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Cytomegalovirus colitis

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To the Editor,

Our patient was a Bolivian woman diagnosed with systemic lupus erythematosus (SLE) in 2003. In July 2008, she developed type IV lupus glomerulonephritis and treatment was started with i.v. methylprednisolone followed by oral prednisone at doses of 1mg/kg/day and mycophenolate mofetil (MMF). She was hospitalised for a month due to respiratory symptoms. *Nocardia spp* were isolated from the sputum culture, antibiotic treatment begun and immunosuppressive medication was reduced.

During hospitalisation, the patient developed fever, abdominal pain, vomiting and diarrhoea. Culture studies, parasite study, identification of *Clostridium difficile* toxin and antigenaemia for cytomegalovirus (CMV) were performed on two occasions, and the results were negative. An abdominal CT (Figure 1) showed a thickening of the wall of the colon with occlusion of the lumen from the cecum to the rectum-sigmoid junction, which was compatible with diffuse pancolitis. A fibrocolonoscopy was requested (Figure 2), which revealed oedematous mucosa with multiple soft nodular lesions compatible with pneumatosis *coli*. The colon biopsy revealed viral inclusions which were confirmed for CMV by immunohistochemistry (Figure 3). The third antigenaemia determination for CMV was positive and the definitive diagnosis was CMV colitis. Treatment with i.v. ganciclovir was begun and the MMF treatment withdrawn, with clinical improvement and a negative antigenaemia for CMV.

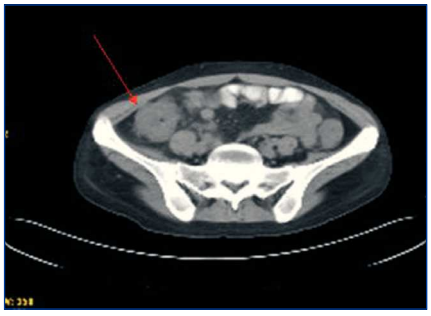

Figure 1. Abdominal TC.

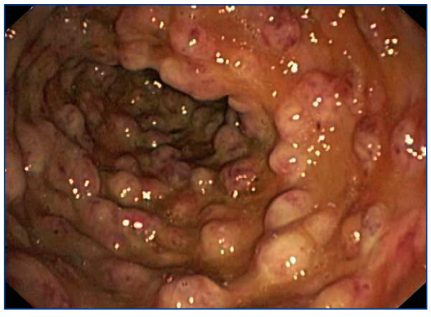

Figure 2. Fibrocolonoscopy.

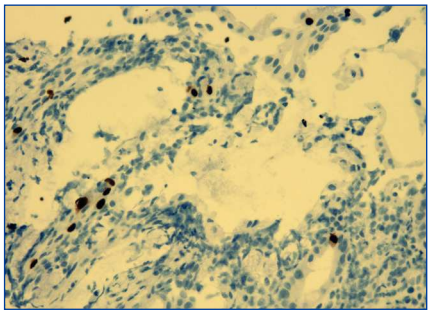

Figure 3. Colon biopsy.

SLE is a chronic autoimmune inflammatory disease of unknown cause with a wide variety of clinical presentations.

It is characterised by an alteration in the immune system which involves the synthesis of autoantibodies and the formation of immune complexes which cause tissue damage, along with the action of inflammatory mediators.

Lupus disease itself and the use of immunosuppressive agents increase the risk of opportunistic infections causing increased morbidity and mortality. In general, patients have bacterial infections, however, there is an increase in viral infections, due especially to CMV, but also to human parvovirus B_{19}, simplex herpes, varicella zoster and hepatitis A.[1]

Viral infections may have symptoms identical to those of SLE, such as malaise, fever, arthralgia, a rash, lymphadenopathy and cytopaenias, so it may be confused with an outbreak of SLE.[2,3] This may lead to increasing immunosuppressive therapy, thereby worsening the latent infection.

