: Vitamin B12 deficiency should be included in the differential diagnosis of patients presenting with atypical psychosis.

Keywords: hallucinations, B12 deficiency, psychiatric symptoms

[Abstract:0700]

ADRENERGIC SPELLS: NOT ONLY ENDOCRINE CAUSES

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Case Description: A 23 year old female patient consulted in emergencies relating several episodes of palpitations, sweating, heat and flushing in the previous month. The patient was obese, with no other medical background. Physical examination revealed high blood pressure tachycardia and hyperglycaemia. No abnormal findings were found in blood test.

Clinical Hypothesis: Adrenergic spells due to pheochromocytoma.

Diagnostic Pathway: Multiple tests were performed in order to rule out endocrine diseases (thyroid function, urine catecholamines, plasma metanephrines, 24h urine cortisoluria, 5-hydroxyindole acetic acid), metabolic diseases (Hoesch test), neoplastic diseases (full body CT scan) and neurologic diseases such as dysautonomia (24h blood pressure Holter, tilting test). Tilting test showed an abnormal response to orthostatism, reaching 170 beats per minute and developing a hypertensive crisis. Therefore, postural orthostatic tachycardia syndrome (POTS) was diagnosed.

Discussion and Learning Points: POTS is a rare disease that can cause paroxysmal spells, so it is important to think about it once that we have ruled out the main causes. It is included in the spectrum of dysautonomic diseases, and it is caused by a misregulation of our physiologic response to orthostatism. There are different subtypes, one of them is hyperadrenergic POTS, which may make us think about endocrine diseases like pheochromocytoma or carcinoid syndrome. The treatment is based on ivabradine, or beta-blockers and it is crucial to rule out other mayor neurologic diseases that can provoke this dysautonomia.

Keywords: dysautonomia, POTS, pheochromocytoma

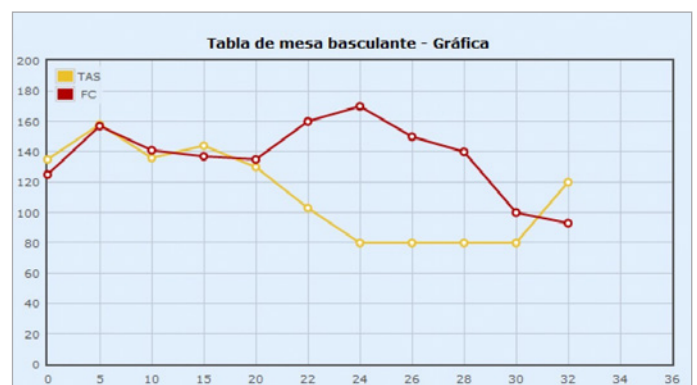


Figure 1. Correlation between heart beat (red) and blood pressure (yellow) with time of standing.



Figure 2. Correlation between heart beat (red) and blood pressure (yellow) with time of standing.

[Abstract:0756]

DIAPHRAGMATIC DYSFUNCTION: A CASE OF MYASTHENIA GRAVIS DIAGNOSIS

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Myasthenia gravis (MG) stands as a captivating enigma within the realm of neuromuscular disorders, characterized by fluctuating muscle weakness and fatigability. A myasthenic crisis is a medical emergency due to respiratory failure related to diaphragm weakness. A 73-year-old woman, without medical history, was admitted to the Emergency Department (ED) complaining of orthopnoea and right eyelid ptosis. Peripheral facial paralysis was assumed and treated with prednisolone with improvement. Due to the persistence of orthopnoea, she returned to the ED and was admitted to the Internal Medicine ward for study.

Analytically without relevant changes. Additionally, the patient started complaining of moderate flaccid dysarthria and fluctuating asymmetry of the palpebral fissures. The diagnosis of MG was considered and a therapeutic trial with pyridostigmine was performed with apparent clinical improvement. Due to the bulbar symptoms, administered intravenous immunoglobulin for 5 days. The differential diagnosis at this point included neuromuscular disease and paraneoplastic process. It was performed computed tomography angiography, cervical soft tissue and magnetic resonance imaging of the brain and cervical spine showing no presence of thymoma or other masses. Nocturnal oximetry in room air without changes and the repetitive electromyography was normal. Collected samples for Anti-acetylcholine receptor antibodies with a positive result and Anti-muscle specific kinase was negative. The patient was discharge referred to a Neurology consultation and under therapy with pyridostigmine and weaning from corticosteroids.

Diaphragm dysfunction is associated with dyspnoea and sleep disturbances. This case underscores the multifaceted nature of MG, emphasizing the need for an understanding of its underlying mechanisms.

Keywords: myasthenia, gravis, dyspnoea, diaphragm

[Abstract:0808]

SARCOIDOSIS AND ISCHEMIC STROKE. ABOUT A CASE

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Purpose: Description of a case and review of ischemic stroke associated with the onset of sarcoidosis.

Materials and Methods: A 38-year-old woman presented with sudden symptoms of weakness of the right limbs, alterations in the understanding and production of language. A NIHSS 2 was observed, and imaging tests showed occlusion of the left M2 middle cerebral artery. Despite low NIHSS, mechanical thrombectomy was performed due to the patient's disabling symptoms.

Results: In an etiological study, a bubble test with transcranial Doppler showed right-left communication with a shower pattern after the Valsalva manoeuvre. Transthoracic and transoesophageal echocardiography without significant findings (no identification of patent foramen ovale). Doppler ultrasound of the lower limbs without findings of deep vein thrombosis. A CT scan of the chest, abdomen and pelvis shows subcarinal adenopathy and multiple non-specific bilateral mediastinal and hilar lymph nodes of size around the upper limit of normal. PET-CT: multiple hiliomediastinal and costodiaphragmatic lymphadenopathy. EBUS of adenopathy was performed, which diagnosed stage I sarcoidosis. A special coagulation study revealed MTHFR homozygosity.

Conclusions: A series of cases of stroke and sarcoidosis showed that stroke as the first symptom of sarcoidosis was 40%, being a rare condition.

In our case, and given the presence of other etiological factors, its causal relationship cannot be concluded. Even so, it should be considered that cerebrovascular events may be the first affection of sarcoidosis, especially given these radiological findings.

Keywords: sarcoidosis, stroke, granulomatous

[Abstract:0825]

THERAPEUTIC AND DIAGNOSTIC CHALLENGES IN MYASTHENIA GRAVIS

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Background: Myasthenia gravis is a rare autoimmune disease that appears most frequently in young women and old men.

Case: We present the case of a 77-year-old woman who was admitted for fatigue cervical pain and vertigo. The physical examination revealed the presence of vitiligo and Dupuytren's contracture, pain at the percussion of the spine (cervical to lumbar).

The laboratory tests detected leukocytosis. The abdominal ultrasound revealed the presence of a hepatic haemangioma and liver steatosis. The cervical spine radiography identified degenerative spondylotic changes.

Throughout hospitalization, the patient's condition progressively deteriorated with the apparition of macroglossia, important headaches, expressive aphasia, difficulty swallowing, weakness and then loss of control in tongue and maxillary and mandibular bones. The brain MRI showed multiple late-acute, subacute, and chronic ischaemic lesions in the pons and diffuse cerebral atrophy. The thoracic computed tomography images revealed the presence of a thymic mass.

Decision making: Based on the findings and on the symptoms, we suspected a neurological disorder and we referred the patient to the Neurology Department in the Emergency County Hospital of Sibiu. They repeated the laboratory tests (including the ACR ab - acetylcholine receptor antibodies, which came back positive). The electromyography showed a decremental response.

A few days following the transfer to the Neurology Department, the patient had the first myasthenia gravis crisis, which needed orotracheal intubation and mechanical ventilation.

Conclusions: The thymic masses can be involved in the apparition of myasthenia gravis because of their capacity to produce acetylcholine circulant receptor antibodies.

Keywords: myasthenia gravis, thymic tumour, acetylcholine circulant receptor antibodies

[Abstract:0868]

FAT EMBOLISM; WHY WE HAVE TO DIAGNOSE IN CRITICAL CARE? CASE REPORT

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A 25-year-old patient who came to the emergency with motor tk and complained of left leg pain was admitted to the intensive care unit due to hazardous consciousness. There was a 5x6 cm abrasion on the left tibia, and a bird-eye open fracture is observed in the lateral thigh. During the follow-up of the patient, incurrence of consciousness developed, and the patient was orotracheal intubated. MRI diffusion and brain tomography were taken on the patient. As a result of the MRI, diffusion restrictions compatible with millimetric embolic ischemia were observed in both centrum semiovale, corona radiata, frontoparietal lobe at the ventricular level, and parieto-occipital lobe periventricular and subcortical white matter. The patient was diagnosed with a fat embolism. The patient was operated, and methylprednisolone was started. The patient's symptoms resolved within days, and he was extubated. An improvement in the patient's course was observed within days. The use of corticosteroids in treatment is still quite controversial. The prognosis of patients with fat embolism is based on early open reduction and internal fixation of the long bone fracture. Therefore, early stabilization of open bone fractures is important.

Keywords: embolism, bone, fracture

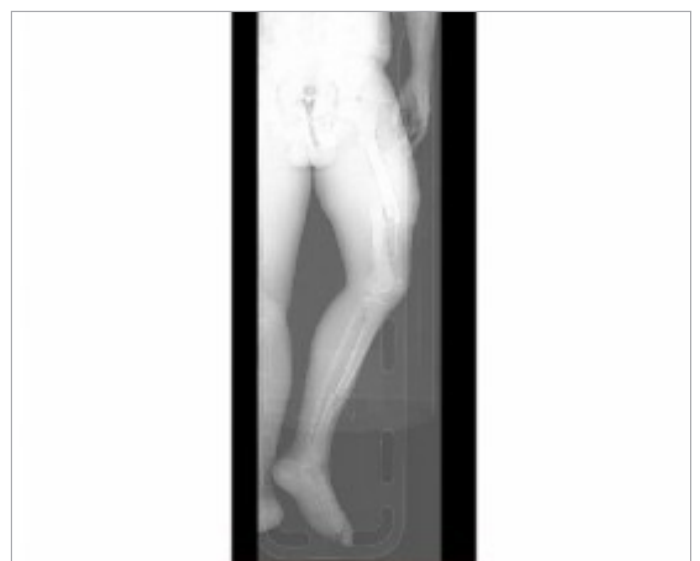


Figure 1. Permission was obtained from the patient, femur shaft fracture is observed.

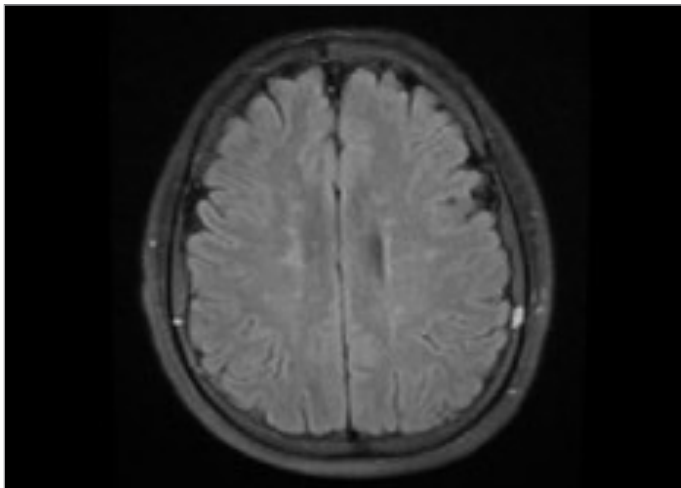


Figure 2. There are diffusion restrictions compatible with millimetric embolic ischemia in both centrum semiovale, corona radiata, ventricular level, fronto parietal lobe, and parieto-occipital lobe periventricular and subcortical white matter.

[Abstract:0902]

UNEXPECTED CONSEQUENCES: VACCINATION, GUILLAIN-BARRÉ, IMMUNOGLOBULIN AND PULMONARY EMBOLISM

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Guillain-Barré syndrome (GBs) is an uncommon immunological disease that targets the peripheral nervous system. Its aetiology remains unknown, microbial infections is related to disease onset. Male, 51 years, no known illness, vaccinated for SARS-CoV-2 (Janssen). Four days past vaccination he develops a clinical condition with fever, nausea, vertigo, and hallucinations. Fifteen days after symptoms onset, he presents with facial droop, with no other neurological symptoms. Bell palsy is diagnosed. Methylprednisolone is initiated with posterior hospital discharge. As he began corticosteroid weaning, a clinical condition of distal paraesthesia of the limbs and ataxia manifests. From its clinical features, a demyelinating condition was hypothesized. The patient was admitted in internal medicine ward. During hospital stay a brain MRI, lumbar puncture (LP), EMG and blood analysis were performed. LP showed an albuminocytological dissociation, while EMG showed sensitive-motor polyneuropathy. Immunoglobulin (Ig) was started, with clinical improvement. GBs secondary to SARS-CoV-2 vaccine was proposed as main diagnosis. Patient kept regular follow-up in Neurology. One year after diagnosis, on a routine thorax CT-Scan, an asymptomatic pulmonary embolism was diagnosed. Ig was stopped, steroid therapy initiated, and the patient was admitted to internal medicine ward. When prescribing any therapy, risk/reward must be equated.

Although one of the main therapies of GBs is Ig, it has a risk associated with increased blood coagulability. Ig reintroduction should be evaluated after a thrombotic event, as should cardiovascular function. Life threatening cause must be excluded.

Keywords: SARS-CoV-2 vaccination, Guillain-Barré, immunoglobulin, pulmonary embolism

[Abstract:0948]

SUCCESSFUL TREATMENT OF INTRAVENTRICULAR RUPTURE OF BRAIN ABSCESS ASSOCIATED WITH INTERNAL JUGULAR VEIN THROMBOSIS: CASE REPORT

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Background: Intraventricular rupture of brain abscess (IVRBA) remains a fatal complication of brain abscess despite advanced radiology techniques, laboratory diagnostics, surgical interventions, and antimicrobial treatment. The association of surgery and neurological resuscitation remains the mainstay treatment axis to improve prognosis.

Besides a good neurologic outcome may still be possible with early diagnosis and appropriate treatment.

Case Presentation: We report a case of a 52 years-old man with no past medical history was admitted to the emergency department for tonico-clonic generalized seizure activity and an acute alteration of consciousness level. Initial investigation showed an intraventricular rupture of brain abscess associated with an internal jugular vein thrombosis, swab cultures from abscess found a *Streptococcus pyogenes* growth. The patient was well managed with neurological resuscitation and surgery.

The objective of this case report is to illustrate that despite the lethal prognosis of intraventricular rupture of brain abscess, a good neurologic outcome may still be possible with early diagnosis and appropriate treatment.

Conclusions: Despite the variety of medical and surgical strategy, the association of surgery and neurological resuscitation remains the mainstay treatment axis.

Further guidelines are necessary to develop our treatment protocol and to clarify our strategy.

Keywords: intraventricular, rupture, brain abscess

[Abstract:0977]

A CASE OF ISCHEMIC STROKE DUE TO POLYCYTHAEMIA VERA

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Haematological diseases are seldom found as the aetiology of ischemic strokes, but are frequently investigated by expensive laboratory tests after a first cerebral vascular event.

Polycythaemia vera (PV) is a disease of stem cells characterized by pan hyperplastic, malignant, and neoplastic bone marrow conditions. It is characterized by an increased absolute red blood cell count due to uncontrolled red blood cell synthesis, as well as excessive white blood cell and platelet production. Although the relationship between PV and stroke, especially ischemic stroke, is widely known around the world, no previous cases have been reported from Tunisia.

