

LETTERS TO THE EDITOR

HAEMATURIA AS THE FORM OF PRESENTATION OF THE HAEMOLYTIC URAEMIC SYNDROME**Mr. Director;**

Haematuria is an infrequent form of presentation of the haemolytic uraemic syndrome (HUS). The HUS most often appears in infancy with manifestations of diarrhoea and altered laboratory tests. The most common cause is a toxin produced by *Escherichia coli*. The mortality is high (5%-15%) with severe sequelae and an unfavourable prognosis in adults¹.

A 24-year-old asthmatic, alcohol and drug consuming male arrived at the emergency department because of haematuria during 48 hours. He had had a previous febrile amygdalar process with antibiotic treatment. Physical examination was anodyne with normal vital measures. Only conjunctival ictericia was of note. Blood analysis showed acute renal failure and a low platelet count (3,000 mm³). Given the severity of the picture a lumbar puncture with bone marrow aspiration was performed in the emergency department showing normal megakaryopoiesis compatible with peripheral thrombocytopenia. The patient was admitted and in the first 48 hours presented a clinical-analytical worsening with deterioration in renal insufficiency, elevated LDG and the progressive appearance of regenerative anaemia and petechias. Erythrocytary morphology detected schistocytes finally suggesting the diagnosis of HUS. The patient was transferred to an intensive care unit for plasmapheresis demonstrating a clear improvement within hours (analytical correction and disappearance of conjunctival ictericia and haematuria). The patient was transferred to the ward and discharged with no further events.

The presence of haematuria in a young patient with a recent history of upper respiratory infection, alcohol and drug consumption, intense peripheral thrombocytopenia with no other signs of bleeding and with acute renal failure led to the immediate suspicion of acute post-streptococci glomerulonephritis without ruling out HUS in the early stages. The age of the patient, the lack of initial anaemia and the petechias made the initial diagnosis difficult.

The most common cause of HUS is a toxin produced by *E. coli* serotype 0157:H7 transmitted by undercooked meat or non pasteurised lacteal products. It may also present with *Streptococcus pneumoniae* in upper respiratory pictures and more rarely with the toxic ingestion of drugs and/or alcohol².

Analytical tests are essential. Peripheral thrombopenia, haemolytic anaemia with schistocytosis (microangiopathic) and the presence of renal insufficiency together with a possible history of infection may help to determine the diagnosis^{3,4}. In the present case, the early suspicion even before the appearance of anaemia facilitated the speed of the initiation of plasmapheresis and probably favoured the good outcome.

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FOCAL SEGMENTARY ISCHAEMIA**Mr. Director;**

Focal segmentary ischaemia of the small intestine is an infrequent form of intestinal ischaemia (less than 5%). Preoperative diagnosis requires a high index of suspicion since physical examination and complementary tests provide few data in the absence of intestinal infarction.

A 90-year-old woman receiving treatment with dicumarinic drugs and with a history of arterial hypertension, auricular fibrillation, hypertrophic cardiomyopathy, respiratory insufficiency and umbilical herniorrhaphy arrived at the emergency department for acute abdominal pain of epigastric onset with posterior constant and very intense diffusion throughout the abdomen of 24 hours of evolution accompanied by anorexia and nausea. Physical examination showed involvement of the general status, paralytic ileus and diffuse pain on palpation with signs of peritonism. Blood analysis and radiography were normal. Abdominal echography showed a layer of free fluid at the hepatic margin and inflammatory thickening of an intestinal loop in the left parietocolic region. Given the history of fibrillation, the clinical presentation and the echography, a diagnosis of acute abdominal pain with a high suspicion of intestinal is-



Figure 1. Image of the laparotomy demonstrating segmentary ischaemia of the small intestine.

chaemia was considered versus other diagnoses such as perforation syndrome or visceral inflammation. The study was completed 6 hours after arrival with correction of anticoagulation with three units of plasma. Urgent laparotomy was performed showing free serohaematic fluid (200 cc) and irreversible signs of ischaemia in 40 cm of jejunum ruling out the presence of bridles, volvulus or internal hernias (Figure 1). Intestinal resection of 70 cm in the small intestine and manual end-to-end anastomosis were performed with a final diagnosis of focal mesenteric ischaemia of the small intestine. The patient died after 7 days due to respiratory complications.

Acute mesenteric ischaemia (AMI) represents 25% of intestinal ischaemias and is due to the deficit of blood from the upper mesenteric artery (UMA)¹. Focal segmentary ischaemia represents less than 5% of the cases of AMI^{1,2}. The causes related to this entity include the presence of strangulated hernias, cholesterol embolisms, cocaine use, radiation lesions or, as in the present case, auricular fibrillation¹. The development of collateral circulation in some cases prevents transmural infarction and thereafter clinical forms of chronic pain or intestinal obstruction may appear². In the forms of sudden onset, a differential diagnosis with other causes of acute abdominal pain should be established with the frequent reporting of disproportionate pain with respect to the physical findings¹. In the initial phases, the abdomen may appear normal but hypersensitivity, muscular contraction and positive decompression indicate the presence of intestinal infarction². Unspecific alterations in the laboratory tests may be found such as: haemoconcentration, metabolic acidosis, an elevation in LDH, creatin kinase, alkaline phosphatase, amylase, D dimmer or C-reactive protein values. Likewise, 75% of the patients have leucocytosis ($15,000 \text{ cell/mm}^3$) at the time of

assessment³. New markers are currently under study such as the determination of albumin modified by ischaemia and protein-binding intestinal fatty acids may provide diagnostic data in the future^{4,5}. Simple radiography and abdominal echography usually provide little relevant data but contribute to the differential diagnosis. Doppler echography of the upper mesenteric artery is not useful in the evaluation of peripheral veins as in the present case⁶. Abdominal computerised tomography (CT) with endovenous contrast may demonstrate thinning of the intestinal wall, intramural haematoma, arterial occlusion, pneumatosis or gas in the portal vein in advanced cases. The recent incorporation of new technologies such as angiographic CT with tridimensional reconstruction or magnetic resonance including oxymetry of the mesenteric veins may, replace diagnostic mesenteric angiography in the future^{2,6,7}. This invasive technique continues to be the gold standard for diagnosis and allows therapeutic options on the diagnosis of intestinal ischaemia⁸.

