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ACQUIRED FANCONI-LIKE PROXIMAL RENAL TUBULOPATHY ASSOCIATED WITH KLEBSIELLA PNEUMONIAE INFECTION

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ABSTRACT

Acquired Fanconi syndrome is a rare proximal renal tubular disorder associated with glucosuria, aminoaciduria, phosphaturia, electrolyte disturbance and metabolic acidosis, usually caused by drugs, toxins, or plasma cell disorders. Although infectious causes are rare, this is a case report of a 68-year-old man with altered sensorium due to metabolic encephalopathy secondary to dyselectrolytemia. Evaluation revealed severe hyponatremia, hypokalemia, hypocalcaemia, hypophosphatemia and hypomagnesemia. Urine examination showed proteinuria, normoglycemic glycosuria, inappropriate urinary electrolyte losses and impaired urine concentrating ability. Arterial blood gas analysis showed a normal anion gap metabolic acidosis with a positive urine anion gap suggesting a renal cause for the acidosis. This clinical and laboratory picture is consistent with Fanconi-like proximal tubulopathy. Radiological imaging showed cavitary lung consolidation and bilateral pyelonephritis. Sputum cultures grew *Klebsiella pneumoniae*. After excluding the usual causes of acquired Fanconi syndrome, the proximal tubular dysfunction was attributed to systemic infection. Following treatment with appropriate antibiotics for the infection and electrolyte replacement, the patient completely recovered clinically and biochemically. This case documents a systemic *K. pneumoniae* infection presenting with reversible Fanconi-like proximal tubulopathy highlighting the importance of exploring infectious causes in patients with unexplained proximal renal tubular dysfunction.

KEYWORDS

Fanconi syndrome, proximal renal tubulopathy, *Klebsiella pneumoniae*, dyselectrolytemia, renal glycosuria

LEARNING POINTS

- This case describes the first documented case of acquired Fanconi-like proximal tubulopathy secondary to a systemic *Klebsiella pneumoniae* infection and demonstrates complete reversibility of proximal tubular dysfunction, as evidenced by resolution of dyselectrolytemia after appropriate antibiotic treatment.
- It extends the spectrum of infectious causes of Fanconi syndrome to include Klebsiella, in addition to previously known causes such as *Legionella*, *Streptococcus pneumoniae*, and *Leptospira*.
- The electrolyte abnormalities were initially attributed to syndrome of inappropriate antidiuretic hormone secretion secondary to pneumonia before the proximal tubular dysfunction was identified leading to a diagnosis of acquired Fanconi like syndrome.

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INTRODUCTION

Fanconi syndrome is a disorder of proximal tubular reabsorption, characterized by excessive urinary losses of glucose, phosphate, amino acids, uric acid, bicarbonate, sodium, potassium, and low-molecular-weight proteins despite normal glomerular filtration rate. Acquired Fanconi syndrome usually occurs due to medications, heavy metal toxicity and plasma cell dyscrasias. Infectious causes are rare, with only isolated case reports describing proximal tubulopathy induced by bacterial infection like Legionella pneumonia, invasive *Streptococcus pneumoniae* and *Listeria*. We report a unique case of acquired Fanconi- like proximal tubulopathy associated with a systemic *Klebsiella pneumoniae* infection in an elderly male.

CASE DESCRIPTION

A 68-year-old male presented with history of cough with expectoration for 1 week followed by giddiness, vomiting and altered sensorium for 1 day. His medical history included hypertension, hypertrophic cardiomyopathy, and atrial fibrillation on regular treatment with amlodipine, metoprolol and acenocoumarol. There was no history of diabetes mellitus, nephrotoxic drug exposure, or recent hospitalization.

On physical examination, the patient was drowsy and dehydrated. His vital signs included a blood pressure of 120/70 mmHg measured in the right upper arm in a supine position, a pulse rate of 67 beats/min irregularly irregular; a respiratory rate of 20/min, and oxygen saturation of 99% in room air. Cardiovascular examination confirmed atrial fibrillation with controlled ventricular rate. Respiratory system examination revealed fine inspiratory crepitations in the left infrascapular region. On neurological examination the patient was drowsy but arousable and he was disoriented to time and place. The patient's Glasgow Coma Scale (GCS) score was 12/15. Kernig's sign and Brudzinski's sign were negative.

Initial investigations showed a normal white blood cell count with raised inflammatory markers. Serum electrolytes were deranged, with hyponatremia (serum sodium 101 mEq/l), hypokalemia (serum potassium 2.4 mEq/l), hypocalcaemia (8mg/dl), hypomagnesemia (1.5 mg/dl), and hypophosphatemia (2.3 mg/dl), hypouricemia (2 mg/dl) and chloride 67 mEq/l. Renal function tests were within normal limits. Arterial blood gas analysis revealed normal anion gap metabolic acidosis.

