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DISSEMINATED VISCERAL VARICELLA PRESENTING AS GASTRIC ULCERS

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ABSTRACT

Introduction: Herpes zoster results from reactivation of varicella-zoster virus (VZV) and classically presents with a dermatomal vesicular rash. Disseminated or visceral involvement is uncommon and predominantly occurs in immunosuppressed individuals. Gastrointestinal manifestations, such as oesophageal or gastric ulceration, are rare and can lead to diagnostic delay due to their non-specific presentation.

Case description: We report the case of 70-year-old man presenting with progressive dysphagia and weight loss over four weeks. The symptoms were preceded by dermatomal facial vesicular lesions consistent with herpes zoster, followed by the appearance of non-dermatomal cutaneous lesions. The patient had underlying type 2 diabetes mellitus and was geriatric, constituting functional immunosuppression. Upper gastrointestinal endoscopy revealed multiple ulcerations in the oesophagus and gastric antrum. Histopathology examination demonstrated viral cytopathic changes compatible with VZV infection, supporting a diagnosis of disseminated visceral varicella. Due to limited antiviral availability, the patient was managed with supportive therapy, systemic corticosteroids and symptomatic treatment, resulting in significant clinical improvement and resolution of dysphagia.

Conclusion: This case highlights disseminated visceral varicella as an important but under-recognised cause of gastrointestinal ulcerations in immunosuppressed elderly patients. Establishing standardised reporting criteria would allow for more persistent comparison and analysis of this rare manifestation of varicella infection.

KEYWORDS

Varicella-zoster virus, gastric ulcer, disseminated varicella, herpes zoster, immunocompromised host

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LEARNING POINTS

- Disseminated varicella-zoster virus infection may involve the gastrointestinal tract and present as dysphagia or gastric ulcers in immunosuppressed patients.
- Elderly patients with diabetes mellitus are at increased risk for atypical and visceral manifestations of varicella-zoster virus, and increased risk of atypical or visceral varicella-zoster manifestations when gastrointestinal symptoms accompany recent or evolving skin lesions.
- While antiviral therapy is generally recommended for disseminated or visceral varicella-zoster infection, clinical improvement may occur with supportive care in selected cases. Nonetheless, close monitoring is mandatory and antivirals should be promptly initiated if clinical worsening is observed.

INTRODUCTION

Herpes zoster is a viral syndrome caused by reactivation of the varicella-zoster virus (VZV), which occurs most frequently in older adults and immunocompromised populations. Reactivation of VZV presents as a unilateral maculopapular rash, which may evolve to become vesicular-pustular. In older adults, the rash may appear atypical, such as a small patch within the dermatome. It may also present as cranial neuritis if the cranial nerves are involved; such is the case for ophthalmic herpes zoster that affects the ophthalmic branch of the trigeminal nerve. Another atypical presentation would be the involvement of viscera, such as the gastrointestinal system[1]. In this case report, we present a geriatric patient presenting with dysphagia, which upon further investigation suffered from gastric ulcers due to disseminated VZV further complicated by diabetes mellitus.

CASE DESCRIPTION

A 70-year-old man was brought to the emergency room (ER) with dysphagia that started four weeks prior to admission and worsened in the previous four days. This was preceded by a sensation of burning in his chest and vomiting two days before that. The patient experienced weight loss due to these complaints in the previous four weeks. Three weeks before admission, the patient complained of headache, accompanied with lesions which appeared from the left cheek, spreading to the chin, neck, left earlobe and occipital area. The lesion was hyperalgesia, allodynic and pruritic. Due to these complaints, the patient had been treated with topical ointments and anti-emetics, with subsequent symptomatic relief. The patient did not experience tinnitus or unilateral facial weakness.

