C) BRIEF CASE REPORTS

HT-pre-eclampsia-postpartum haemolytic-uraemic syndrome: good results can be achieved

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Dear Editor,

Haemolytic-uraemic syndrome (HUS) is a microvascular disorder defined by microangiopathic haemolytic anaemia and thrombocytopenia, which mainly affects the kidneys and is characterised by haematuria, oligoanuria and kidney failure.[1] There is a group of patients (5-10%) with atypical HUS usually associated with immunosupressor users, oral contraceptive users, pregnancy and postpartum. These cases tend to have a poor evolution and a high mortality rate.[2]

We present the case of a 32-year-old woman, with no relevant history of disease. She was 32 weeks pregnant, attending an obstetric check-up and presented oedema on the face and limbs, blood pressure 220/140mmHg and proteinuria 3g/l. She was diagnosed with preeclampsia.

She is referred to the nephrology department due to poor hypertension (HT) control, despite treatment with alpha-methyldopa and labetalol. After foetal maturation, an emergency C-section is performed, with initial improvement of the HT and oedemas.

Three days later, the patient complained of migraine and blurred vision along with HT. The blood work shows kidney function deterioration and haemolysis: creatinine 3mg/dl, haemoglobin 8.7g/dl, platelets 45,000/ml, haptoglobin <0.24g/l, bilirubin 1.5mg/dl, LDH 6,686U/l and 6% schistocytes in peripheral blood smear.

This data reveals a thrombotic microangiopathy (TMA) related to the pregnancy. Its appearance in the postpartum and the manner that it generally affected the kidney, lead us to diagnose the patient with HUS and commence early treatment with plasmapheresis (PP) and prednisone (1mg/kg/24h).

The patient worsened, presenting acute pulmonary oedema during the first PP treatment, which meant that she had to be admitted into the Intensive Care Unit to start haemodialysis. Given that the haemolysis persisted, an abdominal ultrasound is performed to rule out any process that may cause the symptoms to perpetuate.

The ultrasound showed that there were no placental remains, but a subcutaneous haematoma from surgical wound is visible. It was drained and empirical antibiotic treatment with clindamycin and levofloxacin started. The patient showed progressive analytical and clinical improvement (Figure 1).

Fifteen PP sessions and intermittent HD were needed until remission of haemolysis and improvement of kidney function. On discharge, the patient presented creatinine 2mg/dl, MDRD 40, low proteinuria (0.5g/l) and adequate BP control with quadruple therapy (ACE inhibitors, calcium antagonists, diuretics and alpha blocker).

In the six-month check up, kidney function was normal with minimum proteinuria and treatment is maintained only with an angiotensin receptor blocker (ARB).

There are two major entities characterised by pregnancy-related TMA: severe preeclampsia (generally

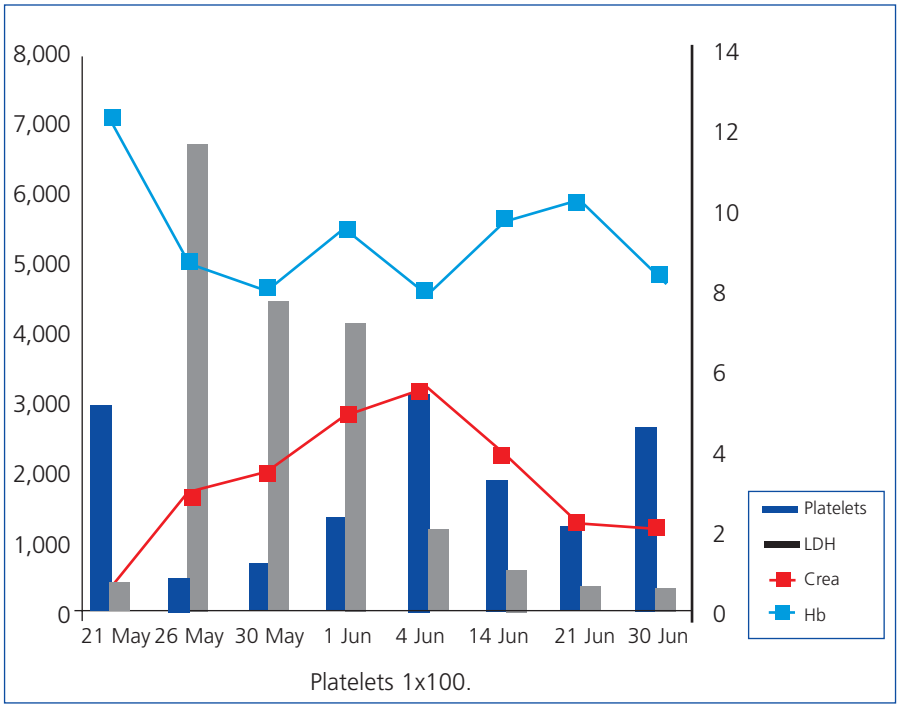

Figure 1. Graph of the analytical evolution of the symptoms.