Urine analysis revealed dilute urine with low specific gravity and urine glucose of 2+ despite a normal serum glucose level (120 mg/dl), suggestive of renal glycosuria. Urine electrolytes revealed excessive excretion of sodium and potassium. Urine osmolality was 231.1 mOsm/kg against serum osmolality of 258.8 mOsm/kg, indicating impaired renal concentrating ability. Urine anion gap was 4.2 mEq/l. Serial laboratory values are listed in *Table 1*.

A high-resolution computed tomography (CT) scan of chest showed left lower lobe cavitary consolidation with mild surrounding ground glass opacities *(Fig. 1)*. Sputum culture grew *K. pneumoniae*. Brain imaging was normal. A CT scan of abdomen revealed an enlarged left kidney with bilateral perinephric fat stranding *(Fig. 2)* suggestive of pyelonephritis although urine cultures were sterile. Blood cultures also remained sterile.

Additional investigations, including serum and urine electrophoresis and heavy metal screening, were negative, ruling out common acquired causes of Fanconi syndrome. Conditions linking cardiomyopathy and Fanconi syndrome were considered including mitochondrial cytopathies, but they typically present earlier in life, demonstrate multisystem involvement, and follow a chronic or progressive course, which were absent in this patient.

The patient was diagnosed with acquired Fanconi- like proximal tubulopathy associated with *K. pneumoniae* infection. He was treated with intravenous ceftriaxone for 7 days. Additional supportive treatment included gradual sodium correction with 3% normal saline infusion, potassium chloride infusion, magnesium sulphate and calcium gluconate supplementation. Clinical improvement was noted within 48 hours, with gradual correction of electrolyte abnormalities. Sodium levels improved to 124 mEq/l over the next 72 hours. The patient's neurological status improved significantly, and he had a GCS score of 15/15 at discharge. He was discharged after 9 days with stable vital signs and near normal electrolyte levels. On review after 1 month his electrolytes had normalised and repeat chest radiography showed resolution of pneumonia.

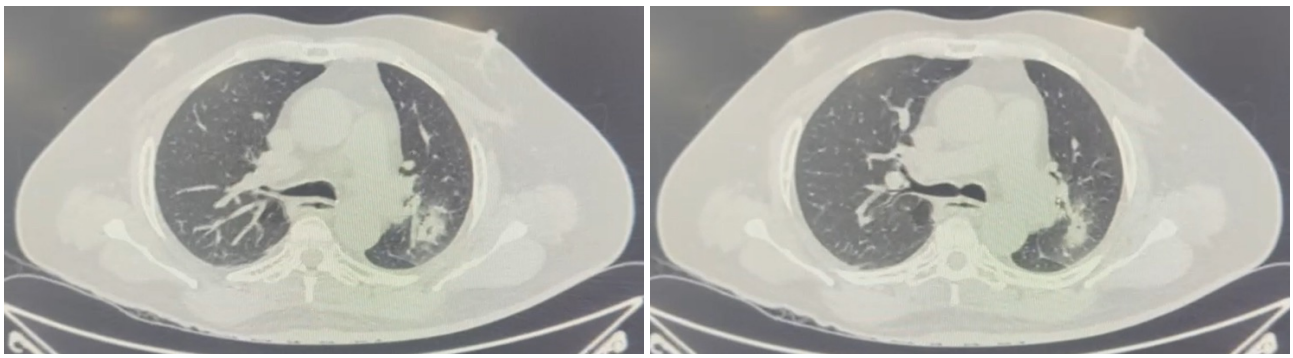


Figure 1. Axial computed tomography scan of the chest showing left lower lobe patchy cavitary consolidation with surrounding ground-glass opacities.

Parameters	Day 1	Day 2	Day 3	Day 4	Day 9	Day 40	Reference range
Serum creatinine (mg/dl)	0.90	0.81	0.79	-	1.16	1.01	0.7-1.3
Blood urea nitrogen (mg/dl)	10	9	6	-	11	12	7-20
Serum sodium (mEq/l)	101	108	117	124	134	139	135-145
Serum potassium (mEq/l)	2.4	2.7	3.1	3.3	4.5	4.2	3.5-5
Serum chloride (mEq/l)	67	74	85	-	97	99	96-106
Serum calcium (mg/dl)	-	8.0	-	8.7	8.9	-	8.5-10.5
Serum albumin (g/dl)	-	4.7	-	-	4.3	-	3.5-5.0
Serum phosphorus (mg/dl)	-	2.3	-	-		3.8	2.5-4.5
Serum magnesium (mg/dl)	-	1.5	-	-	1.6	1.9	1.7-2.2
Serum bicarbonate (mEq/l)	19	-	-	-	22	-	22-29
Serum uric acid (mg/dl)	-	2.0	-	-	-	5.0	3.5-7.2
Serum PTH (pg/ml)	-	34.1	-	-	-	-	15-65
Serum vitamin D3 (ng/ml)	-	21.9	-	-	-	-	-
Serum osmolality (mOsm/)	-	258.8	-	-	278	-	30-50
C- reactive protein	Reactive	-	-	-	-	-	-
Urine (pH)	6	-	-	-	-	6	5-8
Urine glucose (dipstick)	++	-	-	-	-	Negative	-
Urine spot protein creatinine ratio	0.6:1	-	-	-	-	-	-
Urine spot sodium (mEq/l)	83	-	-	-	-	-	-
Urine spot potassium (mEq/l)	22.2	-	-	-	-	-	-
Urine chloride (mEq/l)	101	-	-	-	-	-	-
Urine spot creatinine (mg/dl)	23.5	-	-		-	-	-
Urine anion gap	Positive (4.2)	-	-	-	-	-	-
24hr urine sodium (mmol/day)	-	420	-	-	-	-	-
24hr urine potassium (mmol/day)	-	90	-	-	-	-	-
24hr urine protein (mg/day)	-	620	-	-	-	-	-
Urine osmolality (mOsm/kg)	231	-	-	-	-	-	-
Random blood glucose (mg/dl)	122	-	-	-	-	106	-