Upon arrival at the ER, the patient's Glasgow Coma Score was E4V5M6 and the vital signs were as follows: blood pressure 179/117 mmHg, pulse 81 bpm, respiration rate twenty times per minute, SpO_2 96%, with a visual analogue scale of six. A general examination revealed multiple hyperpigmented lesions on the cheeks, chin, neck, retro auricular and occipital region *(Fig. 1)*. Physical examination revealed tenderness in the left collar region and both sides of the supraclavicular region. There was no lymphadenopathy. The laboratory investigations revealed leucocytosis (11,560 µl), monocytosis (8%), elevated HbA1c (7.01%), elevated random blood sugar (174 mg/dl) and mild hyponatraemia (134.95 mEq/l).

On the second hospital day, new lesions appeared in the right lumbar region with blisters and multiple erythematous vesicles *(Fig. 2)*. The lesions were inflamed and pruritic. An oesophagogastroscopy was performed and showed white plaques along with multiple ulcerations in the middle and distal oesophagus *(Fig. 3)*. In the stomach, there were multiple ulcers in the antrum, along with eroded and gapping pylorus. Histopathology was also performed and revealed nuclei with margination of chromatin, with some cells demonstrating multinucleation and nuclear moulding.

The patient was treated with an IV drip of ondansetron 8 mg TID, ceftriaxone 2 g OD, and omeprazole 8 mg/hour, which was increased to 40 mg BID. Amlodipine 10 mg OD, ramipril 5 mg OD and metformin 500 mg OD was administered due to a history of uncontrolled hypertension and type-2 diabetes mellitus. A combination



Figure 1. *Multiple hyperpigmented lesion on the cheeks, chin, retroauricular and occipital region.*



Figure 2. Blisters and multiple erythematous lesions on the right side of lumbar region.

of 5% topical gentamicin and betamethasone, and oral methylprednisolone 62.5 mg OD was administered. Drainage of bullae was also performed by the dermatology department. The patient improved significantly, with cessation of dysphagia and resolution of skin lesions. The patient was discharged on the seventh hospital day.

DISCUSSION

Reactivation and dissemination of varicella in at-risk populations

Several comorbidities act as risk factors for VZV reactivation, of which several relevant ones will be described. Immunocompromised patients typically present with a more severe clinical progression of VZV infections, particularly visceral disseminated VZV. This has been shown in guinea pigs, where stress and immunodeficiency reactivate VZV in pigs bearing latent infection; several human cases have also reported this phenomenon[2].

Therefore, disseminated VZV should be considered as a significant differential diagnosis in immunocompromised patients presenting with symptoms suggestive of acute abdomen or other acute gastrointestinal events. Our geriatric patient, while otherwise immunocompetent, is assumed to have some extent of immunosuppression due to his age and clinical background. His chief complaints included epigastric pain, dysphagia and vomiting. This is consistent with what was explained before on the correlation between an immunocompromised state and varicella reactivation and dissemination. It is also consistent with findings from previously published cases, where the majority of the recorded patients are over 60 years old, suggesting some degree of related immunocompromise[3,4].

Diabetes is also a prominent risk factor for VZV infection and reinfection. Risk of infection was higher in diabetic patients, and this risk further increases with age, suggesting a grim agonism with age-related immunocompromise[5]. A review has highlighted that hyperglycaemia present in diabetic patients induces immune dysfunction, hence the risk for infections, or in this case reactivation of prior infections[6]. It can be deduced that diabetes promotes VZV reactivation and dissemination in much the same way that immunodeficiency does. Our patient had untreated type-2 diabetes mellitus, also noted in several previously reported cases[3,7]. This is in line with what has been explained regarding the correlation between diabetes and VZV infection and/or reinfection.

Several other comorbidities that may present as risk factors, although not present in our patients, include the use of chemotherapy and the presence of malignancies. Both of these factors seem to also induce immunodeficiency, hence enabling reinfection and dissemination of VZV, as reported by several studies and guidelines examining the same virus[2].

