Mesenteric vein thrombosis associated with a cytomegalovirus infection

Key words: Cytomegalovirus. Portal thrombosis. Mesenteric thrombosis.

Dear Editor,

Acute cytomegalovirus (CMV) infection usually presents as mononucleosis syndrome in adolescence (1).

Mesenteric vein thrombosis has been described as a rare complication of acute CMV infection (2).

We report the case of a 66-years-old male admitted with fever and malaise of 1.5 months duration. He complained of intermittent abdominal pain and distension without altered bowel function or weight loss and an affected general status. The abdomen was soft, painful in the region of the mesogastrium without peritoneal irritation.

The blood count was within normal limits: Hemoglobin 13.5 g/dl (13-18), WBC 9.8 x 10^9/l (3.5-10.5), platelets 312x10^9/l (105-400). As altered parameters were: Fibrinogen 735 mg/dl (150-400), erythrocyte sedimentation rate 58 mm/h (0-10), bilirubin 1.8 mg/dl (0-1.1), conjugated bilirubin 0.8 mg/dl (0-0.4), aspartate transferase 38 U/l (0-18), and alanine transferase 33 U/l (0-22). The stool culture was negative. Computed tomography (Fig. 1) showed thrombosis of the left portal vein, the main trunk of the portal vein, splenoportal axis and the superior mesenteric vein. Serological testing for CMV showed elevated titers of IgM (95.3 IU/ml -negative < 15) and the screening for thrombophilia was negative.

With the diagnosis of portal vein thrombosis secondary to acute mesenteric CMV infection anticoagulant therapy was established for 3 months, with complete resolution in the control CT scan.

Among the inherited causes associated with portal vein thrombosis include: Mutation of factor V Leiden, mutation of prothrombin, protein C and S deficiency, antithrombin III deficiency, mutation of MTFR gene, increased factor levels VIII (3).

Venous thrombosis during acute CMV infection has a predilection for affecting the portal vein and the mesenteric territory. The exact mechanism is not well known, but there is convincing evidence of procoagulant effect in vitro of CMV (4-6). Recently, a prospective study found that CMV seropositivity was independently related to venous thrombosis, which supports the results of in vitro studies (7).

Fig. 1.
We found 19 cases of mesenteric thrombosis associated with CMV infection in the literature. The outcome was usually favorable with anticoagulant therapy for a period lasting 3 to 7 months. One had protein C and S deficiency, one was heterozygous for the factor V Leiden, two were heterozygous for G20210A factor, and seven women taking oral contraceptives. Fifteen of the nineteen had complete resolution without complications. Of these, one did not receive anticoagulant therapy. The remaining four who had consequences, had at least one risk factor for thrombosis (one protein C and S deficiency, another heterozygous prothrombin G20210A mutation, another oral contraceptives and other heterozygous for factor V Leiden). These data suggest a transient natural history of portal thrombosis with a high rate of spontaneous resolution, which is probably underestimated (8,9).

In conclusion, acute infections are associated with a transient increased risk of thromboembolic events. In all patients with thrombotic complications is indicated a screening for thrombophilia. Mesenteric thrombosis is a serious condition that involves high mortality, therefore, in all patients with CMV infection presenting abdominal symptoms should be ruled out this complication, to start anticoagulation as soon as possible (1,2).

Jorge Rojo-Álvaro1, Sara Pérez-Ricarte1, Miren Vicuña-Arregui2, Ione Villar-García1 and Francisco Javier Anniccherico-Sánchez1

Departments of 1Internal Medicine and 2Digestive Diseases. Complejo Hospitalario de Navarra. Pamplona, Navarra. Spain

References