We reported the case of 27-year-old men with a history of non-treated polycythaemia vera, presented to the emergency department with a dizziness and walking disorder. Initial vital signs revealed an apyrexia, a stable hemodynamic and respiratory state. Neurological examination noted a dysarthria, a left-side lower limb paraesthesia and hypoesthesia, instable gait, and a deep tendon reflex hyperreflexia. Brain scan was normal, magnetic resonance imaging (MRI) showed an area of acute right latero-pontal ischemic lesion. Cardiac studies including carotid Doppler, and trans oesophageal echocardiogram were normal. Labs were notable for persistently elevated haemoglobin (18.5 mg/dl), hematocrit (60.2%), leukocyte ($19380/\text{mm}^3$) and platelet count ($826000/\text{mm}^3$). Hydroxyurea and aspirin were initiated as well as a blood withdrawing of 350 ml three times a week until obtaining a normal haematocrit level. After treatment initiation, he had no further complains, his symptoms resolved three days after admission, and he was discharged home.

PV as the cause of ischemic stroke is a rare condition but can be encountered in clinical practice, and clinicians should be familiar with this combination.

Keywords: ischemic, stroke, polycythaemia vera

[Abstract:0990]

AN UNEXPECTED DIAGNOSIS IN A PATIENT WITH ORTHOSTATIC HYPOTENSION

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We present the case of a 69-year-old man, with history of benign prostatic hyperplasia in treatment with tamsulosin. He was recently discharged from a medical department where he presented for vertigo while standing, lower limb pain and muscle contraction which abate and recur over time and shuffling gait; the investigations described vertigo syndrome, (L5-S1) disco-radicular conflict and was prescribed analgesics and betahistine. The patient presented to our clinic for more investigations accusing the same symptomatology. A careful clinical examination showed sweaty-cold skin especially while standing, $\text{SaO}_2=98\%$, orthostatic hypotension, negative Lasegue sign, muscle rigidity and positive Noica sign.

Given the findings, we are in front of an orthostatic hypotension in a patient with tamsulosin medication, lower limb pain and contraction with nonsurgical disco-radicular conflict and extrapyramidal syndrome in observation.

The lab test revealed mild hypomagnesemia, normal calcaemia. The echocardiography disclosed normal left ventricle systolic function; the Doppler ultrasound described bilateral carotid atherosclerosis without hemodynamic significance. The CT-scan revealed cerebral and cerebellar atrophy; the electromyography showed sensitive polyneuropathy. Corroborating clinical and paraclinical data, we are in front of a parkinsonian syndrome with hypotensive manifestations and rigidity. A levodopa test was performed, followed by improvement in walking.

Levodopa diagnostic test can be used to distinguish Parkinson's disease (PD) from other neurological conditions; so, any spinal injury was infirmed.

Neurogenic orthostatic hypotension is a frequent manifestation in PD with symptomatic manifestations in about 20% of cases. It increases with PD duration, severity, age and may be variable through the day.

Keywords: orthostatic hypotension, Parkinson's disease, muscle rigidity

[Abstract:1006]

MISTAKING A CEREBROVASCULAR EVENT FOR PROSTATE DISEASE AND GI BLEEDING: HICCUP AS A SYMPTOM

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Purpose: To emphasize that hiccup should be taken more seriously.

Findings: 63-year-old male patient, without any known comorbidities, applied to the emergency with complaints of vomiting, nausea, bloating, hiccup in the stomach and inability to urinate. When globe vesical was detected on contrast-enhanced pelvis CT imaging, the patient was consulted to urology, and urology outpatient clinic evaluation was recommended after bladder catheter insertion. The patient was prescribed ciprofloxacin and doxazocin. He applied to the emergency department a day later with complaints of heartburn, vomiting and fever. Upon observing a decrease in haemoglobin value from 15.2 g/dl to 13.2 g/dl and that the vomiting was bloody in nature, the patient was evaluated as hematemesis and an urgent endoscopy was arranged and corresponding report revealed ulcers in the oesophagus, erythematous pangastritis and Forrest III ulcers in the antrum. However, it was stated that it was not expected any serious bleeding from the ulcers mentioned. The hiccup did not vanish and sometimes occurred in the form of attacks. A photo of bloody vomit was demanded from the patient. It looked more like gushing vomit. The patient underwent a diffusion MRI and was consulted to the neurology department with diffusion restriction, corresponding to ADC and Flair on the right side, starting from the corona radiata level and including the basal ganglia. Clopidogrel 75 1x1 was started and the patient was taken over by neurology department.

Conclusions: The nature of the cerebrovascular event should be carefully evaluated with hiccup complaint.

Keywords: cerebrovascular event, hiccup, vomiting

[Abstract:1059]

ACUTE DISSEMINATED ENCEPHALOMYELITIS (ADEM) RELATED TO MYELIN OLIGODENDROCYTE GLYCOPROTEIN (MOG) ANTIBODY-ASSOCIATED DISORDER

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A 21-year-old woman entered the emergency room, exposing dizziness, blurred vision and trouble urinating. Without recent drug intake and no medical record or chronic diseases, the patient was discharged with a urinary infection diagnosis. A week later, the patient was brought into the hospital, presenting headache, behavioural disturbance and a deteriorating level of conscience. At first sight, a high temperature of 39°C and a stiff neck were observed. Urgent blood tests revealed high acute phase reactants. A lumbar puncture was performed, and cerebrospinal fluid showed augmented lymphocytic recount, low glucose and a slightly increased protein presence. Neuroimaging protocol, including cerebral CT and CT angiography, did not provide further information. Empiric treatment, including ceftriaxone, vancomycin, acyclovir and levetiracetam was initiated before the patient was admitted to the ICU.

During her six-day stay at the ICU, she progressively recovered consciousness, facilitating her assistance on the hospitalization floor. Clinical improvement was notable, but residual bradypsychia, psychomotor disorders, and infantilized behaviour were still present. Serologic studies resulted in negative ruling out HIV, HCV, HBV, EBV, parvovirus, HSV, VZV and West Nile virus infections. Cerebral magnetic resonance showed hyperintense diffuse and bilateral findings, suggesting rhombencephalitis related to myelin oligodendrocyte glycoprotein (MOG) antibody-associated disorder.

Autoimmunity tests were concordant, manifesting MOG antibody presence.

Treatment with intravenous Methylprednisolone 1g per day during a week proved effective both in radiological and symptomatic improvement.

Keywords: acute disseminated encephalomyelitis, MOG antibody-associated disorder, behavioural disturbance

[Abstract:1229]

UNRAVELING THE NEUROLOGICAL ENIGMA: A JOURNEY THROUGH THE COMPLEXITIES OF AXONAL MULTIFOCAL MOTOR NEUROPATHY

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A 57-year-old woman with a history of postsurgical hypothyroidism, managed with eutirox 88 mcg, presents a five-year history of progressive weakness in her hands and feet. Initially attributing her symptoms to clumsiness, she now reports nocturnal cramps, loss of skills, and paraesthesia in her extremities, accompanied by sharp pain in her hands. Neurological examination reveals severe atrophy in the distal third of her forearms and hands, with motor deficits in this region, while sensitivity remains intact. Atrophy is also noted in the distal musculature of the legs and feet, with a deficit in dorsiflexion (4-/5) and cavus feet.

Comprehensive tests, including a normal hemogram, serum biochemistry, and negative rheumatoid factor, show normal thyroid hormone levels and a slightly elevated vitamin E level. X-rays reveal an exostosis on the third finger's tuft of the right hand. Heavy metals in blood and urine are ruled out, and serological tests for infectious and autoimmune diseases yield normal or negative results. Weak positivity for antiganglioside antibodies (ANTIGQ1B +, weakly positive anti-GM1, and anti-GM3) is noted after multiple checks. Cerebrospinal fluid analysis shows normal results.

Electromyography reveals severe and symmetrical axonal involvement in all explored motor nerves, predominantly distal and in the upper limbs, without conduction blocks. Cervical MRI discloses cervical abnormalities. Genetic testing for hereditary diseases is negative.

The final diagnosis is axonal multifocal motor neuropathy with positive antiganglioside antibodies. Treatment involves intravenous immunoglobulins at 0.4 g/kg/day for 5 days, in cycles of 6-8 weeks, with ongoing evaluation of clinical evolution.

Keywords: neuropathy, paresthesias, antiganglioside antibodies

[Abstract:1266]

MEDROXYPROGESTERONE LEADING TO TRISMUS, STROKE AND SYNCOPÉ

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Background: The medroxyprogesterone, is an injectable form of progesterone used as a contraceptive drug. Its main side effects are dominated by metrorrhagia, secondary amenorrhea and

weight gain. It can exceptionally cause syncope, strokes, and trismus. We report a case of a patient presenting these 3 rare forms 7 years after taking medroxyprogesterone.

Case Report: A 36-year-old patient, with a medical history of mixed dyslipidaemia treated by fibrates, was admitted for right hemiplegia and speech disorders. She used medroxyprogesterone for 7 years as a contraceptive method. She has presented an episode of syncope, 4 months ago. Physical examination noted a hemiparesis and a right hypoesthesia associated with a left central facial palsy, as well as a tight trismus. Laboratory screening only showed high cholesterol level. The search of thrombophilia anti-phospholipids antibodies came out negative. Homocysteine levels were normal. Cerebral angio-scan, magnetic resonance imaging (MRI) and angio-MRI were all normal. Echocardiogram and Holter monitor showed no abnormalities. Immutability of medroxyprogesterone in these side-effects was confirmed by a pharmacovigilance evaluation. The progesterone was stopped, and the patient was treated by antiaggregants. The evolution was marked by a spectacular improvement of its neurological symptoms.


Conclusions: Past medical history of dyslipidaemia and hypertension predispose to cardio-vascular disorders under medroxyprogesterone such as in our patient. Neurological disorders are represented especially by headaches (17% of the patients), syncope, convulsions, facial palsy and trismus were described in less than 1%.

Keywords: trismus, stroke, syncope, medroxyprogesterone

[Abstract:1314]

CEREBELLAR IMPACT ASSOCIATED WITH SJOGREN'S SYNDROME, REPORTING ON A CASE

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¹  The authors did not provide affiliations upon requests from the event organizer

Objectives: Description of a case and review of cerebellar involvement in systemic Sjogren's syndrome (SS).

Materials and Methods: 59-year-old patient with subacute cerebellar symptoms of 8 months' duration, starting with scandal dysarthria and vertiginous symptoms, evolving to severe dysmetria of four limbs with dysphagia, dysarthria and alteration of ocular saccadic tracking. Her personal history includes breast cancer and systemic SS. In the treatment of breast cancer, various chemotherapy drugs were used that caused axonal peripheral polyneuropathy.

Results: Antineuronal antibodies were negative, and PET-CT was normal. Anti-Ro52 (SSA) and anti-La (SSB) antibodies were detected in the serum study, and only anti-Ro52 (SSA) antibodies were detected in the cerebrospinal fluid (CSF). Successive MRI revealed progressive cerebellar atrophy. Treatment with periodic

immunoglobulins was decided, with clinical stabilization. We reviewed the literature looking for cases of cerebellar involvement associated with SS, describing a series of 14 cases of ataxia and Sjogren's syndrome. Of these 14 patients, cerebellar signs were found in 5/14, 4 of them showing cerebellar atrophy on imaging. The response to immunosuppressive treatment (corticosteroid therapy, rituximab, immunoglobulins, etc.) was variable in all. In the original article, it was observed that cerebellar involvement was related to the presence of anti-Ro (SSA) antibodies and not anti-La (SSB), as occurred in our patient.

Conclusions: Cerebellar involvement associated with SS is a rare condition. Anti-Ro (SSA) antibodies are related to cerebellar involvement more than anti-La (SSB).

Keywords: Sjogren's syndrome, cerebellar, anti-Ro (SSA)

[Abstract:1371]

DOCTOR, IS MINE ALZHEIMER'S?

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We present a 78-year-old man who started with behavioural disturbance progressing rapidly with delusions, tremors, cognitive impairment, and loss of 30 kg. He was admitted for study of rapidly progressive dementia and possible occult neoplasia. On physical examination we found intoxication with complex orders, time-spatial disorientation, inexhaustible Glabellar and positive prehension. Cachexia, hypomimia, frontalis hypercontractibility, gaze supraversion limitation. Symmetrical stiffness. No tremor, bradykinesia, or myoclonus. No pyramidalism data. B6 deficiency and hypoalbuminemia. Negative serologies and blood cultures. Body-CT, colonoscopy, and upper gastrointestinal endoscopy without evidence of neoplasia. Normal cranial MRI. PET-FDG without loss of presynaptic dopaminergic terminals. Electroencephalogram with diffuse baseline slowing. Cerebrospinal fluid (CSF) with negative cytobiochemical culture and oligoclonal bands. Positive Alzheimer's biomarkers. Positive 14-3-3 protein. RT-QuIC positive.

We present a patient with rapidly progressive dementia, signs of parkinsonism and psychotic symptoms, ruling out toxic-metabolic, pharmacological, neoplastic, infectious, autoimmune, and vascular causes. We suspected about neurodegenerative storage disease and/or prion. Compatible symptoms and positive biomarkers for Alzheimer's Disease (AD) and Creutzfeldt Jakob disease (CJD). Biomarkers of AD are useful in atypical forms with complex diagnosis. Prionopathies are rare diseases due to pathological deposit of prion protein's abnormal isoform (PrPsc). Probable disease is defined as a progressive neurological condition and positivity for PrPsc (RT-QuIC technique) in CSF, with 99% specificity.

Our patient presented clinical and analytical diagnostic criteria compatible with CJD and AD, making its assessment difficult. Not all dementias are AD. The diagnosis of rapidly progressive dementia is complex. Symptoms, signs, and biomarkers help us in this task.

Keywords: Alzheimer disease, Creutzfeldt Jakob disease, dementia

[Abstract:1396]

IMPROVING POST-STROKE VISION IMPAIRMENT WITH A DEDICATED ORTHOPTIC SERVICE AT UNIVERSITY HOSPITAL SOUTHAMPTON NHS TRUST

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Aim: As many as half of people with stroke suffer with vision problems. These can include poor central vision, eye movement problems and peripheral vision loss. An outpatient-based service to assess visual impairment was less than adequate. We set up an inpatient service led by a senior Orthoptist to provide timely assessment of vision and its management.

Methods: All the patients referred to this service were included in this review over a period of one year. We reviewed the timeliness, follow up arrangements and patient experience survey.

Results: A total of 261 patients were seen as inpatient and 67 patients were seen as new outpatients. 58% of patients referred were seen within the target of 3 days. The remaining were deemed to be medically unwell for assessment. 98% of patients received specialist orthoptic assessment. 76% of patients received sensory support and 66% further driving advice. Patient experience survey showed a majority positive response.

Conclusions: Over 300 patients were seen within one year, with most inpatients seen within 3 days of referral. Feedback from the stroke MDT has been extremely positive. Patient reported experience measures show that patients find the dedicated service beneficial and have confidence in the orthoptist. The orthoptist provides timely review of patients and advises the patient, family, carers, and MDT on how their visual problem may affect their rehabilitation. The orthoptist provides a direct link to additional ophthalmology services as required, and support with returning to driving, sight impairment registration and additional community support.

Keywords: orthoptic, stroke, vision impairment

[Abstract:1399]

EFFECTS OF COCAINE MISUSE ON NEUROCOGNITIVE IMPAIRMENT IN PATIENTS WITH ALCOHOL DEPENDENCE

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Purpose: The impairment of memory and executive functions by chronic cocaine use has been described in previous studies. However, these findings are neither universal nor uncontroversial. Other studies concluded that the cognitive impairment was due to the alcohol abuse.

Methods: Prospective study including 16 patients admitted for detoxification at Hospital Universitario de Canarias and 16 abstainers controls. The following neurocognitive tests were performed: verbal fluency test and the Stroop-colour test (executive function); Rey complex figure-copy (visuospatial function); number key and trail making test (perceptual-motor function); Rey complex figure-memory and the RAVLT test (mnestic function).

Findings: The median age was 54 [45-56.5] years, of which 7 patients used cocaine in addition to alcohol. Patients who used alcohol and cocaine consumed less alcohol than those who used only alcohol (96 [80-220] vs 320 [160-438] grams; $p=0.015$). Patients with dependence on both substances had worse Rey complex figure-memory scores (13 [5-15.5] vs 23 [16-25]; $p=0.004$) and a trend in Rey complex figure-copy (34 [27-36] vs 36 [34-36]; $p=0.089$). The semantic fluency score was better in users of both substances (23 [16-25] vs 15 [10-17]; $p=0.017$), as well as a non-statistically significant trend in the number key (280 [257-539] vs 559 [399-931] seconds; $p=0.081$).

