

## A case presentation of haemolytic uraemic syndrome presenting as gastroenteritis

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A 62-year-old man presented to our department with gastroenteritis. He was found to have decreased platelets and deranged renal function. A diagnosis of haemolytic uraemic syndrome was eventually made. The disease usually presents nonspecifically with gastroenteric symptoms and systemic upset at the initial stage. Without a high index of suspicion, the diagnosis can easily be missed. (*Hong Kong j.emerg.med.* 2003; 10:54-56)

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### Introduction

Haemolytic Uraemic Syndrome (HUS) is a life-threatening condition. It is characterized by haemolysis, thrombocytopenia, and acute renal failure.<sup>1,2</sup> Organs including the brain, intestines, pancreas, heart, and lungs may be affected. It is frequently associated with gastroenteritis caused by *E. Coli* O157:H7 infection.

### Case presentation

The patient was a 62-year-old man who enjoyed good past health. He was a non-drinker and non-smoker. In the past few months, he had made several trips to China. He developed subjective fever 3 days before attending our Accident and Emergency department (A&E). He also complained of headache, generalized

malaise, dizziness and muscle pain. He vomited undigested food on the day of admission. There was also diarrhoea but no history of bloody stool was present. Physical examination showed normal vital signs. Mild tenderness was present in the abdomen. The cardiovascular and respiratory examination was normal. He was given an intramuscular injection of Toradol and Stemetil. Blood was taken for a complete blood picture and showed markedly decreased platelet count of 53,000. He was admitted to our hospital.

His symptoms persisted after admission. CT brain was done in view of his persistent headache and vomiting. The result of the scan was normal. On day 4, his renal function was found to be deteriorating. ALT and LDH levels were also found to be raised. Atypical lymphocytes were present in peripheral blood smear. ANCA, C3, ASOT were normal. Renal team was consulted and he was suspected to be suffering from haemolytic uraemic syndrome. He was treated with several sessions of haemodialysis and **plama exchange**. His renal function and platelet counts improved steadily and he was discharged after 2 weeks of hospitalization. Although the patient had symptoms of gastroenteritis, the stool culture did not have any significant growth.

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## Discussion

HUS most commonly occur after colitis resulting from *E. Coli* O157:H7 infection, but it may also emerge after malignant hypertension, scleroderma, systemic lupus erythematosus, chemotherapy, radiation, HIV infection, and eclampsia.<sup>2,3</sup> *E. Coli* O157:H7 bacteria are usually ingested through contaminated food or water, but they can also be transmitted person to person through the faecal-oral route.

Most strains of *E. Coli* are harmless, but the O157:H7 strain produces a powerful toxin that can cause severe diarrhoea, abdominal cramping, nausea, and vomiting. Though illness usually resolves completely in 5 to 10 days, it may progress to haemorrhagic colitis and HUS, especially in young children and the elderly.

HUS is similar to thrombotic thrombocytopenic purpura (TTP), a disease that also causes microangiopathic haemolytic anaemia. But whereas HUS is most common in children, and is almost always associated with an *E. Coli* O157:H7 infection resulting in diarrhoea and renal failure, TTP is most common in adults and usually involves the neurologic system, although the kidneys are sometimes affected. Because HUS and TTP are so similar, some experts suggest that the two are actually the same syndrome with different manifestations.<sup>4,5</sup>

The patient in this case presented with gastroenteritis symptoms with severe systemic upset. So the aetiological agent causing HUS this time is probably due to enteric pathogen, and the most likely candidate is *E. Coli* O157:H7. It can produce a verocytotoxin that cause haemorrhagic colitis. The absence of bloody diarrhoea in the history made the picture less typical. When *E. Coli* O157:H7 is suspected, a specific enrichment culture should be ordered to isolate the bacteria. The sample should also be collected as early as possible because the culture would be negative 6 days after the onset of the diarrhoea. So the negative stool culture in this case is expected because the specimen was collected rather late in the course of the disease.

The verocytotoxin targets the endothelial cells in the kidney and other organs with glycoprotein genotype Gb3 receptor.<sup>6</sup> Children under the age 2 have more of this kind of receptor and therefore are more prone to renal failure caused by HUS.<sup>7</sup> When endothelial cells of the kidney are attacked by verocytotoxin, protein synthesis will be inhibited and the cells will die. An inflammatory process will be induced and the glomerular capillary lumens become narrowed. This will lead to destruction of platelets and RBCs as they squeeze through the lumen. There will also be platelet activation and clotting, causing further depletion of platelets.<sup>3</sup> The damaged platelets and RBCs would then be removed by the spleen resulting in decrease in platelet count, haemoglobin, haematocrit and RBC count. LDH, conjugated and unconjugated bilirubin levels will be raised as a result of haemolysis. The narrowing of the glomerular lumens cause the decrease in glomerular filtration rate. Urine output decreases while BUN and serum creatinine levels rise.<sup>7</sup>

Plamapheresis and corticosteroids have been proven to be effective in the treatment of haemolytic uraemic syndrome.<sup>7,8</sup> Plamapheresis may not be necessary in mild cases if water and electrolyte balance are well maintained. Fresh frozen plasma can be administered to the patient to replace the loss of plasma proteins and coagulation factors. When there is a suspicion of *E. Coli* O157:H7 infection, antimotility agents and antibiotics should be withheld. Antimotility agents can slow the transit time of the bacteria in the intestine and cause increased toxin absorption. Antibiotics may destroy the normal bowel flora, leading to overgrowth of *E. Coli*. They may also lyse the *E. Coli* organisms, causing additional toxins to be released.<sup>1,8</sup>

## References

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