with HELLP syndrome) and thrombotic thrombocytopenic purpura (TTP)-HUS. It is difficult to differentiate between these entities given that various clinical and laboratory findings overlap. This means that it is important for a precise diagnosis to be made as early as possible, given that the medical treatment and the complications involved can de different.[3]

TMA pathogenesis during pregnancy or in the puerperium period is unknown and in recent years studies have found that mutations in various genes involved in regulating the complement would cause tissues to lose their protective capacity (factor H 20% and factor I 15%). Situations associated with complement inflammation and activation, such as pregnancy or infections (in our case, subcutaneous abscesses), could trigger or perpetuate the process in genetically predisposed people. The final aspect would be the microvascular damage and platelet activation with multiple organ injury.[4]

Plasmapheresis has proven to be the therapy of choice, improving survival up to 80-90% and achieving good kidney function evolution results.

With this case, our aim is to stress that good results can be achieved for this disease with early treatment and to emphasise that it is essential that secondary processes that could perpetuate symptoms are diagnosed and treated.

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Hydrothorax in peritoneal dialysis: a rare peritonitis complication

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Dear Editor,

Hydrothorax is a peritoneal dialysis complication, associated with an increase in intra-abdominal pressure,[1] which on very rare occasions has been reported to be related with peritonitis episodes.[2,3] We present the case of a patient who, after having had other complications related with an increase in intra-abdominal pressure, developed hydrothorax during the course of a peritonitis episode.[4]

She is a 76-year-old woman, with stage 5 chronic kidney disease with interstitial nephropathy. Over the past three years, she has been receiving continuous ambulatory peritoneal dialysis, with three daily 2,000ml refills. Over this period, she has not suffered peritonitis nor has she undergone surgery. In a routine check-up, a small non-incarcerated umbilical hernia and an abdominal hernia were detected, the latter measuring approximately 6cm in diameter in the left paramedian line and confirmed by CT scan and ultrasound. The peritoneal dialysis was not affected by these anatomical alterations, so after surgical evaluation a conservative stance was maintained, modifying the dialysis rate to four daily 1,500ml refills. She returned to the hospital six months later with cloudy fluid, fever and mild abdominal pain, with a 5 hour evolution. In the days before, she had suffered mild diarrhoea. The peritoneal effluent cell count was 4,570cells/ml (98% PMN). Diagnosed with bacterial peritonitis in peritoneal dialysis, she was admitted and put on intraperitoneal antibiotic therapy, following our centre's protocol: vancomycin, ampicillin and tobramycin. On the following day, the fluid was still cloudy and cell count was 9,850cells/ml (94% PMN); the peritoneal dialysis balance showed a gain of 1,200ml and the abdominal pain persisted. Thirty-six hours after admittance, she suffered sudden dyspnoea. The patient presented a generally poor state: she was pale, sweaty, tachypneic and had difficulty breathing. Oxygen saturation was 90%. The respiratory auscultation displayed a reduced breath sounds in the right hemithorax. The chest X-ray displays a moderate right pleural effusion (Figure 1). A short hypertonic refill is administered with no improvement. Due to paroxysmal breathing difficulty and the X-ray results, thoracocentesis was performed, which drained 2,000ml. The pleural fluid was a glucose concentration of 269mg/dl, much higher than the plasmatic concentration (110mg/dl), which confirmed hydrothorax. Improvement was significant after the pleural tap. The peritoneal dialysis was suspended and on receiving the peritoneal fluid culture, positive for *Bacteroides fragilis*, intravenous antibiotic therapy was started with meropenem and metronidazole. The abdominal CT scan ruled out perforation and other intra-abdominal pathologies. After two weeks of treatment, she was discharged and is currently on regular haemodialysis.

Hydrothorax consists of peritoneal fluid passing to the pleural space through a peritoneal-pleural structural