Conclusions: In our sample, patients with alcohol dependence who also use cocaine consume lower amounts of alcohol. These patients have better memory function and better executive functions, probably related to lower alcohol consumption. Further studies are needed to clarify whether the effect of cocaine is really negative.

Keywords: cognitive impairment, cocaine dependence, alcohol dependence

[Abstract:1485]

SEIZURES AS THE FIRST MANIFESTATION OF A REVERSIBLE POSTERIOR LEUKOENCEPHALOPATHY SYNDROME - A CLINICAL CASE

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Reversible posterior leukoencephalopathy syndrome (RPLS) is a clinical radiographic syndrome of heterogeneous etiologies that are grouped together because of similar findings on neuroimaging studies. RPLS has been described in a number of medical conditions and prompt recognition and treatment is important in preventing the permanent damage that can occur in this otherwise typically reversible condition.

A 19-year-old woman, with no relevant medical history, treated only with oral contraceptive, was brought to the emergency room after inaugural generalized clonic seizures, with sphincter incontinence. Laboratory results were all within normal values, and there was no evidence of consumption of illicit substances (negative urine drug test, blood alcohol levels below detection limit). She was admitted in the ward for further study because there was evidence of cortico-subcortical hypodense areas on tomography brain computer scan suggestive of ischemic or vasculitic aetiology. Antibody serology tests (for the most common agents), and autoimmune study were negative. MRI showed findings compatible with posterior reversible leukoencephalopathy syndrome (PRES). During her hospitalization, there were no new seizures and there were no other complications. Oral contraceptive was suspended and discharged, for consultation. We were not able to identify the precipitating factor for RPLS.

Most case series and case reports suggest that RPLS is usually benign. In many cases, RPLS seems to be fully reversible within a period of days to weeks. Radiologic improvement lags behind clinical recovery.

Keywords: leukoencephalopathy, seizures, headache

[Abstract:1521]

CONSIDERATIONS DURING RE-ENTRY OF PATIENTS WITH COGNITIVE IMPAIRMENT IN AN INTERNAL MEDICINE SERVICE OF A SECOND LEVEL HOSPITAL

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Purpose: to study the presence of cognitive impairment in patients admitted to our service and determine if this process determines a higher readmission rates.

Methods: real life study. We select patients admitted to our internal medicine service on an undetermined day. We consider the total number of admissions, the number of patients readmitted, the presence or absence of cognitive impairment, the origin of the patients (family home or nursing home) and readmission with respect to their origin.

Findings: On the cut-off day there were a total of 71 patients admitted to the internal medicine service of our hospital. Of them, 41 came from home and 30 from a nursing home. We found a total of 23 patients (32% of the total) with cognitive impairment, of which 8 lived in a family home and 15 in a nursing home.

Of the total number of patients, 27 of these (38% of total admissions) were re-admitted patients, 13 lived in a family home and 14 in a nursing home. 33% (9 patients) had cognitive impairment, 2 lived in a family home and 7 in a nursing home.

Conclusions: 1. A third of the patients in our sample present cognitive impairment.

2. Patients with cognitive impairment mostly come from nursing homes.

3. Half of the patients who require admission or re-admission, from nursing homes, present cognitive impairment.

4. An extension of the study is necessary in order to reach conclusions that enable decision-making.

Keywords: cognitive impairment, readmission, internal medicine

[Abstract:1558]

UNMASKING NEUROSYPHILIS IN A COMPLEX PSYCHIATRIC PRESENTATION

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A 46-year-old male with a recent psychiatric admission history due to a first episode of reactive brief psychosis following a romantic breakup, displaying megalomaniacal and persecutory delusions along with aggressive behaviour. The symptoms initially responded to antipsychotic medication but reappeared three days later, leading to readmission.

During hospitalization, blood, and urine tests, as well as a cranial computed tomography, yielded unremarkable results. Serological testing was negative for hepatitis C, B, and human immunodeficiency virus but positive for syphilis (positive treponemal test and RPR titer 1/4). A lumbar puncture confirmed neurosyphilis based on cerebrospinal fluid (CSF) analysis, indicating a crystalline appearance with lymphocytic pleocytosis, normal glucose levels, and a positive VDRL.

The patient was treated with intravenous ceftriaxone for 14 days and a subsequent dose of intramuscular penicillin G, showing significant improvement with amnesia of the delusional episode. He was discharged pending cranial magnetic resonance imaging and outpatient follow-up with CSF analysis every 6 months.

Discussion and Learning Points: This case represents a psychotic episode incongruent with the typical age of onset, later determined to be secondary to neurosyphilis. Neurosyphilis can occur at any stage of the infection, and the patient's serological findings suggest a possible late-stage syphilis. The case seems to align with general paresis, a form of neurosyphilis where psychiatric manifestations are more common than the typical symptoms.

It is crucial to rule out organic causes in psychiatric presentations, especially when they occur at an unusual age or follow an atypical course, considering neurosyphilis in the differential diagnosis.

Keywords: neurosyphilis, psychosis, delusions

[Abstract:1583]

CHA₂DS₂-VASC SCORE IN PREDICTING THE RISK OF CONTRAST-INDUCED NEPHROPATHY IN PATIENTS WITH ACUTE ISCHEMIC STROKE

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Purpose: The CHA₂DS₂-VASC score is developed to predict stroke risk in patients with atrial fibrillation. We evaluated the CHA₂DS₂-VASC score to estimate contrast-induced nephropathy (CIN) in patients with stroke who underwent urgent computerized tomography (CT) angiography.

Methods: 201 patients with stroke undergoing CT angiography were enrolled in this study. Patients' age, gender, body mass index, smoking, fasting glucose, urea, and creatinine levels were noted, and CHA₂DS₂-VASC scores were calculated. CIN was diagnosed with any of the following: an increase in serum creatinine (Scr) ≥ 0.5 mg/dl, a decrease in estimated glomerular filtration at a rate of $\geq 25\%$, an increase in SCR $\geq 25\%$ within 48 to 72 hours after contrast exposure. Patients were grouped into two: study group 1 included patients who developed CIN and control group 2 who did not.

Findings: CIN was reported in %16 (N: 32) of the patients. The demographic characteristics of the groups are listed in Table 1. The developed group had a higher frequency of atherosclerotic heart disease and previous stroke history. The patients with CIN had higher CHA₂DS₂-VASC scores (5.59 ± 1.50 vs 4.87 ± 1.38 ; $p=0.008$) and mortality. A significant relationship was observed in the regression analysis between contrast material nephropathy and CHA₂DS₂-VASC score ($p=0.0097$). In ROC curve analysis, the optimum cut-off point for the CHA₂DS₂-VASC score was determined as >5 (sensitivity: 62.5; specificity: 61.54)

Conclusions: In acute ischemic stroke patients undergoing CT angiography, the CHA₂DS₂-VASC scoring system can be safely used to evaluate the risk of CIN development.

Keywords: acute ischemic stroke, contrast induced nephropathy, CHA₂DS₂-VASC score

	group 1 (CIN +) n = 32	group 2 (CIN -) n = 169	p
Age (year)	73.65 \pm 12.86	69.33 \pm 12.52	0.081
BMI (kg/m ²)	28.38 \pm 4.67	27.32 \pm 4.16	0.411
Gender			
Male	16 (50%)	85 (50,03%)	0.564
Female	16 (50%)	84 (49.7%)	
Mortality	7 (21.9%)	4 (2.4%)	<0.001
Creatinine (mg/dl)	0.79 \pm 0.16	0.81 \pm 0.15	0.532
CHA ₂ DS ₂ -VASC score	5.59 \pm 1.50	4.87 \pm 1.38	0.008
ProBNP (pg/ml)	972.1 \pm 2223	2989 \pm 6601	<0.001
EF (%)	60.78 \pm 26.76	57.57 \pm 8.27	0.345
$p<0.005$			

Table 1. Characteristics of patients with and without CIN

[Abstract:1678]

CEFEPIME-INDUCED ENCEPHALOPATHY

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We present the case of an 85-year-old woman who was admitted to the General Surgery service for suspected acute cholecystitis. Conservative management was decided, given the patient's age and comorbidities, so antibiotic treatment was started with cefepime and metronidazole. The patient is progressing favourably from a digestive point of view.

Coinciding with the start of treatment with cefepime, the patient began with acute neurological deterioration consisting of: dizziness with gait instability, dysarthria, tremor, temporospatial disorientation and behavioural alterations with inappropriate language.

The patient is evaluated by Neurology. A CT scan of the skull was performed in which no notable alterations were observed, and a cerebrospinal fluid study was normal. The study is expanded with an EEG that is compatible with generalized non-convulsive status epilepticus. Anticonvulsant treatment was started and cefepime was suspended, with complete resolution of the neurological condition, which is why the condition was attributed to probable neurotoxicity induced by cefepime.

Keywords: cefepime, encephalopathy, neurotoxicity

[Abstract:1682]

WHEN THROMBOTIC AND HAEMORRHAGIC PHENOMENA COEXIST OVER TIME

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76-year-old woman, with no history of interest, except for months-old depression, was admitted for ischemic stroke with M3-M4 occlusion, treated with fibrinolysis. After this, clinical worsening, moving from NIHSS 10 to 25, showing multiple bilateral haemorrhagic foci on CT. In control, decrease in platelets 72,000, drop in Hb 10.7 as well as hypofibrinogenemia (110), which in subsequent controls, despite a total of 3 platelet pools, 2 g fibrinogen, 2 g anchaefibrin and 1000 cc of fresh plasma, continued to decline, with platelets of 58,000, lengthening of clotting times, increase in D-dimer, as well as persistence of hypofibrinogenemia. Peripheral blood smear and Adams 13 were performed, with 5% schistocytes, but ruling out PTT. Third day, clinical worsening, with the appearance of pruritic skin lesions, and reaching NIHSS of 33 points (drowsiness, right hemianopsia, facial paralysis, tetraplegia, mutism, decreased sensitivity), observing new ischemic lesions in the territory of the left ACA, and Right ACP. Telemetry with sinus rhythm. No infection. Transthoracic echocardiography ruled out endocarditis. With the present data, she met criteria for disseminated intravascular coagulation. We requested a CT scan of the chest and abdomen to rule out neoplasia as a predisposing cause, revealing a neoplastic tumour in the body-tail of pancreas, due to infiltrating carcinoma with splenic vein thrombosis and splenic infarcts with metastatic liver disease.

Disseminated intravascular coagulation is due to an activation of the extrinsic coagulation cascade, as well as the fibrinolytic pathway, and can cause both thrombosis and haemorrhage. It can be caused by infections, neoplasms, among other causes.

Keywords: disseminated intravascular coagulation, neurology, neoplasia

[Abstract:1716]

DIFFICULTY IN AMBULATION, AS A MANIFESTATION OF PROGRESSION OF CHRONIC LYMPHOCYTIC LYMPHOMA

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70-year-old male, tobacco user and dyslipidaemia. Moderate L4-L5 stenosis. Follow-up by Haematology for chronic lymphocytic lymphoma (stable at last review). Referred from Rehabilitation,

due to progressive difficulty in ambulation for 4 months. It associates dysesthesia and loss of sensitivity in the lower limbs (MMII), not the ascending ones. In the month prior to admission, there was weakness in the upper limbs and added difficulty in urination (need for permanent bladder catheterization) and use of several drugs due to marked constipation. Weight loss of 20 kg in last year. Cervical trauma 11 months ago, with MRI without findings. On examination, a picture of progressive tetraparesis was observed with tactile-algescic hypoesthesia of the left lower limb and arthrokinetic decrease of the right upper limb, with T5 sensory level and stylo-radial hyperreflexia. Reflexes abolished in MMII. Babinski positive. Given the possibility of spinal cord involvement at the cervical or lumbar level, due to described trauma or already known lumbar narrowing, we observed the symptoms of first motoneuron (stylo-radial hyperreflexia, Babinski positive) and second one (MMII hyporeflexia, atrophy), would not be fully explained by said aetiologies. We performed MRI of the entire spine, revealing compressive cervical myelopathy (not a candidate for surgery), along with longitudinally extensive myelitis, which after two CSF determinations, confirmed tumour origin due to known chronic lymphocytic lymphoma. An enlarged MRI of the skull also confirms the presence of inflammatory-type lesions consistent with extension of myelitis. Ibrutinib was started, with the patient improving, able to walk with a walker upon discharge.

Keywords: chronic lymphocytic lymphoma, neurology, disease

[Abstract:1727]

"LUCKY" STROKE: WHEN TO SCREEN FOR INTRACRANIAL ANEURYSM IN AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE?

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Summary: Intracranial aneurysm (IA) is a serious complication in autosomal dominant polycystic kidney disease (ADPKD). Incidence of IA is about 3-7% (12-15%, if there's family history of IA) in ADPKD patients - five times higher than general population. IA rupture also occurs a decade earlier in these patients, with a mortality rate of 35-55%.

Purpose: 72-year-old man, with personal history of past-smoking, arterial hypertension and ADPKD, goes to the hospital after transient episode of left hemiparesis and right facial hypoesthesia. There were no neurological deficits upon arrival.

Methods: Cranioencephalic computed tomography showed ischemic stroke on the right middle cerebral artery territory and five aneurysmatic formations, the biggest with 10 millimetres. When asked, he reported two early age deaths in his family due to stroke (sister and maternal aunt). Without neurological deficits

and outside of timing for thrombolysis or thrombectomy, he was medicated with antiplatelet agents. He underwent endovascular treatment for the biggest aneurysm and continued surveillance by Neurosurgery.

Conclusions: Currently, there are no standardized societal guidelines about IA screening in ADPKD patients, but routine screening is recommended in the high-risk patients, which include those with: previous IA rupture, positive family history of a brain haemorrhage or stroke, warning symptoms (headaches, loss of consciousness) or high-risk occupation (airline pilot). With this clinical case, we intend to alert to one of the several conditions outside the kidney that can occur in ADPKD, and point out which patients deserving of screening for IA.

Keywords: ADPKD, polycystic, aneurysm

[Abstract:1729]

NEUROLOGICAL IMPAIRMENT AND FEVER. THE DIFFICULTY IN DIAGNOSIS

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This is an 84-year-old man with hypertension, diabetes, hypothyroidism, major depression with psychotic symptoms, chronic kidney disease, atrial fibrillation, aortic prosthesis, and ischemic stroke. His treatment is venlafaxine, mirtazapine, risperidone, omeprazole, acenocoumarol, atorvastatin and digoxin.

He begins the day before with confusion, rigidity, dysautonomia, fever, and neurological impairment; no headache, nausea, or vomiting. On examination, his general condition was poor, his eyes opened spontaneously, he directed his gaze to the call, he did not obey orders, supraversion of his gaze, normal pupils, stiff neck, plegia of the right hemibody with rigidity and spontaneous mobilization of the left hemibody. No other notable data.

Analysis with anaemia. Normal chest X-ray. Electrocardiogram with atrial fibrillation. Cranial tomography without new findings with cerebral corticosubcortical atrophy and small vessel leukopathy; and lumbar puncture with normal cytobiochemistry and sampling for CSF culture as well as HSV and VZV PCR, which were also negative.

He is admitted with suspected neuroleptic malignant syndrome secondary to risperidone. Neuropsychiatric medication was suspended and treatment with dantrolene was instituted with evident clinical improvement, although after its withdrawal he again presented neurological deterioration, so a new cranial study was performed, showing signs of reversible posterior leukoencephalopathy. A study was completed with an electroencephalogram that showed poorly integrated and slightly slowed brain activity without significant data of focal slowed involvement and with an absence of critical/intercritical epileptiform activity. The clinical evolution is favourable,

recovering higher functions and returning to his baseline situation.

