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Acute Pancreatitis Revealing Giant Cell Arteritis

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Abstract

***Introduction:** Giant cell arteritis (GCA) is a primary granulomatous vasculitis of large caliber arteries, with typically segmental and focal involvement, and predominant in cephalic territory. Digestive complications during this disease are rare and far dominated by mesenteric ischemia. Pancreatic involvement remains exceptional and unusual. We report an original observation of acute pancreatitis inaugural of GCA.*

***Case report:** A 62-year-old patient with no pathological medical history was explored for acute abdominal pain. Biological and radiological investigations led to grade A acute pancreatitis. Subsequent etiological investigations did not reveal any underlying etiology, in particular biliary, metabolic, tumoral, malformative, or medicinal. Computed tomography showed thoracic aortitis whose subsequent investigations related to an evolutive GCA: erythrocyte sedimentation rate at 100 mmH1, absence of left temporal pulse and appearance of arteritis with giant cell and fragmentation of the internal elastic lamina at the biopsy of the temporal artery. Ophthalmological examination was normal and anti-phospholipid antibodies were negative. The patient was treated with oral corticosteroid therapy at a dose of 0.7 mg /kg/day and acetylsalicylic acid at a dose of 100 mg/day with favorable evolution.*

***Conclusion:** Our observation is characterized by the inaugural character of the disease by this digestive complication, classically exceptional and unusual during GCA. A pancreatic localization of vasculitis is the most plausible mechanism for this pancreatitis. It is thus advisable to evoke the GCA in front of a pancreatitis which does not prove itself, in particular in the elderly.*

Keywords: *Acute pancreatitis, Giant Cell Arteritis, Horton's disease, Vasulitis, Angiitis.*

Introduction

Giant cell arteritis (GCA), also known as temporal arteritis or Horton's disease, is a primary systemic vasculitis of large caliber arteries, with a predilection for divisional branches of the aorta, especially cephalic arteries [1,2]. It mainly affects the subject of more than 50 years, and seems to be more common in the Nordic (Scandinavian) countries [1,3,4].

Its prevalence is poorly known and its annual incidence varies widely by country; it ranges from 1/100,000 to 22/100,000 [1].

His clinic is largely dominated by cranial involvement, with headaches, claudications of the jaws, ocular mani festations, and secondarily rhizomelic pseudopolyarthritis or plymyalgia rheumatica [1, 3,4]. The systemic character of this angiitis explains the possibility of very polymorphic and sometimes severe visceral involvement, which can be seen even outside of any classical cephalic involvement of the disease [5]. These manifestations are called «extra-cranial» [6-8] and their frequency is estimated according to clinical series at 3-15% [9].

This frequency seems to be very overestimated since, most often, extra-cranial involvement is not confirmed histologically in the majority of reported cases; indeed, in the large Scandinavian autopsy series, the post mortem histological examination of 889 patients with GCA objectified extra-cranial involvement in only 1.4 to 1.7% of cases [10].

Thus, several anecdotal visceral involvements have been reported during this vasculitis: heart, kidneys, intestines, spleen, prostate, liver, peripheral nerves, skin ... etc. [5-7], making this disease a real diagnostic

challenge for clinicians , particularly in the so-called pauci-symptomatic, atypical, silent or unusual forms (masked GCA) [4,8,9,11,12].

Pancreatic involvement remains exceptional during this angiitis [5,13-15], but most often serious or even fatal [14,15].

We report an original observation of acute pancreatitis inaugural of a GCA.

Case Report

A 62-year-old patient with no pathological medical history was explored for acute abdominal pain that had been evolving for two days and not improved by the common analgesics and antispasmodics prescribed by his family doctor. He has been associated with it, since the morning of his admission, two simple episodes of vomiting.

The somatic examination was poor, especially the patient was well conscious, apyretic, and had a stable hemodynamic state. There was just a diffuse pain caused by palpation of the abdomen, with a maximum in the epigastric region. No gastrotoxic drugs intake, no alcohol consumption, or digestive bleeding was revealed.

The basic biology was without abnormalities: leukocytes, hemoglobin, platelets, glycemia, creatinine, ionogram, liver enzymes, serum calcium, total cholesterol, and triglycerides, as well as X-rays of the thorax and abdomen without preparation, and electrocardiogram.

On the other hand, there was a marked biological inflammatory syndrome with an erythrocyte sedimentation rate (ESR) at 100mmH1 and a C-reactive protein at 23 mg/l without evident infectious focus. The infectious survey was negative.

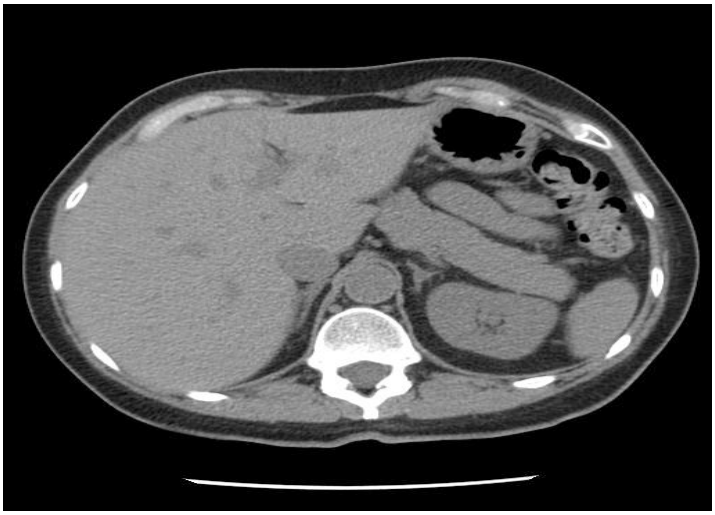

Fig 1. *Axial abdominal CT scan without injection: homogeneous enlargement of the pancreas with polylobed.*