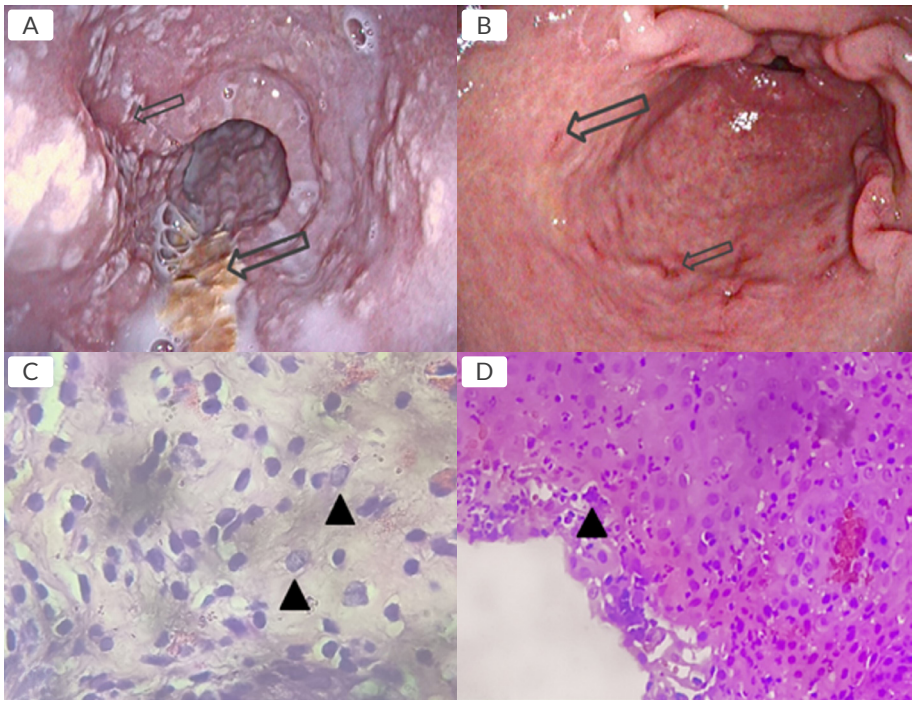


Figure 3. A) Endoscopic image demonstrating multiple ulceration of mid oesophagus; B) Multiple ulceration of antral gastric Forrest III classification; C) High-power histology view of antral gastric demonstrating viral Cowdry type B inclusions (H&E, 1000×); D) Demonstration of multinucleation of oesophageal view (H&E, 400×).

Dermatological features of disseminated varicella

Dermatological features may present as a vital clue to differentiate between disseminated varicella and primary cutaneous varicella. A case report with a review of multiple other cases found that a majority (89%) of cases initially presented with a localised dermatomal lesion, followed by a secondary diffuse papulovesicular rash. The latter would be the distinguishing feature between disseminated varicella and primary cutaneous varicella. Many of the patients (42%) also suffered from cranial nerve involvement, which may suggest their susceptibility to cutaneous dissemination[8]. Our patient presented with initial dermatomal lesions, followed by a non-dermatomal diffuse lesion on the right lumbar region. This can be considered typical of disseminated varicella with the current evidence available.

The mechanism of VZV dissemination has yet to be fully understood. However, a study showed that the haematogenous spread of VZV-induced latency in the enteric neurons and dorsal root ganglia, waiting to be reactivated by an immunodeficient episode. The haematogenous spread of the virus explains the non-dermatomal lesions, and why it primarily forms visceral lesions – especially in the gastric region – coupled with a diffuse dermatomal lesion. Gastric manifestations include herpetic lesions in the gastric antrum, multiple shallow ulcers in the gastric body and mucosal inflammation without ulcerations[2]. In immunocompromised patients, the manifestation of reoccurrence is not limited to being shown as cutaneous only. However, the visceral manifestation also can occur in immunocompromised patients. Upon endoscopic evaluation, this patient was found to have multiple ulcerations in the mid and distal oesophagus, accompanied by gastritis. This is in line with the current available evidence on gastric manifestations of visceral varicella. However, this current evidence describes gastrointestinal lesions in a highly heterogeneous way, making comparison difficult. The study by Gershon and Gershon remains the only one that was described in a consistent manner sufficient for comparison. In this case, the history of a varicella infection before is unclear[2]. However, several immunocompromising risk factors were present such as diabetes mellitus, high dose steroid use and advanced age.