Keywords: neurological impairment, leukoencephalopathy, neuroleptic syndrome

[Abstract:1748]

AN UNEXPECTED TURN OF EVENTS

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52 years old female, former smoker with no other relevant history, presents with abdominal pain during the last months, complicated with vomiting and 8 kg weight loss in the last 2 months. Examination shows ill-defined mass in the epigastrium. Blood tests show mild acute phase reactants elevation, hypoalbuminaemia and folate deficiency without anaemia.

CT finds diffuse gastric wall infiltration, perigastric adenopathies and omental thickening (figure 1). Upper endoscopy confirms the presence of a gastric neoplasm suggestive of diffuse carcinoma with progressive stenosis, and biopsies are taken (figure 2).

The patient is started on parenteral nutrition due to the impossibility of enteral nutrition.

Sudden onset of diplopia and dizziness requires a neurological exam, finding a multidirectional nystagmus, a positive Romberg test (right sided) and unstable right-leaning gait. Multimodal brain CT rules out ischemic causes or metastatic disease.

Suspicion of Wernicke encephalopathy (WE) prompts treatment with thiamine. Serologic tests for paraneoplastic syndromes (autoantibodies) come back negative, and the patient improves with thiamine treatment, confirming the diagnosis.

The patient is finally started on chemotherapy (after definitive histological confirmation of gastric adenocarcinoma).

This case exemplifies a less known manifestation of a classical syndrome, and it underscores the importance of having WE present in settings different to alcoholism. WE is increasingly seen in cases secondary to excessive vomiting, intestinal obstruction and malignancy. Diagnosis is made on a clinical basis, preferably using Caine criteria. Treatment must be quickly started to prevent sequelae, and treatment response serves as diagnostic confirmation (1).

References:

1) <https://doi.org/10.1016/j.mayocp.2019.02.018>

Keywords: nystagmus, Wernicke, thiamine

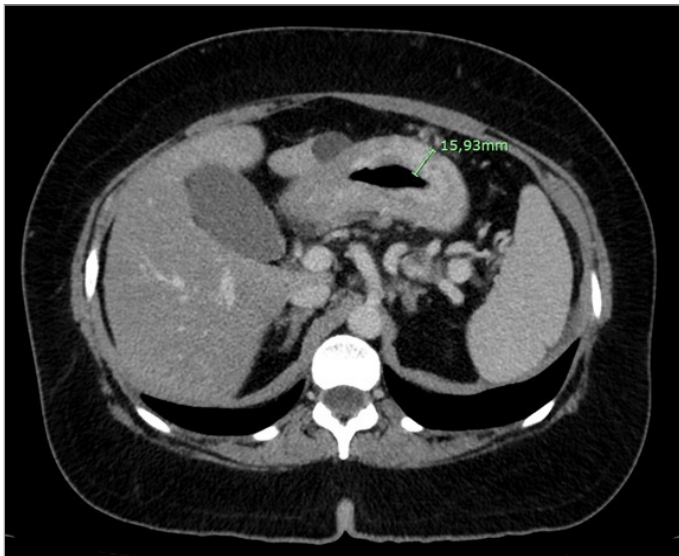


Figure 1. CT scan demonstrates pangastric wall thickening compatible with gastric linitis plastica



Figure 2. Upper endoscopy showing gastric neoplasm compatible with gastric carcinoma with progressive stenosis, which makes it impossible to reach the gastric antrum.

[Abstract:1797]

THERAPEUTIC ADHERENCE IN A COHORT OF PATIENTS WITH STROKE AND HEART FAILURE

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Adherence to therapy is difficult in populations with low socioeconomic status or for those whose dosage interferes with their rhythm of life. We studied a cohort of patients admitted to the Stroke Unit with known heart failure, conducting a survey to determine the level of therapeutic adherence and the reasons for non-adherence to treatment.

Methods: From January 1, 2023, to November 15, 2023, 126 patients with ischemic stroke and heart failure were included. An interview was conducted to determine adherence to therapy and, in the case of suboptimal adherence, to determine the reasons for it.

Results: The median age was 63 years. Fifty-six percent were women and 44% men. In the subgroup 67% of women had poor adherence to treatment, the main cause being difficulty in taking medication because they were the main caregivers of dependent

persons (59%) and/or their work schedule (43%). Twenty-six percent acknowledged that they could not afford to pay for their treatment. At the male subgroup, the main cause was the side effects associated with treatment (71%), followed by not being able to afford treatment (49%).

Conclusions: Socioeconomic factors should be taken into account when reviewing adherence to treatment and the success of drug treatment and lifestyle changes.

Keywords: therapeutic adherence, socioeconomic factors, lifestyle changes

[Abstract:1799]

INCIDENCE OF ELEVATED LIPOPROTEIN (A) IN A COHORT OF PATIENTS WITH STROKE

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Background: Lipoprotein (a) is a well-established risk factor in coronary heart disease, but its role is poorly defined in etiopathogenesis of cerebrovascular disease. Accumulation of lipoprotein (a) has been demonstrated in the arterial walls of human coronary and cerebral vessels.

Methods: An observational retrospective study considered admission to a Stroke Unit in Neurology Department from January 2022 to October 2023. We collected demographic data, antecedent of heart failure, hypercholesterolemia or previous stroke.

Results: Inclusion criteria were met by 310 patients. The mean age was 67 years. The 62% were male. There was a 58% prevalence of heart failure and a 26% prevalence of previous ischemic coronary heart disease. Overall, 89% of the patient had hypercholesterolemia at the moment of admission at the Stroke Unit and 21% had a lipoprotein (a) over 50 mg/dl.

Conclusions: Lipoprotein (a) is a biomarker that should be the object of further research. It could be useful as biomarker predictor of future ischemic stroke.

Keywords: lipoprotein (a), stroke, hypercholesterolemia

[Abstract:1801]

GUILLAIN-BARRÉ SYNDROME SECONDARY TO CHRONIC MYELOMONOCYTIC LEUKAEMIA

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Case: A 68-year-old woman consulted for general weakness and

peribuccal paraesthesia. On examination, she presented mild paraparesis in both lower limbs, osteo-tendon reflexes abolition and indifferent bilateral cutaneous-plantar reflex.

Laboratory tests, chest X-ray and skull CT were performed, highlighting only monocytosis and macrocytic anaemia. Cerebrospinal fluid analysis showed albumin-cytological dissociation.

Rapidly progressive neurological deterioration with decreased level of consciousness, bilateral facial paralysis and diplegia of the upper limbs occurred, developing respiratory arrest and requiring invasive mechanical ventilation.

As differential diagnosis of acute flaccid areflexic tetraparesis we considered second motor neuron diseases:

- Acute spinal cord pathology, unlikely due to the absence of a traumatic history.
- Infectious cause, ruled out due to the cyto-biochemical characteristics and CSF cultures.
- Toxic-metabolic neuropathies, excluded due to the absence of heavy metals in the blood.
- Demyelinating diseases such as Guillain-Barré syndrome (GBS), compatible due to the time course and clinical evolution.

Additional tests revealed elevated vitamin B12 and beta2-microglobulin. A peripheral blood smear showed monocytosis with signs of double granulocytic population (pseudo-pelger), suggestive of chronic myeloproliferative, and bone marrow study confirmed the diagnosis of chronic myelomonocytic leukaemia. Finally, neurophysiological study showed demyelinating sensory-motor polyneuropathy with axonal degeneration highly compatible with GBS and antiganglioside antibodies resulted negative.

Discussion: GBS is characterised by areflexia and motor paralysis. Its association with some haematological processes has been described. Immune system dysfunction in chronic myelomonocytic leukaemia explains its association with certain autoimmune phenomena, considering the existence of GBS to be a paraneoplastic manifestation of the haematological pathology.

Keywords: Guillain-Barré, leukaemia, polyneuropathy

[Abstract:1860]

HAEMATOLOGICAL DISEASES AS A RARE CAUSE OF CEREBROVASCULAR PATHOLOGY. DESCRIPTIVE STUDY

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Background: Haematological diseases are considered atypical causes of cerebrovascular pathology. However, many can present as ischemic strokes, intracerebral haemorrhages or cerebral venous thrombosis.

Aim and Methods: We retrospectively studied all cases of ischaemic strokes, intracranial haemorrhages or cerebral venous thrombosis admitted to a Neurology Department from January 2020 to October 2023. We selected all cases in which the aetiology was a haematological disease, known at the time of admission to the Neurology Department or diagnosed during their clinical stay. 42 patients of 427 were eligible for this descriptive study.

Results: Mean age was 42 years old, 53% females and 47% males. 18 patients had hereditary or acquired thrombophilia, 7 patients were diagnosed with polycythaemia vera, 5 patients were diagnosed with essential thrombocythemia, 11 patients had known multiple myeloma or were diagnosed at the time of ischaemic stroke and 1 patient had Waldenström's macroglobulinemia. Overall, the diagnosis was made during the clinical stay in 64 % of cases.

Conclusions: Haematological aetiology is a rare cause of cerebrovascular pathology. However, it needs to be included in the differential diagnosis.

Keywords: haematological disease, cerebrovascular pathology, ischaemic stroke

[Abstract:1864]

MISUSE AND ABUSE OF OMEPRAZOLE AS A CAUSE OF ENCEPHALOPATHY. OBSERVATIONAL STUDY

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Background: Omeprazole is a proton pump inhibitor commonly prescribed as a prophylaxis for stress ulcers or for use of non-steroidal anti-inflammatory drugs. However, side effects include hypomagnesaemia, which may promote encephalopathy.

Methods: We studied all patients admitted to the Department of Neurology from January 2021 to October 2023 for encephalopathy. We reviewed how many patients suffered encephalopathy due to infectious causes, how many due to drug use, how many due to metabolic causes (e.g. diabetic ketoacidosis) and how many due to hypomagnesaemia.

Results: We studied 87 patients. The median age was 41 years. 52% were male and 48% were female. We observed that in 43.7% of the cases, the cause was infectious. 12.6% of the cases were due to diabetic ketoacidosis. 37.9% of the cases were caused by drugs and 5.7% by drugs of abuse.

In the subgroup of patients with drug-induced encephalopathy, this was caused by omeprazole, manifesting as severe hypomagnesaemia. Some patients were taking omeprazole without prescription, or its indication had not been reviewed. Two patients were taking omeprazole as an antacid after meals.

Conclusions: Encephalopathy caused by misuse or abuse of omeprazole, manifesting as severe hypomagnesaemia, is a cause to be considered in the differential diagnosis. It is our responsibility

to review the prescription of proton pump inhibitors and to educate the population.

Keywords: omeprazole, hypomagnesaemia, encephalopathy

[Abstract:1895]

SERUM C-REACTIVE PROTEIN TO ALBUMIN RATIO AS A RELIABLE MARKER OF DIABETIC NEUROPATHY IN TYPE 2 DIABETES MELLITUS

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Objectives: Type 2 diabetes mellitus (T2DM) may be complicated with chronic complications including diabetic neuropathy (DN). Timely diagnosis is crucial in treatment and maybe reversal of this complication. C-reactive protein to albumin ratio (CAR) is considered as a marker of inflammation in various diseases that characterized with inflammation. Since diabetic neuropathy and T2DM are also associated with chronic, low-grade inflammation, we aimed to study CAR levels of type 2 diabetic subjects with diabetic neuropathy and to compare to those in patients without diabetic neuropathy.

Methods: T2DM patients presented to our institutional outpatient clinics were divided into two according to the presence of DN. Subjects with DN were listed as DN group and others were as non-DN group. Characteristics and laboratory data, including CAR, in DN and non-DN groups were compared.

Results: Median CAR of the DN and non-DN groups were 2,19 (0,2-49%) and 0,56 (0,02-5,8%), respectively ($p<0.001$). CAR was significantly and positively correlated with weight ($r=0.19$, $p=0.01$), BMI ($r=0.11$, $p=0.03$), waist circumference ($r=0.10$, $p=0.046$), fasting glucose ($r=0.14$, $p=0.004$), serum creatinine ($r=0.25$, $p<0.001$), triglyceride ($r=0.17$, $p<0.001$), and LDL-cholesterol ($r=0.13$, $p=0.001$) levels, and inversely correlated with eGFR ($r=-0.16$, $p<0.001$). The sensitivity and specificity of CAR (when higher than 1,02%) in predicting diabetic neuropathy were 78% and 73%, respectively (AUC: 0.84, $p<0.001$, 95% CI: 0.82-0.87), (Figure 1). Moreover, high CAR level was an independent risk factor of diabetic neuropathy ($p<0.001$, OR: 1.34, 95% CI: 1.08-1.62).

Conclusions: We suggest that elevated CAR levels could be considered as a marker of diabetic neuropathy.

Keywords: type 2 diabetes mellitus, diabetic neuropathy, inflammation, C-reactive protein to albumin ratio

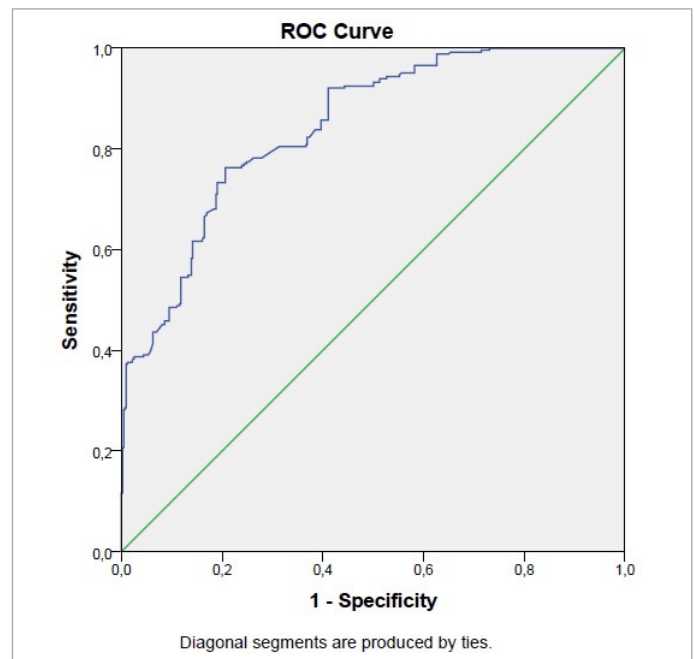


Figure 1. ROC curve of CAR in detecting diabetic neuropathy.

[Abstract:1945]

ACUTE PARAPARESIS: A MUST-KNOW CLINICAL SIGN OF BILATERAL ACA STROKE

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A woman in her 70's with a history of hypertension, dyslipidaemia and type 2 diabetes mellitus went to the Emergency Room presenting acute onset paraparesis with no previous trauma, fever, or sphincter dysfunction. Physical exam showed 3/5 symmetrical lower limb weakness with absent Babinski sign. Head computed tomography (CT) revealed a cortico-subcortical frontal left ischemic lesion in the territory of the anterior cerebral artery (ACA). No thrombolysis or thrombectomy was performed. 24 hours after admission the patient was found unresponsive, opening the eyes only to painful stimuli, left central facial palsy and flaccid tetraparesis. A second head CT showed bilateral distal occlusion of the A4 segment of the ACA, affecting the bilateral superior frontal cortex, anterior portion of the corpus callosum, predominantly in the left hemisphere. Angiography demonstrated occlusion of the left pericallosal azygos branch of ACA showing a type IV variant (irrigated both hemispheres). The patient presented frontal lobe stroke as akinetic mutism, stereotyped dorsiflexion movements of the left hand, primitive reflexes. Moreover, had basal ganglia symptoms such as grasping sign, snout reflex and enhanced glabellar tap response. The patient maintained her neurological status and was discharged to a long-term care facility.

ACA strokes are rare, representing less than 3% of stroke cases, most showing anatomical anomalies of the anterior part of the circle of Willis in bilateral ACA strokes. This particular patient had an anatomical variant in the ramification of the left ACA segment.

Bilateral ACA stroke might appear as akinetic mutism, paraplegia, and amnesia with apathy.

