Case Report

Haemolytic uraemic syndrome associated with Klebsiella empyema-case report and literature review

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We report a 13 month old male with haemolytic uraemic syndrome (HUS) associated with Klebsiella empyema. He presented with fever, gastroenteritis and respiratory distress. Investigations showed left pleural effusion, and HUS. Blood and pleural exudate cultures grew Klebsiella pneumoniae. He died during chest tube insertion. Post mortem showed fibrinous empyema. Renal histology confirmed HUS. Klebsiella has not been previously described to cause HUS. This is the first case of HUS and empyema associated with invasive K. pneumoniae in the Niger Delta Region.

Key words: Acute renal failure, empyema, Haemolytic uraemic syndrome and Klebsiella.

INTRODUCTION

Haemolytic uraemic syndrome, (HUS) is characterized by acute renal failure, (ARF), haemolytic anaemia, and thrombocytopenia, Loirat et al (1984). It is the commonest cause of ARF in children aged from 1 to 2 years. HUS has been classified into (a) typical or diarrhoea associated (D + HUS) and (b) atypical or non-diarrhoeal (D-HUS). The commonest form, D + HUS is caused by infection with Enterotoxigenic Escherichia coli, Shigella, Salmonella or Campylobacter. D-HUS is caused by Streptococcus pneumoniae, (Cabrera et al., 1998; Waters et al., 2007; Brandt et al., 2002; Lee et al., 2006). Some cases of atypical HUS have been reported in Africa, based on genetic mutations Habib et al, (2010). Klebsiella species and Klebsiella empyema have not been reported as causes of HUS. Data associating empyema and HUS are rare but a recent report suggests that one third of under-2year olds with empyema are at risk of developing HUS, Nyman et al. (2009). We report this case of a 13 month old male with HUS and empyema following infection in which only Klebsiella pneumoniae was isolated.

CASE REPORT

C.J. was a 13 month old male who was referred from a private clinic with a 2 week history of fever, cough and fast breathing. He had a five day history of vomiting and abdominal distension and a two day history of non-bloody diarrhoea, scanty urine and leg oedema. He had normal neonatal and developmental history. He was the first child of non-consanguineous parents.

At the referring hospital, his PCV was 15%. He had leukocytosis and neutrophilia. He was transfused with packed red blood cells and given intramuscular artemisinin and intravenous ceftazidime for a clinical diagnosis of severe malaria and pneumonia.

On arrival at our centre, he was afebrile (T-37.1°C). He was tachypnoeic with a respiratory rate of 68 cycles / min. Heart rate was normal 132 beats/min; blood pressure low 60/30 mmHg. He was grossly oedematous, severely pale and jaundiced. He had signs of left pleural effusion with dull percussion notes and crepitations over the hemithorax. He had marked ascites and soft non tender hepatomegaly of 10 cm. An initial diagnosis of streptococcal HUS was made.

Dipstick urinalysis showed massive proteinuria and
haematuria. Urine microscopy showed no casts. Full blood count showed anaemia (PCV 22%), leukocytosis and thrombocytopenia. Fragmented cells and Burr cells were seen on the peripheral blood film. Biochemistry showed metabolic acidosis with a very low bicarbonate of 10 mmol/L. Unfortunately anion gap was not calculated as chloride was not measured. Urea and serum creatinine were both elevated at 47.8 mmol/L and 465 µmol/L respectively. Chest X-Ray showed left lobar consolidation with pleural effusion. Blood and pleural exudate inoculated into a Columbia agar diphasic medium and thioglycollate broth grew *K. pneumoniae* sensitive only to ciprofloxacin but resistant to most drugs including cefuroxime, cefazidime and ceftriaxone. Blood was cultured only once as the patient died within 24 h of admission. He was transfused with sedimented blood cells. He was billed for peritoneal dialysis before he stopped breathing a few minutes after a chest tube was inserted. At autopsy cortical and medullary hemorrhages were seen in his kidneys, Figure 1 and fibrinous exudates in the lungs, Figure 2. He had a fatty liver. Renal histology showed arteriolar thrombi, Figure 3, schistocytes and mesangiolysis, Figure 4. There was also acute tubular necrosis with tubular casts and extramedullary haemopoiesis. The cause of death was pneumonia and ARF.

