no evidence of pathological radiotracer uptake. Follow-up gastroscopy and colonoscopy showed no lesions. Given the patient's good response to intensive therapy, treatment was consolidated with autologous peripheral blood stem cell transplantation (PBSCT). One year after the PBSCT, the patient remains in full remission on maintenance treatment with rituximab.

Our patient is under 60 years of age, with the distinctive characteristic of presenting with a single large polyp instead of multiple lesions. In 90% of patients, symptoms are non-specific, and include: weight loss, asthenia, lethargy, fatigue, anaemia, palpable abdominal or rectal mass and palpable lymphadenopathy. Bone marrow involvement is seen in advanced stages.[7] In the case of gastrointestinal involvement, digestive symptoms have been reported in 15–30% of patients. Endoscopic studies are recommended mainly if there is abdominal pain, changes in bowel movement or rectal bleeding, which was observed in our patient.[6]

Mantle cell lymphoma is considered an aggressive, rapidly progressive type of lymphoma, and 80% of patients are diagnosed at advanced stages.[7] A prognosis must be performed to improve treatment. Prognostic indices permit clinicians to develop treatment strategies based on the patient's particular risk factors. The Mantle Cell Lymphoma International Prognostic Index (MIPI) is used for prognosis in mantle cell lymphoma.[8] In patients under 60 years of age and/or with a high MIPI index score, intensive therapeutic strategies with R-CHOP, R-bendamustine and/or R-DHAP followed by PBSCT are recommended.[9] Our patient had a score of 8 points on the MIPI, which indicates a high risk (estimated average survival of 29 months), and for this reason received intensive therapy with R-macro CHOP/R-DHAP followed by consolidation with PBSCT. He remains in full remission 30 months after the initial chemotherapy treatment.

Prognosis is poorer in older patients with advanced disease at diagnosis, low albumin, splenomegaly, anaemia and elevated LDH levels.[9]

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María Fernanda Guerra Veloz[a,*], María Belén Maldonado Pérez[a], Angel Caunedo Álvarez[a], Alvaro Gutiérrez Domingo[b]

[a] *Unidad de Gestión Clínica de Aparato Digestivo Intercentros, Hospitales Universitarios Virgen Macarena-Virgen del Rocío Sevilla, Sevilla, Spain*
[b] *Unidad de Gestión de Anatomía Patológica, Hospital Universitario Virgen Macarena, Sevilla, Spain*

* Corresponding author.
E-mail address: maferguerrita@hotmail.com (M.F. Guerra Veloz).
2444-3824/

Catastrophic antiphospholipid antibody syndrome presenting as severe ischaemic colitis☆

Colitis isquémica grave como presentación del síndrome por anticuerpos antifosfolípidos catastrófico

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Antiphospholipid syndrome (APS), or Hughes syndrome, is an acquired autoimmune thrombophilia characterised by venous or arterial thrombosis and/or recurrent miscarriages due to the presence of autoantibodies to phospholipid-binding plasma proteins, such as the lupus anticoagulant, anticardiolipin antibody (aCL), and the anti-beta-2-glycoprotein antibody (aβ2GPI).[1] There are some primary forms that are not associated with another autoimmune disease. Catastrophic antiphospholipid syndrome (CAPS) is a severe and rapidly progressive form that affects multiple organs simultaneously and causes multiple organ failure. Microangiopathy is more commonly found

Table 1 Causes of ischaemic colitis.

1. Atherosclerosis
2. Hypovolaemia or low cardiac output:
Heart failure
Arrhythmias
Sepsis
Haemorrhage
Dehydration
Haemodialysis
3. Large vessel occlusion (superior and inferior mesenteric artery):
Mesenteric artery thrombosis
Mesenteric vein thrombosis
Cholesterol embolism
4. Small vessel occlusion:
Hypercoagulability states
Protein C deficiency
Protein S deficiency
Antithrombin III deficiency
Prothrombin 20210A gene mutation
Factor V Leiden
Antiphospholipid syndrome
Other haematological disorders
Sickle cell anaemia
Polycythemia vera
Paroxysmal nocturnal haemoglobinuria
Thrombocytopaenic purpura
Vasculitis and vasculopathies
Buerger's disease
Wegener's granulomatosis
Fibromuscular dysplasia
Kawasaki disease
Polyarteritis nodosa
Rheumatoid vasculitis
Systemic lupus erythematosus
Takayasu's arteritis
Other
Amyloidosis
Radiation-induced lesions
Iatrogenic causes
Surgical
Aneurysmectomy
Aortoiliac reconstruction
Coronary artery bypass
Barium enema
Inferior mesenteric artery ligation during colectomy
Gastric bypass
Colonoscopy
Gynaecological surgery
Lumbar aortography
5. Colon obstructions:
Tumours
Adhesions
Volvulus
Strangulated hernia
Diverticulitis
Invagination
Faecal impaction
6. Intra-abdominal inflammatory processes:
Pancreatitis
7. Infections:
Bacterial (E. coli *O157/H7*)
Parasitic (Angiostrongylus costaricensis, Entamoeba histolytica)
Viral (norovirus, hepatitis B virus, cytomegalovirus)
8. Ruptured ectopic pregnancy
9. Pheochromocytoma
10. Long distance runners
11. Air travel
12. Irritable bowel syndrome
13. Constipation
14. Allergy
15. Idiopathic

Source: Montoro et al.[4]

in CAPS than in the classic APS form.[2] CAPS, with a mortality rate of nearly 50%, accounts for less than 1% of all cases of APS.[2] CAPS presenting as ischaemic colitis is extremely rare, and although treatment is initially medical, surgery may be required in some cases[2–4] (Table 1). We present the case of a 47-year-old man, an active smoker and former drinker with a personal history of obesity, hypertension, dyslipidaemia, complete occlusion of the left common carotid and right posterior cerebral arteries, amputation of both lower limbs due to ischaemia, carrier of the prothrombin gene mutation (G20210A) and MTHFR gene mutation, with hypercoagulability syndrome and hyperhomocysteinaemia. He was undergoing tests for suspicion of APS. The patient was admitted to the emergency department due to a flare-up of chronic abdominal pain in the previous 48 h. The physical examination showed abdominal distension with absent bowel sounds and signs of peritoneal irritation. Of note in the urgent laboratory test findings were: leukocytosis (16,500/μl) with neutrophilia (91%), thrombocytopaenia (85,000/μl), elevated CRP (132 mg/l), creatinine (2.5 mg/dl) and urea (97 mg/dl), and low haemoglobin (9.8 g/dl). The patient's condition deteriorated rapidly, with criteria for multiple organ dysfunction syndrome. Abdominal-pelvic CT angiography showed dilation of the transverse colon and caecum (9 cm), increased parietal uptake in the sigmoid and pneumatosis intestinalis, with patent coeliac trunk, superior and inferior mesenteric arteries and renal arteries (Fig. 1). Urgent colonoscopy revealed extensive mucosal ischaemia, and urgent immunological tests showed elevated aCL IgM (12 MPL U/ml; positive: >10 MPL U/ml) and fibrinogen (583 mg/dl; normal: 200–400 mg/dl), negative for aβ2GPI IgM and Russell test. Given the suspicion of severe ischaemic colitis secondary to CAPS, treatment with corticosteroids (methylprednisolone: 200 mg/day) and enoxaparin 100 mg/24 h was started. In view of his haemodynamic instability, the patient underwent emergency surgery, which revealed ischaemic colitis extending along the length of the colon and the distal ileum. Pancolectomy with a Brooke ileostomy was performed. The patient died 24 h after surgery. The full results of the autoimmunity study showed,