Keywords: acute paraparesis, ACA stroke, anatomical variant

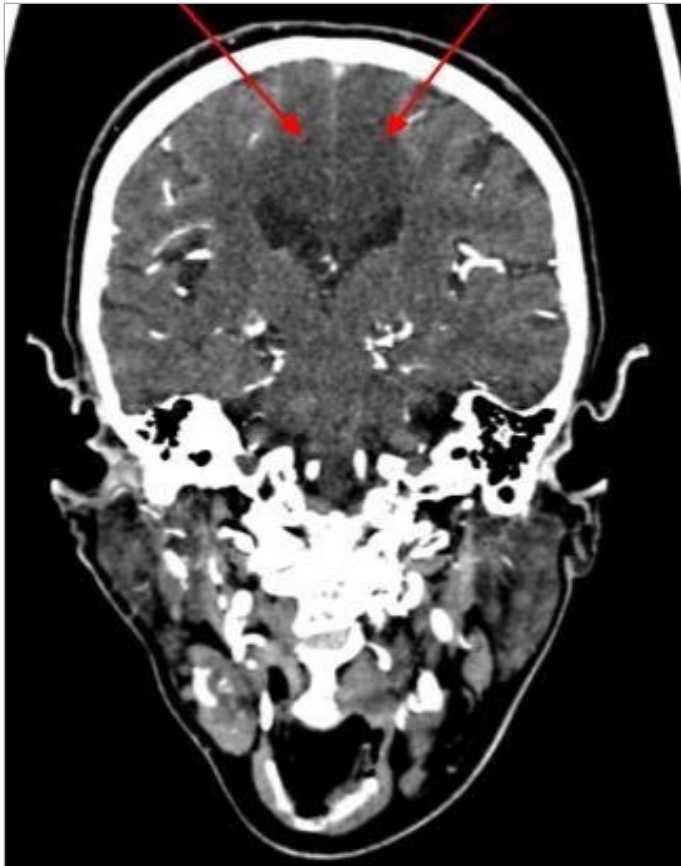


Figure 1. CT head scan showing the bilateral stroke.

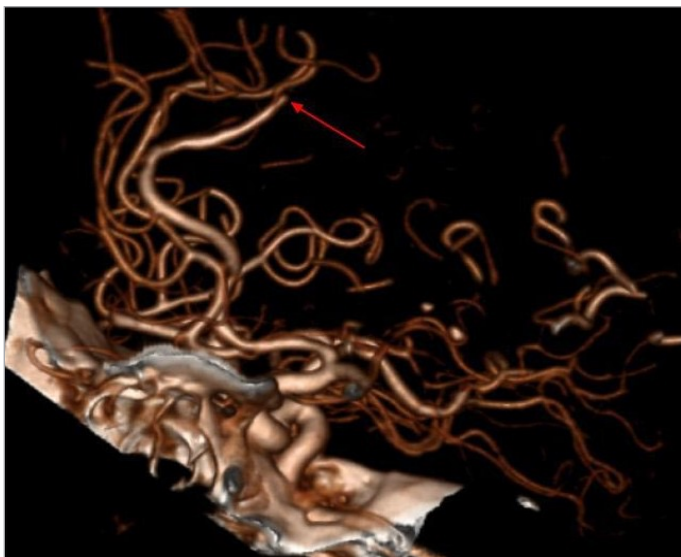


Figure 2. Angiography showing the anatomical variation of the ACA segment bifurcation.

[Abstract:2039]

THE FREQUENCY OF CAROTID ARTERY DISEASE IN PATIENTS WITH HYPERTENSION AND ASSOCIATION WITH URIC ACID LEVELS

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Background: Carotid artery disease (CAD) is a significant cause of morbidity and mortality. Several studies have shown a positive correlation between cardiovascular diseases, stroke, hypertension, metabolic syndrome, diabetes mellitus, and hyperuricaemia. In this retrospective study, we aimed to evaluate the frequency of carotid artery disease due to atherosclerosis and its relationship with uric acid levels in patients with hypertension.

Materials and Methods: This study included 166 patients aged 18 years and older with hypertension who were screened for carotid artery disease by carotid Doppler ultrasonography. Patient information was obtained from patient files and the hospital automation system.

Results: The mean age of 166 patients with hypertension included in the study was 57.67 ± 13.70 years. Of these patients, 45.8% (n=76) were male and 54.2% (n=90) were female and the mean age of hypertension was 9.49 ± 9.45 years. The mean uric acid value was 5.58 ± 1.68 mg/dL. A significant positive correlation was found between carotid artery disease and uric acid value (p: 0.017). A significant positive correlation was also found between age at onset of hypertension and carotid artery disease (p < 0.01).

Conclusions: Hypertension, hypercholesterolaemia, diabetes, smoking, increased body mass index, and age are the main risk factors for CAD. Severe CAD is closely associated with the development of stroke and other cerebrovascular events. Early detection of elevated uric acid and early treatment of hyperuricaemia is crucial in the early diagnosis and treatment of hyperuricaemia-related diseases such as stroke, metabolic syndrome, and cardiovascular disease.

Keywords: carotid artery disease, hypertension, uric acid

Characteristics		N (%)
Gender	Male	76 (45.8)
	Female	90 (54.72)
Age	Mean ± SD	
	57.67 ± 13.70	
Age at onset of Hypertension	Mean ± SD	
	9.49 ± 9.45	
Laboratory findings		Mean ± SD
Glucose (mg /dL)		112.95 ± 37.46
Uric acid		5.58 ± 1.68
HbA1c (%)		6.1 ± 1.37
Total cholesterol (mg /dL)		204.07 ± 49.11
LDL (mg /dL)		125.99 ± 41.37
HDL (mg /dL)		48.9 ± 13.71
Triglyceride (mg /dL)		155.23 ± 75.193

Table 1. Baseline demographic information and laboratory parameters of the study population.
Abbreviations: HbA1c, glycated haemoglobin; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol.

IMC			
Findings		All Population	Ratio (%)
CA-DUSG	Normal	42	25,3
	IMC	33	19,9
	≤ %49 stenosis	48	28,9
	% 50-69 stenosis	25	15,1
	% 70-79 stenosis	12	7,2
	% 79-100 stenosis	6	3,6
	Total	166	100,0

Table 2. Carotid artery doppler USG findings and ratios.
Abbreviations: CA-DUSG, Carotid artery doppler ultrasonography; IMC, Intima media thickening.

[Abstract:2046]
**DESCRIPTIVE ANALYSIS OF PATIENTS
DIAGNOSED WITH NON-CONVULSIVE
STATUS EPILEPTICUS IN A TERTIARY
HOSPITAL**

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This retrospective analysis delves into the clinical characteristics, therapeutic approaches, and outcomes of patients diagnosed with non-convulsive status epilepticus (NCSE) through electroencephalogram (EEG) within a tertiary-level hospital setting. The study encompassed 39 patients, of whom 24 exhibited EEG-confirmed NCSE. Within this subset, 70.8% were female, with an average age of 73.8 years. Noteworthy factors included a significant percentage with a history of epilepsy, dementia, central nervous system structural abnormalities, and outpatient treatment involving psychotropic drugs. Compelling findings emerged, revealing interruptions in chronic benzodiazepine and opioid therapies with hospitalization. Systemic infection was reported in 62.5% of cases, accompanied by prevalent ionic imbalances, renal dysfunction, and hepatic abnormalities. All patients demonstrated impaired consciousness, with 75% developing negative symptoms (such as aphasia, amnesia, and catatonia) and 54.2% experiencing positive symptoms (including myoclonus, nystagmus, and automatisms). Treatment protocols incorporated benzodiazepines and antiepileptic drugs, predominantly levetiracetam. Despite a 58.3% favourable clinical response, a strikingly high mortality rate of 58.3% was observed. In conclusion, this study underscores the intricate and multifaceted nature of NCSE, shedding light on the prevalence of comorbidities and the challenges associated with therapeutic management. Although positive clinical responses were evident in a considerable percentage of cases, the study emphasizes the urgent need for a comprehensive understanding of NCSE to refine therapeutic strategies and ultimately improve patient outcomes.

Keywords: non-convulsive status epilepticus, electroencephalogram, antiepileptics, tertiary hospital

	N = 24
Mean age	73,8
Gender	
Male	7 (29,2%)
Female	17 (70,8%)
PH epilepsy	4 (16,7%)
PH convulsive/non-convulsive status epilepticus	1 (4,2%)
PH dementia	5 (20,8%)
PH structural CNS abnormalities	6 (25%)
Tumor	2 (8,3%)
Post-Traumatic Brain Injury	1 (4,2%)
Ischemic stroke	3 (12,5%)
Previous surgical intervention	1 (4,2%)
Home psychotropic treatment	12 (50%)
SSRIs	8 (66,6%)
Other	4 (33,3%)
Antibiotic treatment on admission (prior to NCSE development)	16 (66,7%)
Cefepime	9 (37,5%)
Quinolones	4 (16,7%)
Discontinuation of habitual benzodiazepine treatment	7 (29,2%)
Discontinuation of habitual opioid treatment	10 (41,7%)
Presence of systemic infection	15 (62,5%)
Ionic disturbances	14 (58,3%)
Hypercalcemia	3 (12,5%)
Hypocalcemia	2 (8,3%)
Hyperkalemia	6 (25%)
Hypokalemia	5 (20,8%)
Hypomagnesemia	1 (4,2%)
Other	11 (45,8%)
Impaired renal function (GFR <40)	13 (54,2%)
Impaired liver function	7 (29,2%)
Impaired level of consciousness	24 (100%)
Negative symptoms	18 (75%)
Positive symptoms	13 (54,2%)
Benzodiazepine treatment post-diagnosis	22 (91,7%)
Antiepileptic drug (AED) treatment post-diagnosis	21 (87,5%)
Phenytoin	4 (17,4%)
Valproic acid	11 (45,8%)
Levetiracetam	20 (83,3%)
Lamotrigine	11 (45,8%)
Other	2 (8,3%)
Lumbar puncture performed	10 (41,7%)
Control EEG performed	17 (70,8%)
Electroencephalographic improvement	12 (70,6%)
Worsening or absence of EEG changes	5 (29,4%)
Positive clinical response	14 (58,3%)
Deceased	14 (58,3%)

PH: personal history, CNS: central nervous system, TBI: traumatic brain injury, NCSE: non-convulsive status epilepticus, AEDs: antiepileptic drugs, EEG: electroencephalogram

Table 1.

[Abstract:2078]
HAEMATOMA WITHIN A NEUROFIBROMA

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We present a 46-year-old woman with neurofibromatosis type 1 (NF1), admitted due to oedema, pain and functional limitation of the left lower limb, that appeared the day before. Analytically, acute anaemia was found, requiring transfusion. The emerging echo doppler and CT angiography showed a heterogeneous area on the postero-external aspect of the thigh (35 longitudinal x 9 anteroposterior x 16 centimetres transverse) and in its thickness

an arterial ectasia with active haemorrhage. Arteriography confirmed a pseudoaneurysm in the terminal branch of the deep femoral artery and the haemorrhage was treated by embolization. Neurofibromas are one of the main features of NF1. Half of patients have vascular anomalies, but haemorrhage is rare, however it can cause significant morbidity and mortality.

Keywords: neurofibromatosis type 1, pseudoaneurysm, acute anaemia

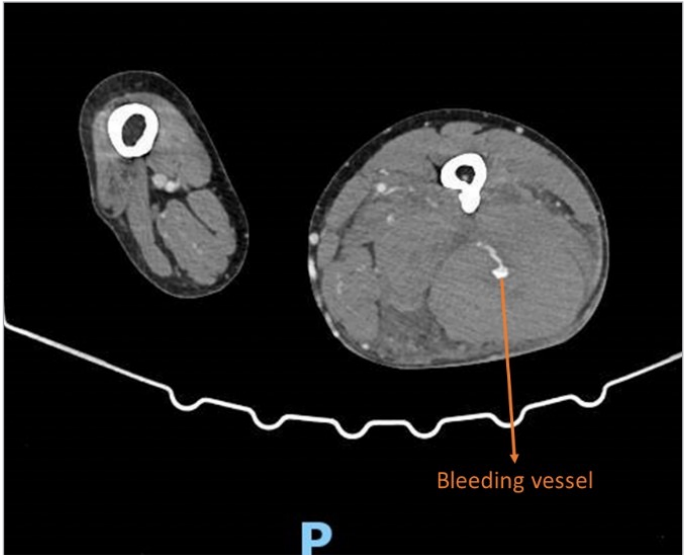


Figure 1. Bleeding vessel.

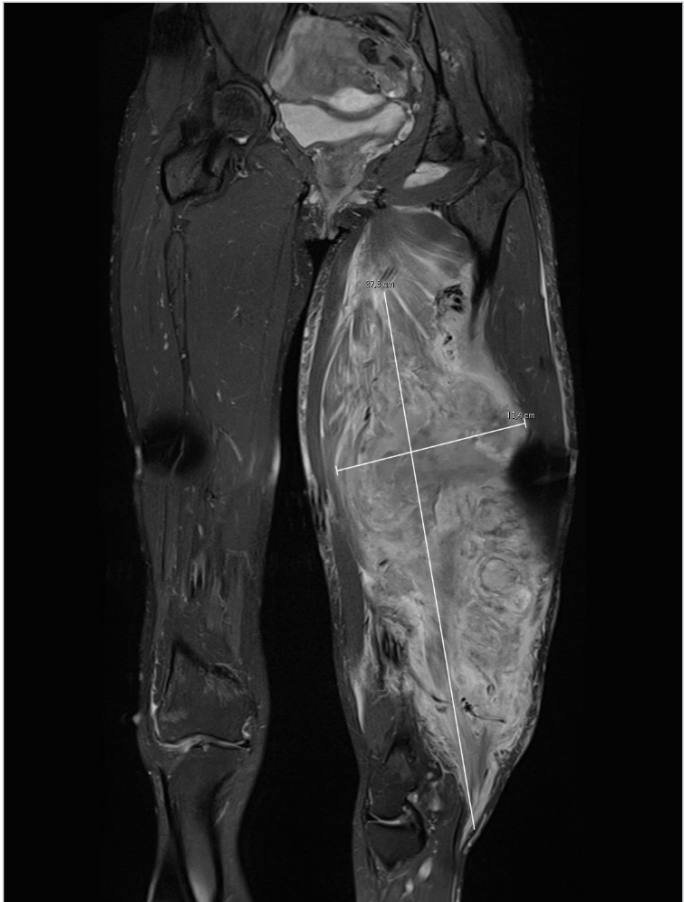


Figure 2. Magnetic resonance.



Figure 3. At discharge from hospital stay.

[Abstract:2204]

CENTRAL VENOUS THROMBOSIS, A RARE CAUSE OF HEADACHE: RELATING TO A SPECIFIC CASE

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Case Description: 20-year-old woman, no allergies or toxic habits. No oral contraceptives.

Cousin with scleroderma and previous episode of thrombosis in the arm. obesity. She suffered from migraine with aura since she was 13 years old. She consulted the emergency department on three occasions due to numbness in the left hemibody of <1 h of evolution, intense posterior headache with blurred vision that interrupted night rest. The patient had taken NSAIDs and the pain did not stop. A cranial CT scan with contrast was performed showing bilateral extensive central venous thrombosis. Platelets $248000 \times 10^9/L$, fibrinogen 415 mg/dL, D-dimer 2090 ng/ml and renal function normal. Enoxaparin 100 mg/12 h was started. Control MRI with partial recanalization of venous sinuses. During admission, she reported bilateral difficulty in focusing on objects and an eye fundus examination was performed without papilledema or haemorrhage data. Negative viral PCR, negative autoimmunity and antiphospholipid antibodies, normal thoracoabdominal CT, negative genetic study and negative test for paroxysmic nocturnal haemoglobinuria. The patient was switched to sintrom with favourable clinical evolution. A repeat thrombophilia study was performed three months later that was negative.

Clinical judgment: extensive central venous thrombosis of uncertain cause.