**DISCUSSION**

HUS is characterized by ARF, haemolytic anaemia, and thrombocytopenia. It occurs mainly in children < 3 years old, an age similar to that of our patient who was 13 months old.

HUS usually follows an episode of diarrhoea with Enterotoxigenic *E. coli* 0157:H7. *Shigella dysenteriae* type1 and *citrobacter freundii* also cause D + HUS, (Johnson and Taylor, 2009). Our patient had D + HUS as he had diarrhoea, ARF and the haematological features of HUS with anaemia, thrombocytopenia, schistocytosis and fragmented red blood cells.

*Klebsiella* spp has not been previously reported as a cause of HUS following episodes of diarrhoea but *Klebsiella* urinary tract infection has preceded HUS, (Loirat et al., 1984). *K. pneumoniae* was the only isolate in both the blood and pleural exudate in our patient, unlike previous reports where *Klebsiella* peritonitis was a secondary infection in HUS caused by *Streptococcal Pneumoniae* in a 2 year old, (Obata, 1998).

A study, found that irradiation, chemotherapy, viral infections such as mumps, chicken pox and arbovirus may all be precipitants of an episode of HUS. Our patient had no clinical evidence of these other precipitants of HUS. D-HUS is associated with invasive *Streptococcal* disease, and disorders of complement regulation, (Caprioli et al., 2006) while similar histology is seen in adults with thrombotic thrombocytopenic purpura related to ADAMTS13 (a disintegrin and metalloprotease with thrombospondin type 1 repeats) abnormalities and defective cobalamin metabolism, (Valavi and Alemzadeh, 2008). In India, a scorpion sting has caused HUS, Valavi and Alemzadeh (2008).
Klebsiella spp are a group of Gram negative bacteria that belong to the Enterobacteriaceae family. They are found in the human gastrointestinal tract, eyes, respiratory tract and genitourinary tract, (Gupta et al., 2003). A study done in the United States on children who had Klebsiella bacteremia found no case that led to HUS (Bonadio, 1989). However, K. pneumoniae isolated in both blood and hamburger was shown to be a cause of renal and multi-organ failure in an adult male following ingestion of a hamburger (Sabota et al., 1998). The
The patient had ARF requiring dialysis. The Klebsiella isolate in both the blood and hamburger was shown to be enteroinvasive as full biochemical profiles; antimicrobial sensitivity, plasmid profile, and toxin assay by DNA hybridization probe were completely concordant.

Reports on *K. pneumoniae* as a cause of ARF are rare but was shown in blacks and Indians in South Africa (Seedat and Nathoo, 1993). The clinical presentation of this 13 month old child with diarrhoea and vomiting, Klebsiella empyema and bacteremia, haemolytic anaemia and ARF together with autopsy and histological findings consistent with microangiopathy and HUS suggest an enteroinvasive organism.

Unfortunately, further investigations such as urine detection test for the causative organism could not be done in our laboratories. These would have been used to determine if this was a case of HUS from invasive streptococcal disease complicated by *K. pneumoniae* infection. However streptococcus was not isolated in our cultures and the presence of diarrhoea in our patient was not typical of streptococcal D-HUS (Nyman et al, 2009).

Data associating empyema and HUS are rare but a recent report suggests that one-third of under-2 year olds with empyema are at risk of developing HUS.

This patient is not likely to have had purpura fulminans, an emergency haematological disease associated with purpura, disseminated intravascular coagulation, skin necrosis and multi organ failure, (Chalmers et al., 2011) as there was no purpura and no skin lesions.

**CONCLUSION**

We present a rare case of HUS and empyema associated with invasive *K. pneumoniae*.

**REFERENCES**