In our case, the patient developed an invasive CMV disease despite reducing the immunosuppressive treatment. The persistence of abdominal symptoms without encountering an infectious cause, the finding of non-specific lesions in the colonoscopy and reduced immunosuppression, made us suspect that it was an outbreak of SLE with abdominal involvement. The anatomopathological study allowed us to make the diagnosis of CMV colitis, and administer appropriate treatment to prevent further immunosuppression.

We believe it important to maintain a high degree of suspicion for both bacterial and viral opportunistic infections that mimic outbreaks of lupus disease in immunosuppressed patients. They will benefit from prompt diagnosis and appropriate treatment.

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Methanol poisoning. Evolution of blood levels with high-flux haemodialysis

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To the Editor,

Although rare in our area, and related to accidents or suicide attempts, methanol poisoning is increasing due to the arrival of immigration.[1] We report the case of a man with severe methanol poisoning treated with haemodialysis (HD). Serial samples were taken to assess the decrease of blood methanol levels during dialysis.

Case report

A 38-year old male with a history of alcoholism was admitted to the emergency department with visual disturbances, abdominal pain and vomiting.

For days before, he had been drinking perfume and 96° alcohol from a chemist. The day before admission, he voluntarily ingested 200ml of methylated spirit and ethanol (not knowing it was toxic). He was admitted to the emergency room with blurred vision, unsteadiness, epigastric pain and an episode of vomiting which could not be distinguished as containing blood or not. He also reported fatigue and dyspnoea.

Physical examination in the emergency department

His breathing rate was 40 breaths per minute, with right hypochondrium pain. No bowel sounds were detected. His pupils were dilated, poorly reactive, and he was very sleepy. There was no language impairment, nystagmus or dysmetria in the finger-nose test.

Additional tests in the emergency department

Arterial blood gases: pH 6.99; pCO_2 8mm Hg; pO_2 138mm Hg; bicarbonate 3mEq/l.

Biochemistry: glucose 132; urea 32, creatinine 1.15mg/dl; Na 133, K 5.5; Cl 101mEq/l; osmolality 371mOsm/kg.

Haemogram: WBC 23 400 (N 78, C 11, L 9, M 2); Hgb 18.3g/dl; Hct 56%; platelets 264 000.

Osmolar gap calculation: 92.3mOsm/kg (osmolality measurement - {Na [mEq/l] x 2 + urea/6 + glycaemia /18 (mg/dl)}).

Anion gap calculation: 34.5mEq/l ([Na + K] - [Cl + HCO_3]).

Methanol poisoning was suspected and 1M bicarbonate was prescribed and the patient was transferred to the ICU, where ethanol infusion is started. The patient was intubated for agitation and respiratory depression. The intravenous route was accessed and HD was begun with the following programme: two consecutive sessions of 4 hours, with a change of filter in between, to prevent blood clots and loss of efficiency.

Monitor: Fresenius 4008S (Fresenius Medical Care).

Duration: 4 + 4 hours. Only stopped to change the filter and lines and purge the new system (10 min).

Membrane: polyarylethersulphone (Arylane M9, Gambro). Surface: 2.01m^2; UF coefficient 23ml/h/mm Hg, C. urea (Qb 300 and Qd 500, 264ml/min).

Qb (blood flow): 350ml/min, starting at 250ml/min, then after finding good haemodynamic tolerance at 1h, increased to 350ml/min.

Qd (dialysate flow): 800ml/min.

Dialysate: Fresenius A34 supplemented with KCl up to 4mEq/l.

UF: zero.

Anticoagulation: enoxaparin (Clexane) 20 at the start of each session.

Dose of ethanol doubled during the dialysis.

After dialysis the patient improved clinically, and was extubated and transferred to the psychiatric ward the same day.

Hourly methanol extractions were performed (at the start of HD, and then hourly) and 4 hours after ending the second session (Figure 1). The levels were significantly reduced, reaching minimum values after the third hour. However, when stopping dialysis (while changing the filter) a relapse (64mg/dl) was noted, but the patient returned to minimum values after restarting the session.