Discussion: Venous sinus thrombosis is a rare cerebrovascular disease (<0.5%) that can affect to any age but is more frequent in women and young people. The most frequent symptom is headache accompanied by other symptoms similar to those of stroke. It usually has sudden onset and prevents night rest. It

is a neurological emergency and early treatment is extremely important to avoid worse consequences. The prognosis in most cases is favourable with antithrombotic treatment.

Keywords: headache, cerebral venous thrombosis, intracranial pressure

[Abstract:2224]

ELECTROCARDIOGRAPHIC PREDICTORS OF ATRIAL FIBRILLATION IN PATIENTS WITH CRYPTOGENIC STROKE AND PROLONGED MONITORING USING IMPLANTABLE HOLTER

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Objectives: In this study, we present our experience and results in patients with cryptogenic stroke who underwent monitoring with an insertable Holter, focusing on the presence of atrial extrasystole and atrial tachycardia in previous recordings.

Materials and Methods: We retrospectively studied patients with cryptogenic stroke who had a subcutaneous Holter (Reveal LINQTM, Medtronic®) implanted from October 2017 to March 2023 at our centre. Patients who had undergone a 24-hour Holter prior to implantation were selected.

Results: During this period, 101 devices were implanted (mean age 73.5 ± 11.9 years, 40.4% women; 22.2% TIA) with a mean follow-up of 656.2 ± 331.0 days; the median time to implantation was 9 (IQR 6-14) days. In 28 patients (mean age 73.1 years, 35.8% women), a 24-hour Holter was performed before implantation. Of these, 12 out of 28 had frequent supraventricular extrasystole (>350 beats/24 hours), and 16 had <350 beats/24 hours. 100% of patients with frequent supraventricular extrasystole developed fibrillation during follow-up. 10 out of 16 patients with infrequent extrasystole developed fibrillation. Of these 10 patients, 8 had atrial tachycardia, while none of those who did not develop fibrillation had atrial tachycardia in the previous Holter.

Conclusions: The presence of frequent supraventricular extrasystole (>350 ESV/24 hours) in previous monitoring was significantly associated with the development of atrial fibrillation during follow-up ($p = 0.012$). The presence of runs of atrial tachycardia in patients with infrequent atrial extrasystole may increase the probability of finding fibrillation during follow-up.

Keywords: atrial fibrillation, cryptogenic stroke, hidden atrial fibrillation

[Abstract:2237]

THE IMPORTANCE OF ECHOCARDIOGRAPHY IN ACUTE ISCHEMIC STROKE PATIENTS: A CASE REPORT OF ATRIAL FIBRILLATION AND LEFT ATRIAL MYXOMA

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Acute ischemic stroke is a common medical emergency with diverse underlying causes. Identifying these causes is crucial for appropriate treatment and prevention of recurrent events. Here, we present the case of a 79-year-old male who experienced an acute ischemic stroke and the subsequent discovery of newly diagnosed atrial fibrillation and a left atrial myxoma.

The patient arrived at the emergency department following a loss of consciousness and fall. Neurological examination revealed dysarthria, mild left facial weakness, and an alert but disoriented state. Atrial fibrillation was detected upon cardiac examination, and additional investigations uncovered a left atrial myxoma. The patient had a history of arterial hypertension and irregular medical check-ups. Diagnostic assessments included head computed tomography, electrocardiogram, carotid and vertebral artery doppler, transthoracic echocardiogram, transoesophageal echocardiogram, and coronary angiography were conducted. These tests confirmed the presence of an acute ischemic infarct, atrial fibrillation, and a left atrial myxoma, highlighting the complexity of the case.

This case underscores the importance of thorough evaluation, including transthoracic echocardiography, in patients presenting with acute ischemic stroke, even in the presence of primary risk factors. Early detection of underlying heart conditions, such as myxomas, is vital to prevent major complications.

Clinicians should remain vigilant in their pursuit of comprehensive diagnostic approaches for stroke patients.

Keywords: ischemic stroke, atrial fibrillation, myxoma, transthoracic echocardiography

[Abstract:2243]

ATRIAL FIBRILLATION, HYPOCOAGULATION AND THE PREVALENCE OF ACUTE CEREBROVASCULAR EVENTS

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Summary: Atrial fibrillation (AF) represents an important risk factor for major cerebrovascular events. Therefore, it is important to correctly hypocoagulate these patients as primary prevention of such events, even though it may increase the risk of haemorrhagic stroke (HS) occurrence.

Purpose: To study the impact of hypocoagulation in ischemic and haemorrhagic cerebrovascular events occurrence among patients with AF.

Methods: A cross-sectional study was undertaken in a tertiary Portuguese hospital (2021-2023). Patients admitted to internal medicine ward with a diagnosis of stroke were included and divided between ischaemic [ischaemic stroke (IS) and transient ischaemic attack (TIA)] and haemorrhagic [haemorrhagic stroke (HS)] events. We analysed which patients had AF and if they were hypocoagulated or not. Data analysis was performed using univariate statistics (IBM SPSS v.20.0).

Findings: A total of 110 patients were included, with 56.4% (n=62) being male and with a mean age of 75.9±6.6 years old. Most of the sample (75.5%; n=83) had IS, followed by TIA (17.2%; n=19) and lastly HS (7.3%; n=8). About 73.6% (n=81) of patients had AF and from those, there was 33.3% (n=27) that were not hypocoagulated and had an ischaemic event, while only 4.9% (n=4) that were hypocoagulated and had an HS.

Conclusions: As expected, ischemic events represent the vast majority of strokes. Lack of hypocoagulation seems to be a more relevant factor in ischemic events than when it is present in haemorrhagic events. We conclude that hypocoagulation should be considered the optimal choice for patients with AF.

Keywords: atrial fibrillation, oral anticoagulation, acute cerebrovascular event

[Abstract:2298]

A CORRECT MEDICAL RECORD, THE KEY TO SUCCESS

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A 52-year-old woman who was admitted with nausea, vomiting and diarrhoea of 48 hours of evolution, generalized weakness, confusion and later agitation with tremor and muscle rigidity. She had no allergies, worked in the hotel business, and took omeprazole 20 mg at breakfast. On examination, disconnected from the environment; auscultation and palpation of the

abdomen showed no abnormalities and no alterations in the lower limbs were observed. On examination, we found that she was disconnected from the environment; auscultation and palpation of the abdomen showed no abnormalities, and no alterations were observed in the lower limbs. She presented isochoric pupils with vertical nystagmus, hyperreflexia, muscle spasms with doubtful Trousseau's sign, frequent postural changes in bed and choreoathetotic movements. We requested blood tests with biochemistry, hemogram, coagulation, thiamine, B12 and folic acid levels, ions, venous gasometry, cranial CT scan and lumbar puncture. Laboratory tests showed hypokalemia and severe hypomagnesemia.

She presented clinical improvement 24 hours after starting replacement therapy. After evaluation of this case, we concluded that the main cause was hypomagnesemia induced by chronic use of omeprazole and daily alcohol consumption, precipitated by profuse vomiting and diarrhoea. Hypomagnesemia is usually accompanied in up to 60% of cases by hypokalaemia and, to a lesser extent, hypocalcaemia. The most common signs and symptoms are vertical nystagmus, epileptiform episodes, choreoathetotic movements and, due to its frequent association with hypocalcaemia, tetany with positive Chvostek and Trousseau's sign. A high index of suspicion and rapid magnesium replacement helped us to confirm the diagnosis.

Keywords: omeprazol, trousseau, hypomagnesemia

[Abstract:2318]

SILENT CASE OF A GIGANTIC HIGH-GRADE GLIOMA IN THE LEFT TEMPOROPARIETAL REGION OF THE BRAIN

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High-grade gliomas are malignant, rapidly progressive primary brain tumours, being isocitrate dehydrogenase (IDH) wildtype glioblastoma the most common in adults, as well as the most fatal. Symptomatic presentation of brain tumours is non-specific, depending upon the location and size. A 54-year-old-man with history of drug abuse and hepatitis C presented to the ER after being found unconscious with loss control of his bladder and bowel, probably related to a postictal state. In retrospect, he was reported of having inappropriate behaviour assumed as a consequence of drug abuse. On evaluation he had global aphasia and grade 4 right hemiparesis. MRI showed large left temporal lesion with a central soft tissue component and multiple peripheral cystic areas, with extensive peri-lesional vasogenic oedema and signs of hydrocephalus, consistent with a primary brain tumour of high grade. Patient was referred to Neurosurgery where a resection of the mass was performed. Postoperative MRI showed complete lesion resection with no complications, maintaining

global aphasia and grade 4 hemiparesis. Pathology revealed wildtype IDH glioblastoma with C228T mutation of TERT gene promoter related with some resistance to radiotherapy. He was referred to neuro-oncology but missed all appointments with no follow-up available. We present the case of a 54-year-old man late diagnosed with a large glioblastoma rarely seen nowadays in western countries, probably consequence of drug abuse that mistaken any neurologic signs and symptoms. Despite recent progress, glioblastoma remains an incurable tumour with survival under a year and a half. In appropriately selected patients, aggressive surgery may relieve symptoms and prolong survival.

Keywords: high-grade glioma, glioblastoma, IDH, C228T mutation, TERT gene

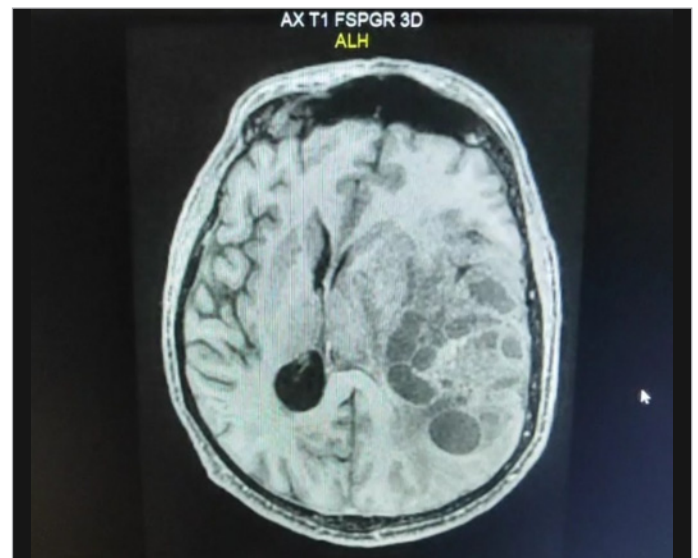


Figure 1. Axial view of the MRI showing lesion in the left temporoparietal region. In this figure se can see the large tumor with signs of hydrocephalus.

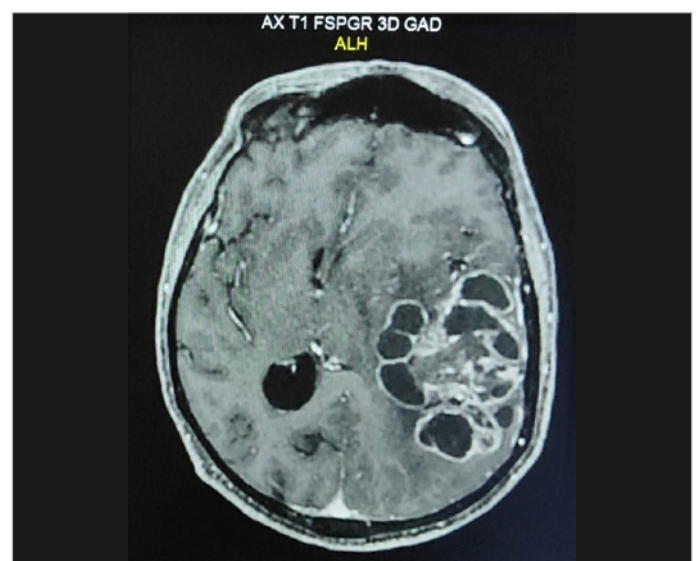


Figure 2. Axial view of contrast-enhanced MRI showing lesion in the left temporoparietal region. In this figure we can ser the intense rim enhancement with central clearing typical from glioblastomas.

[Abstract:2403]

RETROBULBAR OPTIC NEURITIS REVEALING SYSTEMIC LUPUS ERYTHEMATOSUS

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Introduction: Systemic lupus erythematosus (SLE) is a multi-system autoimmune disease, characterized by the production of antinuclear autoantibodies. Episodes of optic neuritis are rarely part of the SLE picture. This is a diagnostic and therapeutic emergency. We report the case of retrobulbar optic neuritis revealing SLE.

Observation: Patient E.S aged 24, with no previous history, admitted to our department for the exploration of a reduction in visual acuity, with pain on ocular mobilization. Orbital-cerebral MRI revealed active left retrobulbar optic neuritis with white matter signal abnormalities. The clinical and paraclinical examination were in favour of SLE, with an ACR-EULAR score of 15 points. The patient received boluses of methylprednisolone (1g/day, 3 days in a row), followed by oral corticosteroid therapy at a dose of 1mg/day.

Results: Although no criteria for SLE include ocular involvement, optic neuritis is present in a third of patients.

In the literature, the evolution of ocular damage under treatment is variable: favourable if corticosteroid therapy was started early, unfavourable with blindness if treatment was started late.

Conclusions: RON is a rare, exceptionally revealing, but potentially serious complication of SLE, it is a diagnostic and therapeutic emergency. Corticosteroid therapy has revolutionized visual prognosis.

Keywords: retrobulbar optic neuritis, systemic lupus erythematosus, optic neuropathy

[Abstract:2447]

HEALTH-RELATED QUALITY OF LIFE AND ITS PREDICTORS AMONG PEOPLE LIVING WITH EPILEPSY, KHARTOUM STATE, SUDAN: A CROSS-SECTIONAL STUDY

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Background: Epilepsy causes a significant psychological and social impact on daily living conditions and usually has lifelong consequences for the patient and family. Measuring the quality of life (QOL) of people with epilepsy (PWE) is increasingly recognized as an important component of clinical management. The QOL measures differ between countries and there is limited information regarding PWE in Sudan. The aim of this study was to determine the health related QOL and its related factors in PWE in Omdurman city Outpatient Clinics.

Methods: A total of 75 adults with epilepsy attending Various neurology outpatient clinic in Omdurman city were interviewed in this cross-sectional study. The QOL was measured using a validated Arabic translated version of the quality of life in epilepsy scale-31 (QOLIE-31).

Results: The mean age was 27.46 (11.59) years old (standard deviation (SD) 11.59) and 52% were females. The mean total score of QOLIE-31 was 61.72 (SD15.13). The highest subscale score was the overall quality of life, 67.33 (19.54) and the lowest was seizure worry subscale, 43.41 (SD 31.22). The subscales of seizure worry, and overall quality of life showed significant mean difference among employed and unemployed individuals P value <0.05.

Conclusions: This study stress on the importance of seizure control for a better QOL in Sudanese patients with epilepsy. Worrying about seizure had the major contribution on QOL.

Keywords: epilepsy, quality of life, QOLIE-31, control of seizures, SUDAN

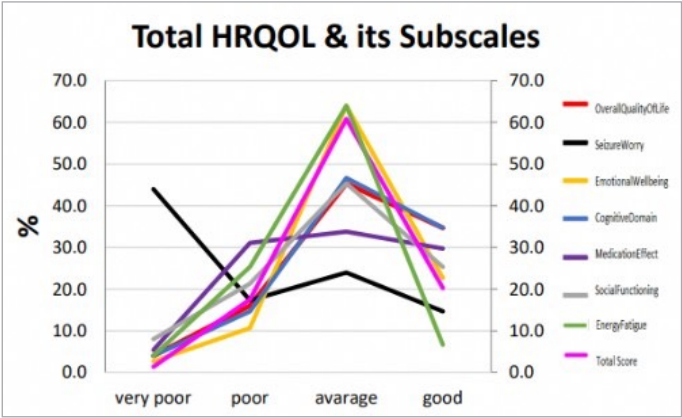


Figure 1. Total HRQOL and its subscales.

