ACUTE PANCREATITIS PRESENTING AS SUDDEN BLINDNESS

PÉRDIDA DE VISIÓN COMO DIAGNÓSTICO DE PANCREATITIS AGUDA

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ABSTRACT

Case report: A 36-year-old man with a history of alcohol abuse presented with sudden blindness. The ophthalmologic examination showed Purtscher-like retinopathy. The presumed diagnosis was acute pancreatitis, which was confirmed by complementary laboratory studies.

Discussion: Sudden acute visual loss with Purtscher-like retinopathy may be present in acute pancreatitis, although it is a very rare as a presenting symptom. Early diagnosis based on ophthalmic symptoms may help in the recognition and treatment of the disease and prevent later complications (Arch Soc Esp Oftalmol 2006; 81: 161-164).

Key words: Acute pancreatitis, Purtscher-like retinopathy, Sudden blindness

INTRODUCTION

Purtscher-like retinopathy is characterised by a sudden reduction of visual acuity (VA) associated to ophthalmological signs of ischaemia in the posterior pole in one or both eyes, with whitish exudate and haemorrhage around the papilla and in the posterior pole of patients who suffered severe trauma, particularly in the head or chest. There may be cases not associated with trauma, known as Purtscher-like retinopathy, which include associations with acute pancreatitis. The first description of this association was made in 1975 (1) by Inkeles and Walsh. Usually, ophthalmological alterations appear in the first week after the condition has expressed, and in exceptional cases the beginning of acute pancreatitis is preceded by a sudden reduction of visual acuity and a Purtscher-like retinopathy (2,3).
CLINICAL CASE

A 36-year old man, with a history of drinking 350 gr/alcohol/day, smoking 20 cigarettes/day, former heroin and cocaine addict, VHB +, VHC +, VIH -, and multifactorial chronic hepatitis and Child’s stage A, without portal hypertension data. The subject went to the urgency ward of our hospital due to sudden and important loss of visual acuity within a period of several hours. The ophthalmological exploration showed visual acuity with movement of hands in both eyes, with eye fundus with infiltrate in the posterior pole and macular serum detachment (fig. 1). The remainder of the ophthalmological exploration yielded normal results. In addition, the subject had a temperature of 38.4ºC and a discreet abdominal pain with palpation and hepatomegalia measuring two fingers across.

As acute pancreatitis was suspected, the patient was referred to the Internal Medicine Service which confirmed the diagnostic after performing lab analysis with the following results: Leucocytes 12.4 (4.8-10.8 x10³ ul); Neutrophiles 88% (42.2-75.2); Mean Corpuscle Volume 111 fl (85.9-95.9); Bili-rrubine total 8.23 mg/dl (0.2-1.0); Amylase 1.523 u/l (55-201); GOT 200 u/l (5-45); GPT 227 u/l (5-45); GGT 1.346 u/l (3-52); Alkaline phosphatase 1.159 u/l (98-295); Fibrinogen 157 mg/dl (200-400) and a CAT and abdominal echograph showing a fatty liver problem and infiltration of the peripancreatic tissue, particularly at the level of the tail, where infiltration continued along the anterior left pararenal space. Fluoresceine angiography initially showed areas without capillary perfusion corresponding to the presence of exudate followed by belated diffusion and screening effect caused by retinal haemorrhage. The outcome of the digestive condition was favorable with a medical treatment based on serum, absolute diet and antibiotic therapy which produced a rapid improvement of clinical symptoms and an important improvement of analytical results within two weeks, although one month later the patient presented Wernicke’s encephalopathy with normal cranial CAT scan.

Two months later the patient had recovered a visual acuity of 1 in AO and the eye fundus study showed only residual pigments (fig. 2).

DISCUSSION

Purtscher-like retinopathy can appear in acute pancreatitis, chronic renal insufficiency or after labour. Retina damage is due to the conclusion of the arterioles with microparticles generated by the underlying disease. Although this alteration was considered to be restricted to the internal retina, clinical and pathological studies have demonstrated an involvement of the chorio capillar (4). Generally, symptoms begin within 48 hours of the precipitating event. Our case was exceptional due to its presentation, which preceded the clinical symptoms of acute pancreatitis, thus allowing an early treatment thereof. In these cases it has been suggested that the cau-
se lies in the formation of micro-emboli of granulocytes by activation of the supplement as a consequence of the release within the systemic circulation of proteolytic enzymes from the inflamed pancreas (5), although this viewpoint is under debate.

The prognosis for visual recovery is uncertain, with a resolution of the lesions within about 4 months, although the visual function may not recover completely. Our case had a full recovery of visual acuity. There is no known treatment for this condition. The Purtscher-like retinopathy associated to acute pancreatitis is described in cases related to consumption of alcohol, although the role played by the latter is not known. The presence of retinopathy is not related to the severity of the acute pancreatitis nor is it considered to be a factor in the prognosis thereof. Our case had a favourable evolution with the acute pancreatitis treatment, without needing to prescribe steroids.

The above case reminds us that 2% of patients with acute pancreatitis may present atypical expressions, including sudden loss of VA without other clinical causes except abuse of alcohol and chronic liver conditions. Taking this possibility into account allows for a presumptive diagnosis which, in these cases, can be important for preventing complications in the development of acute pancreatitis.

REFERENCES