Table 1. Key laboratory findings.
Abbreviation: PTH, parathyroid hormone.

DISCUSSION

Fanconi syndrome results from impaired solute transport in the proximal renal tubule. The classical features of electrolyte derangement, proteinuria, glucosuria in the absence of hyperglycaemia, and metabolic acidosis were all present in this case[1]. Preexisting idiopathic proximal tubulopathy is unlikely in this case, given the previously normal serum electrolytes and evidence of complete resolution of tubulopathy after the acute infectious episode. Infections such as *Legionella pneumophila*, invasive *S. pneumoniae* and *Leptospirosis* have been documented in rare, isolated instances leading to Fanconi-like tubulopathy[2-6]. In

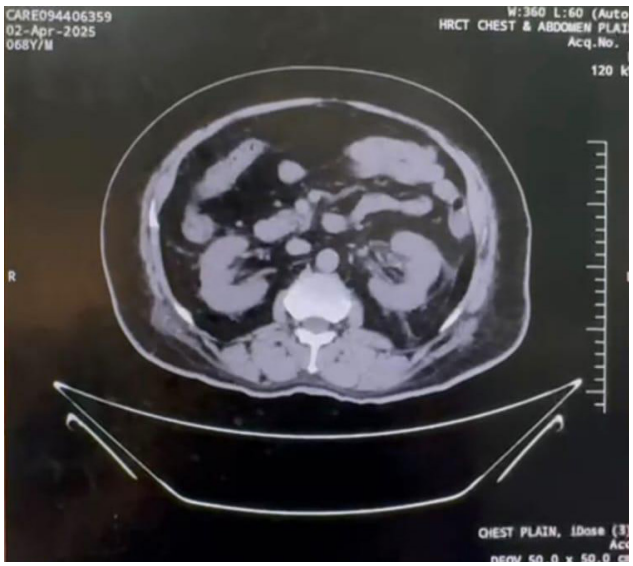


Figure 2. Axial computed tomography image of the abdomen demonstrating an enlarged left kidney associated with bilateral perinephric fat stranding.

our patient there was an association between *K. pneumoniae* infection and acquired Fanconi-like syndrome, adding to emerging evidence that gram-negative organisms like *Klebsiella* may cause transient proximal tubulopathy, particularly during systemic infections. Though acute pyelonephritis can cause tubular injury, it predominantly affects the distal tubules. Pyelonephritis causing proximal tubulopathy would be associated with severe renal parenchymal destruction or obstruction causing acute kidney injury, which was absent in our patient. Also, all abnormalities resolved with appropriate antibiotic therapy, indicating a direct toxin effect or immune-mediated mechanism rather than permanent structural damage. Taken together, the clinical picture, laboratory findings, multisystem involvement and reversibility of the tubular dysfunction favour a diagnosis of Fanconi-like proximal tubulopathy secondary to systemic *K. pneumoniae* infection rather than isolated pyelonephritis.

The pathophysiological mechanism linking *K. pneumoniae* to proximal tubulopathy is unclear. Potential mechanisms include direct bacterial toxin effects on tubular transport proteins, immune-mediated tubular injury or systemic inflammatory response causing mitochondrial dysfunction in renal tubules[2-6]. *Klebsiella* species produce various virulence factors including lipopolysaccharides and capsular polysaccharides that could theoretically impact renal tubular function[2].

This case highlights the importance of considering infectious aetiologies in unexplained proximal tubular dysfunction, when common causes have been excluded. The favourable outcome following antimicrobial therapy supports the causal relationship between *K. pneumoniae* infection and the development of Fanconi-like proximal tubulopathy. Early recognition and treatment of the underlying infection, combined with appropriate electrolyte management, led to complete clinical recovery.

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