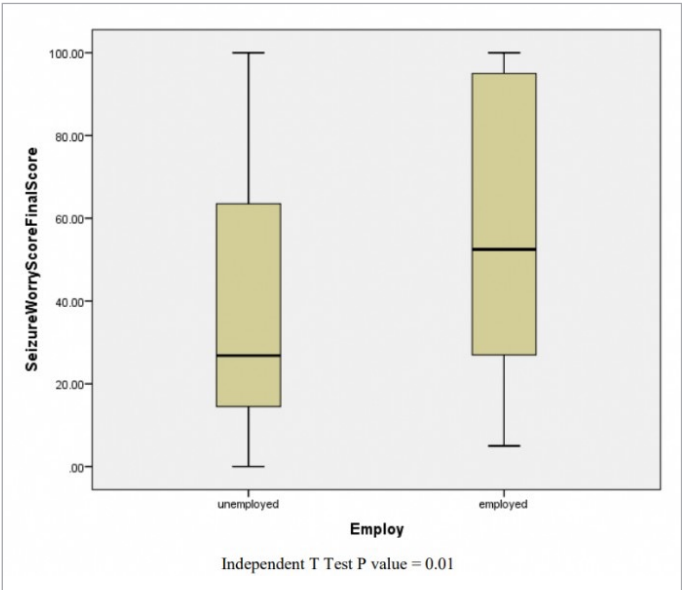


Figure 2. Seizure worry sub score and participant employment (mean plot).

Sub-Scales of QOLIE-31	Number of Items	Observed Range	Mean (SD) score	Reliability Cronbach's Alpha
Seizure worry	5	0-100	43.41 (31.22)	0.85
Overall Quality of life	2	5-100	67.33 (19.54)	0.46
Emotional Wellbeing	5	20-100	64.37 (15.90)	0.71
Energy /Fatigue	4	10-90	58.59 (14.57)	0.49
Cognitive Functioning	6	9-100	64.24 (21.51)	0.83
Medication Effects	3	0-100	63.32 (26.05)	0.79
Social Functioning	5	0-100	62.09(24.76)	0.86
Subjective Overall Health (item-31 visual analogue scale)		24.24 - 95.80	61.72(15.13)	

Table 1. Total score of QOLIE-31 sub-scales.

[Abstract:2532]
RETROBULBAR OPTIC NEURITIS

Hajdsadok Abdelhamid Mohamed, Mokhtar Malika, Taleb Abdelhalim, Elayadi Nazli, Djebbar Yousra, Aksas Wafa, Bachir Cherif Abdelghani
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Results: Although no criteria for SLE include ocular involvement, optic neuritis is present in a third of patients. In the literature, the evolution of ocular damage under treatment is variable: favourable if corticosteroid therapy was started early, unfavourable with blindness if treatment was started late.

Conclusions: RON is a rare, exceptionally revealing, but potentially serious complication of SLE, it is a diagnostic and therapeutic emergency. Corticosteroid therapy has revolutionized visual prognosis.

Keywords: retrobulbar optic neuritis, systemic lupus erythematosus, optic neuropathy

[Abstract:2588]
PALPEBRAL PTOSIS AND DYSPHAGIA, IS THERE A RELATIONSHIP?

Marina Andrea Martínez Vacas, Lourdes Porras Leal, Ana García Pérez, María José Redondo Urda, Beatriz La Rosa Salas, Isabel Carmona Moyano, Miri Kim Lucas, Sandra Cruz Carrascosa, Beatriz González Castro
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A 73-year-old male with a history of bilateral ptosis surgeries and lumbar canal stenosis presents to the emergency room with chest pain. Admitted with a community-acquired pneumonia diagnosis, further investigation reveals a two-year history of progressively worsening oropharyngeal dysphagia, substantial weight loss, and a family history of unexplained dysphagia-related deaths. Neurological examination uncovers various abnormalities, including gaze limitations, bilateral sixth cranial nerve palsy,

tongue atrophy, hypophonia, and bilateral shoulder girdle weakness. Diagnostic tests include normal cranial CT, a CT scan revealing muscle fat degeneration, brain MRI displaying diffuse fat infiltration in tongue and mouth muscles, negative amyloidosis, and autoimmune studies, and fibroscopy indicating saliva retention. Gastroscopy reveals chronic gastritis. Neurophysiological studies support a generalized myopathy, predominantly affecting facial muscles. Given these findings, a genetic study confirms the patient as a heterozygous carrier of the gene associated with oculopharyngeal muscular dystrophy (OPMD).

OPMD is a rare myopathy characterized by ptosis and dysphagia, typically emerging in midlife. As the disease progresses, additional symptoms may include impaired upward gaze, tongue weakness, chewing difficulties, dysphonia, and weakness in facial and limb muscles, predominantly in the lower limbs.

Keywords: ptosis, dysphagia, oculopharyngeal muscular dystrophy

[Abstract:2595]

CLINICAL INSIGHT INTO WERNICKE'S ENCEPHALOPATHY: A CASE STUDY

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Introduction: Wernicke-Korsakoff syndrome is a neurological disorder that arises from a severe deficiency of vitamin B1 and its presentation depends on clinical condition progression. Wernicke encephalopathy (WE), a medical emergency characterized by mental confusion, ophthalmoplegia, and gait ataxia, can often be difficult to distinguish from other conditions such as delirium. This critical situation requires emergent treatment to prevent chronicity and irreversible neurological damage. Although often linked to chronic alcoholism, can also stem from malnutrition.

Case: A 63-year-old woman with a gastric adenocarcinoma underwent subtotal gastrectomy (meanwhile dismissing follow-up procedures) and chronic liver disease due to alcohol use disorder, was brought to the Emergency Department by her neighbour who observed significant functional deterioration - reduced hygiene-care, confused speech and frequent falls.

Neurological examination portrayed temporo-spatial disorientation, left spontaneous nystagmus and imbalance. Cerebral angiography-computed tomography had no changes that could justify the symptoms. According to clinical history, WE was suspected and a brain magnetic resonance was performed, revealing hyperintensity in the mammillary bodies, a typical finding of this disease. Despite treatment with thiamine supplementation

since admission, neurological alterations such as confusion, nystagmus and gait instability have persisted, which is why it was considered that the patient would benefit from a rehabilitation program in order to recover some of her previous autonomy.

Discussion: The diagnosis of WE is difficult to confirm and, untreated, most patients progress to critical neurological damage, resulting in coma and death. Despite mental confusion that may persist for weeks, prompt administration of thiamine leads to significant improvement in deficits.

Keywords: Wernicke encephalopathy, chronic alcoholism, deficiency of vitamin B1 (thiamine)

[Abstract:2607]

CLINICAL AND IMMUNOLOGICAL CHARACTERISTICS OF MONOCLONAL GAMMOPATHY IN NEUROLOGICAL PRACTICE

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Purpose: Monoclonal gammopathies (MG) are a heterogeneous group of diseases with a variable clinical presentation and different aetiologies. The aim of our study was to describe clinical, immunological and etiological characteristics of MG in neurological practice.

Methods: During a period of 12 years, 446 immunofixation tests (Hydragel, Sebia®) were performed for patients followed in the neurology department. Cases of MG identified by this technique were enrolled. Clinical data were collected from patients' medical files.

Findings: A MG was identified in 35 patients (sex-ratio:1,9; 21-80 years). MG corresponded to complete immunoglobulins (n=31: IgG:21 cases, IgA:6 cases, IgM:3 cases, IgD:1 case) or free light chains (n=4). MG was already known in 3 cases and was identified in the neurology department in the other cases. Neurological manifestations included neuropathies (48%), amyotrophic lateral sclerosis (14%), cerebral vascular stroke (10%) and medullar compression (7%). Malignant aetiologies of MG were: multiple myeloma (n=4), plasmacytoma (n=2), POEMS syndrome (n=2), Waldenstrom disease (n=2), AL amyloidosis (n=1) and B lymphoma (n=1). MG was classified "of undetermined significance" in 55% of cases.

Conclusions: Detection of a MG in neurology practice is not rare. The association with a peripheral neuropathy is well established, however it isn't always obvious to prove the causality link. Other neurological manifestations can be concomitant to MG detection; however, the association need to be confirmed. Some MG considered as «of undetermined significance» could correspond

to MG of «neurological significance». A multidisciplinary approach is needed to adequately handle both the neurological disorder and the MG.

Keywords: monoclonal gammopathy, neuropathy, immunofixation

[Abstract:2628]

ADHERENCE TO AN INTEGRATED CARE PATHWAY FOR STROKE IS ASSOCIATED WITH LOWER RISK OF MAJOR CARDIOVASCULAR EVENTS: A REPORT FROM THE ATHENS STROKE REGISTRY

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Background: Recently the European Society of Cardiology Council on Stroke proposed a holistic integrated care approach for stroke patients.

The impact of implementing the ABCstroke pathway ‘concept’ on clinical outcomes has never been estimated before. In order to investigate the potential effect of ABCstroke pathway adherence to cardiovascular outcomes post stroke, we performed a post-hoc analysis from the Athens Stroke Registry.

Methods: This analysis was performed in the Athens Stroke Registry, which includes all consecutive patients with acute first-ever ischemic stroke. Kaplan-Meier product limit was used to estimate the cumulative hazard of each outcome according to adherence with the ABCstroke pathway.

Results: We studied 2,513 patients [median (IQR) age 71 (62-78) years; 37.7% female] with ischemic stroke with median follow-up period of 30 (6-75) months.

Full adherence to the ABC pathway was identified in 156 (6.2%) of the patients, while 192 (7.6%) did not adhere to any of the therapeutic pillars of ABCstroke.

Full adherence to ABC treatment pathway was associated with significant reduction of stroke recurrence, compared to patients with no or partial adherence (aHR: 0.61; 95%CI: 0.37-0.99), as well as a lower risk of MACE (HR: 0.59; 0.39-0.88) and death (aHR: 0.22; 95%CI: 0.12-0.41).

Conclusions: Full adherence to the ABCstroke pathway based on the current guidelines was evident in 6.2% of our ischaemic stroke cohort but was independently associated with lower risks of stroke recurrence, major cardiovascular events and mortality. This highlights a potential opportunity to improve clinical outcomes

post-stroke with a holistic or integrated care management approach.

Keywords: stroke, major adverse cardiovascular event, holistic management

[Abstract:2755]

LACUNAR STROKE PRESENTING AS AN ISOLATED UPPER MOTOR NERVE (UMN) FACIAL PALSY: A RARE MANIFESTATION OF ACUTE ISCHEMIC STROKE

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Introduction: Lacunar strokes result from small non cortical infarcts due to occlusion of a single penetrating branch of a large cerebral artery (1). Pure motor hemiparesis with face-arm-leg weakness is the commonest presentation accounting for about 45% of the presentations (2). This is a rare case of isolated UMN facial palsy presenting as an ischemic lacunar infarction.

Case Presentation: A 69-year-old male presented with right sided mouth deviation and drooling of saliva from left side for two days duration. He was a known patient with hypertension. There was no arm/leg weakness, sensory impairment, dysphagia, dysphonia, diplopia, or inability to close eyelids. Further, no history of antecedent fever, trauma, or seizures.

On examination, the Glasgow coma scale was 15/15. Blood pressure was 160/90 mmHg with a pulse rate of 80 bpm which was irregular. Neurological examination revealed a left UMN facial palsy. There were no other focal neurological signs.

Electrocardiogram revealed a rate controlled atrial fibrillation (AF). Non-contrast computed tomography (NCCT) of the brain revealed a subacute lacunar infarction in the right periventricular white matter (Figure 1).

He was treated with dual antiplatelets, statins and physiotherapy. Anticoagulation was deferred for two weeks.

Discussion: Isolated UMN is a rare manifestation of lacunar stroke that can easily be missed (3-5). Such isolated lesions can occur in the periventricular region infarcts where corticospinal tracts are not densely packed as in the internal capsule (6). This case highlights the importance of clinical vigilance for early detection of such rare stroke syndromes.

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Keywords: isolated UMN facial palsy, lacunar infarction, ischemic stroke

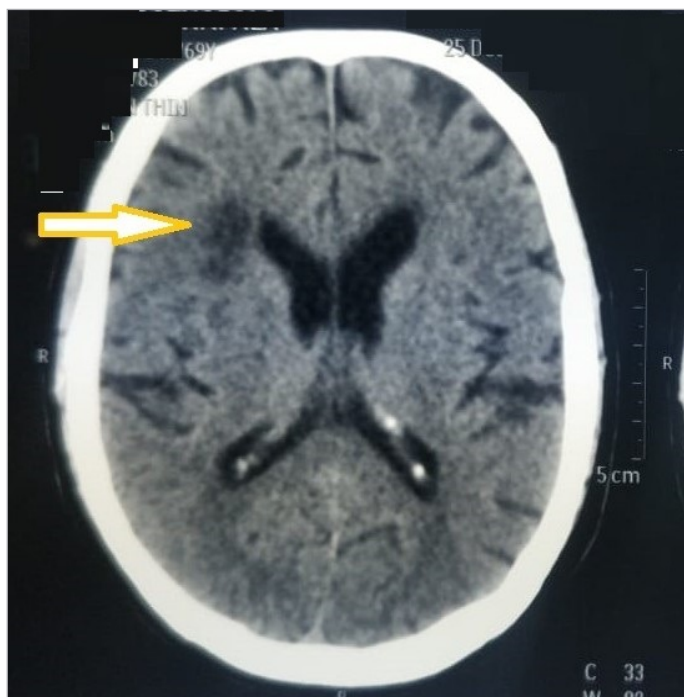


Figure 1. NCCT brain showing right periventricular infarction (arrow).

[Abstract:2762]

UNVEILING THE ENIGMA: CONCURRENT PRES AND SIADH, AN UNCERTAIN ETIOLOGY

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A 76-year-old woman with a history of poorly controlled hypertension consulted for asthenia and headache for 2 weeks. In the physical examination we remarked a high blood pressure reading (170/75 mmHg) and bradypsychia without any discernible

neurological abnormalities along the central nervous system pathways.

Blood tests revealed a sodium level of 126 mEq/L, a plasma osmolality of 265 mOsm/kg, with a urinary sodium level of 62 mEq/L and a urinary osmolality of 490 mOsm/kg. After ruling out adrenal and thyroid insufficiency (TSH 0.73 mU/l and cortisol 33.3 µg/dl), a diagnosis of syndrome of inappropriate antidiuretic hormone secretion was performed.

The etiological study was completed with a body CT scan that ruled out paraneoplastic causes, and a brain MRI was performed (Figure 1) which showed findings compatible with PRES syndrome. After undergoing treatment involving antihypertensive medication, water restriction and urea, the patient presented a disappearance of symptoms and a resolution of hyponatremia to values within the normal range.

PRES syndrome involves an abnormal brain perfusion leading to neurological dysfunction. It often manifests as visual disturbances and sudden blood pressure changes. A crucial aspect of diagnosis involves a heightened suspicion, especially when consistent MRI brain scan findings are observed across various scenarios.

Although there are cases described linking hyponatremia and PRES syndrome, to our knowledge, this is the first case of PRES syndrome developing within SIADH. However, it remains unclear whether SIADH is the cause or the consequence.

Keywords: PRES, SIADH, sodium, hypertension

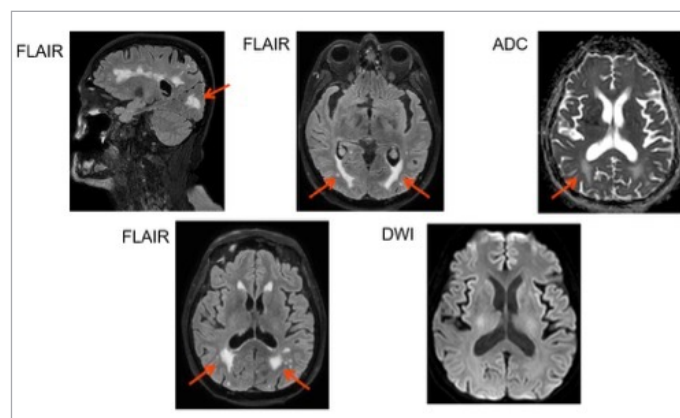


Figure 1. PRES syndrome MRI.