In our setting, which does not include interventionist radiologist, this test should be requested and the patient must be transferred to a reference hospital, thus, the free use and early implementation of this technique is not available. On the other hand, the presence of peritoneal signs requires the performance of urgent laparotomy or laparoscopy with excision of the segments involved and arterial revascularisation when necessary^{1,2}.

Emergency physicians should have a high index of suspicion with patients over the age of 60 years presenting in the emergency department with sudden, intense abdominal pain, an unremarkable physical examination and predisposing risk factors. The creation of multidisciplinary teams made up of emergency physicians, surgeons and radiologists is essential for early recognition and aggressive management of these cases and is the only way to achieve a reduction in the high mortality (70%-100%) observed in diagnostic failure during the stage prior to the appearance of gangrene^{1,2}.

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CEREBRAL ABSCESS AS A COMPLICATION OF SUPERATIVE OTITIS MEDIA IN A YOUNG ADULT

Mr. Director;

The current estimated incidence of intracranial complications of otogenous origin is around 0.05 – 0.36%. The most frequent endocranial complication is meningitis followed by cerebral abscess (with a mortality of up to 50%) and thirdly, thrombosis of the lateral sinus¹⁻⁷.

A 25-year-old male arrived at the emergency department for pain in the left ear which was diagnosed with otitis and treated with paracetamol and a macrolide. He was allergic to penicillin and had had several episodes of otitis in the previous 2 years, with the last having been around three months before. Two days later he returned to the emergency department due to persistence of otalgia in addition to pulsating headache, fever and vomiting. He was conscious, oriented, with neck rigidity and yellowish suppuration was observed from the left extreme auditory tube (Figure 1). Blood pressure was 120/60 mmHg and the temperature was 38.2°C. Given the general involvement and the headache a lumbar puncture (LP) was performed (fundus of the eye was not previously performed) with the following results: leucocytosis 686 ml (77% PMN), glucose 40 mg/dL/glycaemia 108). Gram staining did not show microorganisms. With these data a cranial computerised tomography (CT) was carried out (Figure 1) demonstrating suppurating otitis media with an encephalitis-cerebritis foci. Treatment with vancomycin, aztreonam and metronidazol was initiated and radical mastoid surgery

of the left ear was carried out. Due to an unfavourable outcome drainage of the abscess was performed. The picture finally resolved and the patient was discharged after 42 days.

The usual origin of an otogenous abscess is direct or adjoining propagation. Bacteriological studies most frequently demonstrate the presence of Enterobacteria such as *Proteus*, *Pseudomonas* or *Escherichia coli* and *Staphylococcus aureus* and *Streptococcus*^{1,2,4-8}. The increase in Gram negative bacilli, anaerobes and the association between different bacteria in recent years is of note. In addition, it should also be taken into account that fluid cultures obtained from the abscess are sterile in 25% to 30% of the cases^{6,8}.

The neurological symptoms are the most important and correspond to an increase in intracranial pressure (ICP): headache, nausea, vomiting and lethargy. Between 30% to 50% of the cases report hemiparesis and convulsions^{6,8}.

Today, diagnosis with imaging tests is fundamentally based on CT. In the phase of cerebritis there is no enhancement. Cerebral magnetic resonance (MR) is more useful than CT in detecting multiple cerebral abscesses and in differential diagnosis.

The role of LP is very debateable since it does usually provide relevant information except in cases with ventricular drainage mild leucocytosis and an increase in proteins may be observed. In addition, on very few occasions, the microorganism responsible for the infection may be identified on LP and cultures are positive in 6%-22% of the cases⁸, thus, LP is only recommended on suspicion of meningitis or subarachnoid suppuration (after ruling out intracranial hypertension)^{6,8,11}.

Differential diagnosis should be performed with the remaining otogenous intracranial complications such as lateral sinus thrombosis, otoge-



Figure 1. Supuration of otitis media through the external auditory tube (left). Cranial CT with a hypodense area* which is enhanced after the injection of contrast (right).

nous meningitis or subarachnoid or epidural empyema and with expansive intracranial processes.

Treatment of otogenous intracranial abscesses is both medical and surgical. First choice antibioticotherapy includes the use of a third generation cephalosporin such as ceftazidime (1.2 g/8 h) or cefotaxime (1.2 g/8 h) combined with metronidazole (500 mg/8 h)⁷.

Ear surgery generally consists in a radial mastoidectomy. Neurosurgical intervention consists in aspiration-puncture (allows material for culture and diagnosis confirmation to be obtained as well as rapid alleviation of intracranial pressure with little aggressiveness and local anaesthesia) or exeresis by open surgery (for some types of abscesses, such as multiloculi abscesses, due to the difficulty in complete aspiration).

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