Possible adverse effects of steroid and antiviral therapy

A previous case report described patients who received acyclovir, at doses ranging from 5-10 mg per kilogram bodyweight with consistent and significant clinical improvement following antiviral therapy[9]. In contrast, our patient did not receive acyclovir due to drug unavailability, yet still demonstrated progressive clinical improvement with supportive and symptomatic treatment, including systemic corticosteroids.

Steroid administration has been reported in several cases. In a previous review, patients reviewed corticosteroids such as methylprednisolone or prednisone[8,10]. Our patients received 62.5 mg of methylprednisolone daily with symptomatic improvement afterwards.

Nevertheless, the role of corticosteroids in visceral varicella remains controversial. Earlier reports suggested symptomatic rebound following steroid withdrawal. However, more recent evidence supports the cautious use of corticosteroids for symptomatic relief in immunocompetent adults, especially in the absence of antiviral therapy[9,10]. The favourable outcome in our case suggests that, in selected patients, supportive therapy including corticosteroids may contribute to clinical improvement, although antiviral therapy remains the standard of care when available.

REFERENCES

1. John AR, Canaday DH. Herpes zoster in the older adult. *Infect Dis Clin North Am* 2017;**31**:811–826. doi:10.1016/j.idc.2017.07.016
2. Gershon AA, Gershon MD. Pathogenesis and current approaches to control of varicella-zoster virus infections. *Clin Microbiol Rev* 2013;**26**:728–743. doi:10.1128/CMR.00052-13
3. Matsuo Y, Igarashi Y, Aoyama N, Satta Y, Oikawa R, Noguchi Y, et al. Visceral disseminated varicella-zoster virus infection in an immunocompetent host. *Clin J Gastroenterol* 2022;**15**:568–574. doi:10.1007/s12328-022-01607-7
4. Krones E, Petritsch W, Valentin T, Durchschein F, Gorkiewicz G. Visceral dissemination of herpes zoster with multiple ulcers in the upper gastrointestinal tract of an apparently immunocompetent patient. *Endoscopy* 2012;**44**:E302–E303. doi:10.1055/s-0032-1309926
5. Saadatian-Elahi M, Bauduceau B, Del-Signore C, Vanhems P. Diabetes as a risk factor for herpes zoster in adults: a synthetic literature review. *Diabetes Res Clin Pract* 2020;**159**:107983. doi:10.1016/j.diabres.2019.107983
6. Berbudi A, Rahmadika N, Tjahjadi AI, Ruslami R. Type 2 diabetes and its impact on the immune system. *Curr Diabetes Rev* 2020;**16**:442–449. doi:10.2174/1573399815666191024085838
7. Mizu D, Nishida H, Matsuoka Y, Ariyoshi K. Visceral disseminated varicella zoster infection: a rare cause of acute abdomen in a patient with well-controlled diabetes mellitus – a case report. *BMC Infect Dis* 2022;**22**:215. doi:10.1186/s12879-022-07183-y
8. Gomez E, Chernev I. Disseminated cutaneous herpes zoster in an immunocompetent elderly patient. *Infect Dis Rep* 2014;**6**:5513. doi:10.4081/idr.2014.5513
9. Sprung B, Werth T, Huber AR. An unusual cause of gastritis. *Gastroenterology* 2016;**151**:e11–e12. doi:10.1053/j.gastro.2016.07.002
10. Nohr EW, Itani DM, Andrews CN, Kelly MM. Varicella-zoster virus gastritis: case report and review of the literature. *Int J Surg Pathol* 2017;**25**:449–452. doi:10.1177/1066896917696751