Multiple hyperintense foci are identified in the FLAIR sequence in the subcortical and juxtacortical white matter, with a distribution that characteristically affects both parietooccipital regions and the 'last meadow' territory (red arrows), bilaterally and symmetrically. These correspond to extensive areas of vasogenic oedema, showing no contrast enhancement or restriction in diffusion sequences.

[Abstract:2787]

CEREBRAL ANEURYSM AND CHRONIC MIGRAINE, A CHALLENGE FOR THE CLINICIAN

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Ruptured cerebral aneurysm is a serious condition that can carry an ominous prognosis if not diagnosed early. Cerebral aneurysm in patients with chronic migraine and headache due to painkiller abuse is rare, but its occurrence leads to diagnostic confusion. We present the case of an elderly female patient, smoker, who consulted multiple times for her chronic migraine attacks, with a change in her headache pattern, who required neuroimaging and endovascular therapy for a saccular aneurysm in the right anterior cerebral artery without complications. In conclusion, the role of clinical and neuroimaging are the key to diagnose possible cerebral aneurysm.

Keywords: chronic migraine, cerebral aneurysm, headache due to analgesic abuse

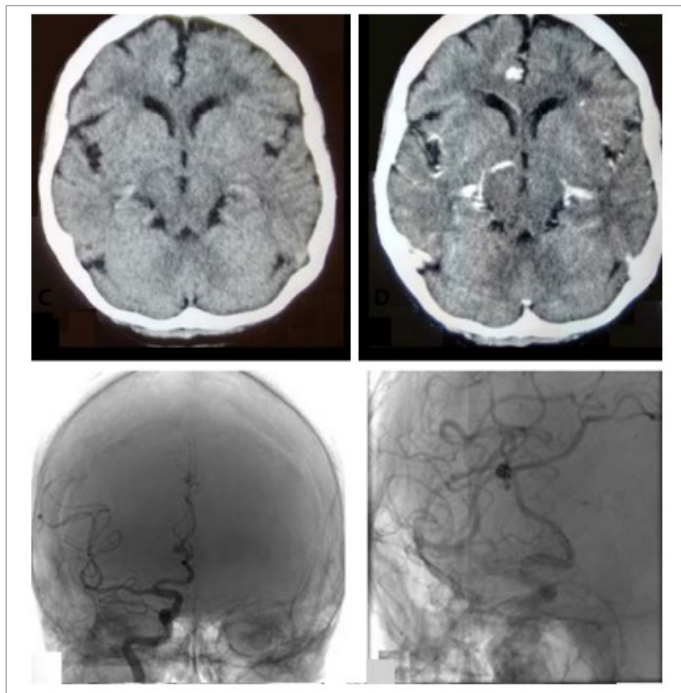


Figure 1. Imaging tests.

A: Simple CT scan, within normal limits. B: Contrast CT, with rosette or saccular aneurysm in the right anterior cerebral artery, without bleeding. C and D: cerebral angiography; following endovascular therapy with presence of coils occupying the anterior segment A3 of the anterior cerebral artery. A3 segment of the right anterior cerebral artery.

[Abstract:2789]

HASHIMOTO'S ENCEPHALOPATHY: CASE REPORT

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Hashimoto encephalopathy is an infrequent neurological entity, rarely suspected by the clinician. It is of a probable autoimmune nature, it generally presents with subacute symptoms; in thrusts and remissions, high titers of antithyroid antibodies and good response to corticosteroid treatment. We present the clinical case of a 78-year-old man with multiple pathological antecedents who consulted in Hospital Maciel, Montevideo, Uruguay for symptoms of 3 weeks of evolution characterized by psychomotor excitement, language and behavioural alterations. Other causes of encephalopathy were excluded. Clinical tests revealed euthyroidism with elevated antithyroid antibodies and the improvement of symptoms after starting corticotherapy was evidenced.

Keywords: hashimoto encephalopathy, autoimmune, corticoids

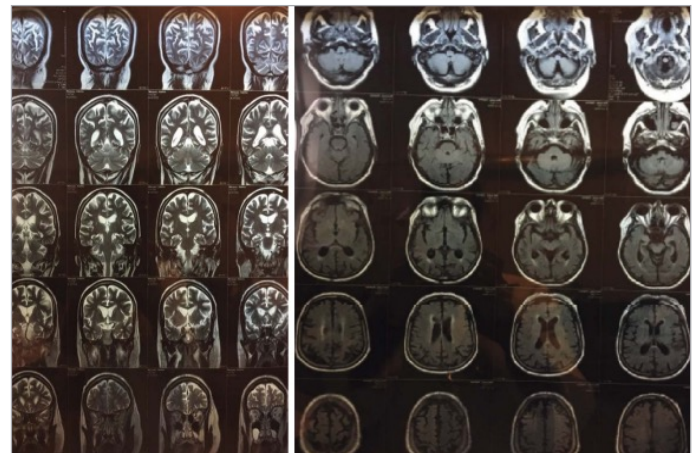


Figure 1. Magnetic resonance imaging shows compatible alterations.

[Abstract:2834]

A 57-YEAR-OLD MALE WITH WEAKNESS IN THE SCAPULAR AND PELVIC GIRDLES

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A 54-year-old male with no significant medical history is admitted due to a condition characterized by weakness in the shoulder and pelvic girdle, solid food dysphagia, lower back pain, and an inability to walk for about four days. This presentation is preceded

by a diarrheal syndrome and fever. The patient’s examination is mostly normal, except for the inability to walk and weakness in the proximal musculature of both upper and lower limbs.

Additional tests are requested:

- Complete blood work, highlighting a positive campylobacter stool culture.
- Normal cerebrospinal fluid analysis.
- Normal brain MRI.
- Lumbar-sacral MRI revealing a herniated disc at D5-D6.

Considering these findings, a differential diagnosis is pursued with the initial suspicion of inflammatory myositis secondary to GI infection (Table 1). Due to the lack of improvement, further investigation includes an electromyogram (EMG), which indicates demyelinating polyneuropathy at the motor level, symmetrically and with minimal motor axonal involvement. Ultimately, the final diagnosis is Guillain-Barré syndrome of the pure motor variant. Guillain-Barré Syndrome is a rare immune-mediated inflammatory disease affecting peripheral nerves and nerve roots. There are different variants (Table 2), with the most common being demyelinating sensorimotor involvement. The neurological manifestations can vary widely, although it typically presents as rapid and progressive bilateral weakness in the limbs. Diagnosis is primarily clinical and poses a challenge for healthcare professionals, as an extensive differential diagnosis is necessary. The interest in this case stems from the difficulty in identifying an atypical presentation of a rare disease.

Keywords: weakness, polyneuropathy, Guillain-Barré

CNS (Central Nervous System)	Anterior Horn	Nerve Roots	Peripheral Nerves	Neuromuscular Junction	Muscles
Infections	Flaccid Myelitis	Infection	Polioomyelitis (PDIC)	Myasthenia Gravis	Metabolic Disorders
Malignancy		Mechanical Compression	Metabolic Disorders	Eaton-Lambert Syndrome	Myositis Inflammatory
Mechanical Compression		Leptomeningeal Malignancy	Vitamin Deficiency	Toxins	Rhabdomyolysis
Stroke (ACV)			Toxins		Intoxication
			Vasculitis		Mitochondrial Disease
			Infection		

Table 1. Differential diagnosis of acute symmetric proximal non-painful tetraparesis with bulbar symptoms following gastrointestinal infection.

Variant	Frequency	Clinical Characteristics
Classic Guillain-Barré Syndrome	30-85	Progressive symmetric weakness in limbs, often preceded by viral or bacterial infection. Affects both motor and sensory nerves.
Pure Motor Variant Guillain-Barré Syndrome	5-70	Predominance of motor weakness without significant sensory involvement. May be more challenging to diagnose.
Paraparetic	5-10	Paresis restricted to the legs
Pharyngeal-Cervical-Brachial	<5	Weakness of the pharyngeal, cervical, and brachial muscles without weakness in the lower extremities.
Bilateral Facial Paralysis with Paresthesias	<5	Bilateral facial weakness, paresthesias, and reduced reflexes
Pure Sensory	<1	Acute or subacute sensory neuropathy without other deficits
Miller Fisher Syndrome	5-25	Classical triad of ataxia, areflexia, and ophthalmoplegia. Can follow respiratory or gastrointestinal infections.
Bickerstaff Brainstem Encephalitis	<5	Ophthalmoplegia, ataxia, areflexia, signs of the pyramidal tract, and altered consciousness, often overlapping with Guillain-Barré Syndrome (GBS) sensory-motor

Table 2. Variants of Guillain-Barré syndrome.

[Abstract:2864]

ANTITHYROID ANTIBODIES IN THE CEREBROSPINAL FLUID OF PATIENTS WITH HYPOTHYROIDISM AND NEUROLOGICAL IMPAIRMENT

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Anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-TG) antibodies (Abs) are serological biomarkers of autoimmune thyroid diseases. Their dosage in the cerebrospinal fluid (CSF) is rarely prescribed but could be justified in the context of Hashimoto encephalopathy (HE). The aim of our study was to determine CSF levels of these Abs in patients with central nervous system (CNS) impairment and hypothyroidism. We measured anti-TPO and anti-TG in paired CSF and serum samples of 10 patients with CNS impairment and hypothyroidism using an enzyme-linked-immunosorbent-assay (detection thresholds:10 U/mL and 20 U/mL, respectively). Total IgG were measured in CSF and serum to calculate specific antibody index (ASI): ratio between CSF/serum quotients for anti-TPO/TG antibodies (Qspec) and CSF/serum IgG quotient (QIgG) (normal values<1.4). Anti-TPO Abs were undetected in serum and CSF in 3 cases and detected both in serum and CSF in 7 cases. Anti-TPO ASI were comprised between 0.20 and 1.36 for these 7 patients. Anti-TG Abs were undetected in serum and CSF in 2 cases, detected only in serum in 1 case and detected both in serum and CSF in 7 cases. Anti-TG ASI were comprised between 0.10 and 0.85 for these 7 patients. The diagnosis was a probable HE (n=2), a probable limbic encephalitis (n=1), an inflammatory CNS disease (n=4), a HIV infection (n=1) or undetermined (n=2). Our results suggest that CSF antithyroid Abs derive from blood and not from an intrathecal synthesis, either for patients with a probable HE or for those with other causes of CNS impairment.

Keywords: anti-thyroid antibodies, cerebrospinal fluid, Hashimoto encephalopathy

[Abstract:2904]

ARNOLD-CHIARI TYPE I MALFORMATION PRESENTING AS A HEADACHE IN AN ADULT PATIENT

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Chiari Malformation type I (CM-I) is a congenital anomaly characterized by the protrusion of cerebellar tonsils through the foramen magnum. Often associated with spinal cord cavitations like syringomyelia, the posterior fossa is usually small, leading to crowding and impaction at the foramen magnum. Typically asymptomatic, this malformation is often incidentally discovered. Recognition of Arnold-Chiari Type I malformation is crucial in differentiating adult headache cases. The average age of onset for Chiari malformations is approximately 24.9 ± 15.8 years, emphasizing the condition's potential to remain asymptomatic until adulthood.

We present a 55-year-old patient initially diagnosed with migraines; our case reveals an unexpected finding of an incidental Arnold-Chiari type I malformation identified late in adulthood—a remarkably uncommon age for such a diagnosis. Patient reported the sudden onset of forehead pain, radiated to occipital area, described as a pressure sensation, worsened with exposure to light and sounds and associated with nausea and vomiting. No prodromal symptoms. Patient disclosed recurrent history of similar headaches over the past five years, noting a progressive worsening symptoms recently.

CT brain showed inferior displacement of the cerebellar tonsils at the foramen magnum. Further workup with brain MRI confirmed incidental 10 mm cerebellar tonsillar ectopia consistent with Chiari I malformation. The significance of the presented case lies in the fact that even though the working diagnosis was headaches, which is an unspecific chief complaint, this prompted consideration of less common aetiologies, leading to a comprehensive review of images that ultimately revealed a subtle abnormality, culminating in the final diagnosis.

Keywords: Arnold Chiari malformation, headaches, cerebellar tonsils

[Abstract:2905]

MULTIPLE SCLEROSIS: TRANSITIONING FROM A SUSPECTED TO A CONFIRMED DIAGNOSIS

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Multiple sclerosis (MS) stands as the predominant immune-mediated inflammatory demyelinating ailment affecting the central nervous system. Pathologically, MS manifests through multiple

demyelinated regions, leading to the loss of oligodendrocytes and the formation of astroglial scarring. Notably, axonal injury becomes increasingly prominent in the later stages of the disease. While specific clinical characteristics are indicative of MS, the condition exhibits a widely variable progression and presents in numerous atypical forms. Genetic factors appear to contribute to the risk of MS, especially variation involving the HLA-DRB1 locus. The core (MS) phenotypes are those of relapsing and progressive disease.

Here we exhibit the case of a 38-year-old female who presented with dysphagia and dysarthria that started suddenly after she woke up. No prior episodes. CT brain demonstrated mild asymmetric patchy cerebral oedema in the right frontal and posterior frontal/parietal lobes. Further MRI brain confirmed a 2.1 cm T2/flair hyperintense lesion in the right corona radiata/centrum semiovale, along with a 0.6cm focus of enhancement along the medial aspect of this lesion. Findings favoured to represent a focus of active demyelination, especially given that there are additional lesions within the supratentorial white matter that likely represent chronic demyelinating plaques. Patient underwent a LP, CSF studies pertinent for protein elevation, mononuclear pleocytosis and positive oligoclonal bands. Started on methylprednisolone 1g q24 hrs 3 days, with symptoms improvement. Numerous potential prognostic indicators exist. Despite their presence, none have been firmly established as reliable, and our capacity to predict outcomes accurately for individual MS patients remains quite restricted.

Keywords: multiple sclerosis, demyelinating, dysarthria, dysphagia, oligoclonal

[Abstract:2999]

CORTICO-RESPONSIVE CORTICAL SPACE-OCCUPYING BRAIN LESIONS WITH PERSISTENT UNDETERMINED DIAGNOSIS

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Introduction: The aetiology of space-occupying lesions in the central nervous system (CNS) is highly varied, necessitating exhaustive investigation.

Case: 64-year-old female patient with history of hypertension, latent pulmonary tuberculosis, and immunosuppression due to seropositive rheumatoid arthritis. Previously treated with methotrexate and leflunomide, self-discontinued 2 months before admission due to gastrointestinal side effects. Admitted to Internal Medicine for an inaugural seizure. Contrast-enhanced MRI revealed multiple larger space-occupying lesions in the right medial temporal gyrus, suggestive of secondary infiltrative lesions. Treated with levetiracetam 500mg twice daily and systemic corticosteroids for cerebral oedema. No recurrence of seizures. Exhaustive studies showed no evidence of primary neoplasm: unremarkable blood count; thoraco-abdomino-pelvic CT, breast ultrasound and mammography, upper and lower gastrointestinal endoscopy, evaluations by Gynaecology, Ophthalmology, and Dermatology without suspicious lesions. Positron emission tomography showed heterogeneous cerebral uptake, with low avidity. Three adenopathies were noted (<25 mm): supraclavicular (inaccessible due to proximity to vascular structures); axillary (aspiration biopsy inconclusive), and paratracheal (small, non-capturing on PET - not biopsied). Cerebral CT was repeated about a month later to consider biopsy of the brain lesions. The lesions had almost disappeared and were not suitable for biopsy.

Conclusions: Between the initial image and first reassessment, dexamethasone was introduced for cerebral oedema and leflunomide was reintroduced. Upon follow-up consultation, 11 months after admission and without corticosteroid therapy, the patient remains without significant clinical events, and imaging reassessment shows complete remission of contrast-enhancing lesions compared to the initial study. The diagnosis continues to be undetermined.

Keywords: cortical space-occupying brain lesions, diagnostic challenge, corticosteroid therapy