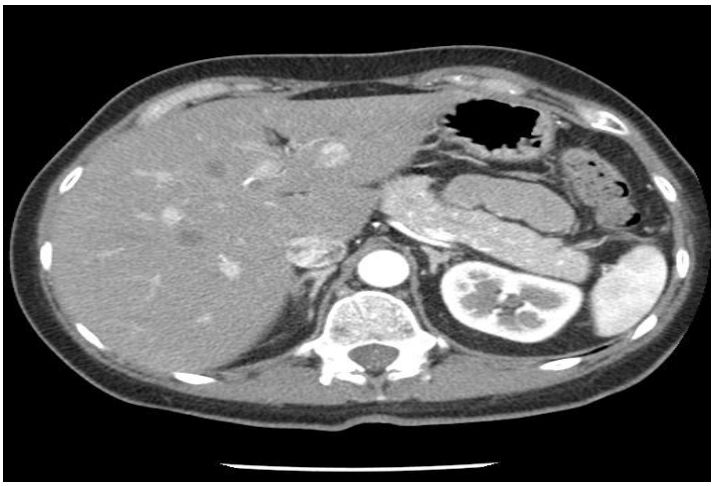

Fig 2. *Axial abdominal CT scan with injection: homogeneous enlargement of the pancreas with polylobed contours without necrosis and other abnormalities.*

Amylasemia was increased (400 IU/l) suggesting acute pancreatitis. This diagnosis was subsequently confirmed by the elevation of amylasuria, lipaseemia (4-fold normal), and CT scan data showing homogeneous increased in pancreas volume with polylobed contours (***Figs 1 and 2***) compatible with the diagnosis of acute pancreatitis stage A of Balthazar. No pancreatic calcifications, malformation or tumors, biliary or gallbladder lithiasis, intra-abdominal tumors or lymphadenopathies was noted on this examination.

Thoracic computed tomography showed regular, circumferential, and homogeneous contrast-enhanced thickening in the thoracic aorta compatible with the diagnosis of inflammatory aortitis.

Given this aspect of aortitis, the high ESR, and the age, as well as the negativity of the infectious and tumoral investigation, the diagnosis of GCA was evoked. The resumption of the detailed somatic examination noted the absence of left temporal pulse. The temporal biopsy performed at this level was positive confirming the diagnosis by showing focal and segmental arteritis with giant cell and fragmentation of the internal elastic lamina.

The ophthalmologic examination was without abnormalities and the anti - phospholipid auto antibodies were negative.

The patient was treated with oral corticosteroid therapy at a dose of 0.7 mg/kg/day combined with acetylsalicylic acid at a dose of 100 mg/day with a favorable course for both pancreatitis and temporal arteritis. Pancreatic CT was normal at two weeks of treatment, and chest CT showed a strictly normal aorta at six months of treatment. No recurrence has been noted four years later.

Discussion

Digestive disorders during GCA remain rare and are largely dominated by intestinal necrosis and perforation in relation to mesenteric infarcts [16-18] caused by a specific mesenteric localization of temporal arteritis [17,18]. These digestive disorders can sometimes be the first signs revealing the disease [16].

Acute pancreatitis remains an exceptional and unusual complication during GCA; indeed, only two cases of acute pancreatitis following a histologically confirmed GCAwere found in the world literature [13,14]. In both cases pancreatitis was a complication of systemic corticosteroid therapy at high doses and not of vasculitis itself [13,14].

On the other hand, a few sporadic cases of acute pancreatitis of vasculitic mechanism proved by histological examination (pancreatic giant cell-vasculitis) were noted on post mortem examination, without cephalic involvement characteristic of an associated GCA [5,15]; these localized forms of GCA led some authors to define a distinct clinical entity called «disseminated visceral giant cell arteritis» [5,15].

The onset of acute pancreatitis during GCA is often a fatal accident [5,14].

In our observation, the concomitant occurrence of acute pancreatitis and aortitis, the highly inflammatory and progressive character of GCA, the negativity of the etiological investigations for acute pancreatitis, as well as the rapid improvement under systemic corticosteroid therapy, made it possible to relate directly this acute pancreatitis to temporal arteritis. The plausible mechanism for this pancreatitis is that of pancreatic giant cell vasculitis (pancreatic localization of GCA). To our knowledge this is the first case of acute pancreatitis secondary to GCA reported in the literature, it is characterized in addition by the revealing character of the disease that presented this exceptional complication.

During GCA, and Apart from Cortico-Induced Cases, Acute Pancreatitis Could be Explained, at Least Theoretically, by Three Mechanisms:

- A specific pancreatic localization of GCA (pancreatic giant cell vasculitis); histologically proven cases in the context of a «disseminated visceral giant cell arteritis» simulating GCA reinforced this hypothesis [5,15].
- Autoimmune pancreatitis, especially since now the autoimmune nature of GCA is increasingly evoked and confirmed by several studies [19].

- A specific complication of the antiphospholipid syndrome (pancreatitis of necrotic micro-thrombotic and/or autoimmune mechanism); Indeed anti-phospholipid antibodies are found in more than half of cases of GCA [20], and the occurrence of acute pancreatitis, with both mechanisms, during the anti phospholipid syndrome was also reported [21,22].

Conclusion

As exceptional as it is, this unusual complication deserves to be kept in mind during the management of GCA, especially at the beginning of the treatment by systemic corticosteroids. The diagnosis of temporal arteritis must be evoked and screened in front of any acute pancreatitis that is not proven, in particular in elderly.

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