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Full Length Research Paper

Polyarteritis nodosa associated with hepatitis C virus infection

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Abstract

Polyarteritis nodosa (PAN) is a systemic, necrotizing vasculitis of medium and small arteries, without glomerulonephritis and not associated with the presence of ANCA. This is a rare condition occurring at any age, with a slight male predominance. In the majority of PAN cases, there is no evidence of aetiology. However, an infectious cause is sometimes found, foremost among which is infection with the hepatitis B virus. Hepatitis C virus, on the other hand, is little associated with vasculitis. We report here a case of PAN associated with hepatitis C virus infection in a 40-year-old patient, a former drug addict, whose progress under treatment was satisfactory.

Keywords: Polyarteritis nodosa, hepatitis C virus, vasculitis, drug addict, Dijon.

INTRODUCTION

Polyarteritis nodosa (PAN) is a systemic, necrotizing vasculitis of medium and small arteries, without glomerulonephritis and not associated with the presence of ANCA [1]. The clinical manifestations of the classic PAN combine with an alteration of the general condition, vascular purpura, subcutaneous nodules, multineuritis, articular manifestations, diffuse myalgia, renal impairment in the form of renal infarction with arterial hypertension. Severe, ischemic digestive impairment and more rarely myocardial involvement [2]. It is a rare condition (prevalence = 1 / 33,000) occurring at any age, with a slight male predominance [3]. In the majority of P cases, there is no evidence of aetiology. However, an infectious cause is sometimes found, prominent among which is infection with the hepatitis B virus (HBV) [2]. Hepatitis C virus (HCV) is poorly associated with this vasculitis. We report here a case of PAN associated with HCV infection in a young patient who was a drug addict.

Observation

A 35-year-old patient was admitted for pain in the right hypochondrium on the background of a deterioration of the general condition evolving for 3 months with a 10 kg weight loss, anorexia and the appearance of inflammatory periarthralgia with myalgia in an apyretic context. As a history,

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the patient was a carrier of chronic hepatitis C in a context of drug addiction withdrawn for 1 year. There was no notion of recent travel, nor of contagion. The physical examination found abdominal pain with defense of the right hypochondrium and a blood pressure of 160/110 mmHg, without skin or neurological abnormalities.

Biologically, there was an inflammatory syndrome with CRP at 90 mg / L, moderate cholestasis with Gamma GT at 139 IU / L and alkaline phosphatases at 134 IU / L. The serum creatinine was 61 µmol / L with proteinuria at 0.47 g / 24H. The test for cryoglobulinemia was positive at 189 mg / L, type III. ANCAs were negative and C4 complement collapsed at 0.074 g / L. CMV, HBV, syphilis and HIV serologies were negative. HCV serology was positive, genotype 3a, with a viral load of 2,693,310 IU / mL.

Abdominal imaging with ultrasound, CT scan and biliary MRI found an alithiasis gallbladder with an appearance of thickened gallbladder walls consistent with cholecystitis. The CT scan found a gastroduodenal artery aneurysm (Figure 1) and several small pancreatic artery aneurysms (Figure 2) as well as renal infarctions. The brain MRI was unremarkable. A transesophageal ultrasound was also performed which ruled out endocarditis. In this context, the main diagnostic hypotheses were either cryoglobulinemic vasculitis or polyarteritis nodosa linked to HCV.

A cholecystectomy was then performed along with a neuromuscular biopsy. Neuromuscular histology noted lesions of vasculitis affecting the medium-caliber arteries. The arterial wall was the site of a dense, predominantly mononuclear inflammatory infiltrate associated with scattered eosinophils. In some vessels, aspects of fibrinoid necrosis of the intima, without thrombosis were visible. These were mainly CD3 + T lymphocytes (CD4> CD8) labeled with anti-interferon α antibody, associated with CD68 + macrophages and a few CD20 + B lymphocytes.

Histological analysis of the cholecystectomy specimen showed chronic fibro-inflammatory changes in the gallbladder wall with focal necrotizing vasculitis lesions, comparable to those described in muscle biopsy. These vasculitis lesions were accompanied by fibrinoid endarterial necrosis.

The patient, initially neurologically asymptomatic, developed 2 months after paresthesia of the limbs. A new EMG revealed an essentially asymmetric axonal profile suggesting a confluent multiple mononeuropathy against the background of the initial demyelinating involvement.

An initial EMG was suggestive of diffuse axonal demyelinating neuropathy and the patient underwent a lumbar puncture which did not reveal meningitis or hyperproteinorrachia or intrathecal synthesis of immunoglobulins.

Faced with these anomalies, we retained the diagnosis of PAN-type vasculitis with multisystem involvement in the context of HCV infection.The patient was initially put on antiviral treatment with Sofosbuvir and Daclatasvir in combination with corticosteroid therapy. Given the worsening observed on the neurological level, one month later he benefited from plasma exchanges relayed by boluses of cyclophosphamide which allowed a good improvement in his peripheral neuropathy. The course was marked by healing of hepatitis C with disappearance of HCV after 12 weeks of treatment and complete remission of PAN with disappearance of symptoms and aneurysms (Figure 3).

DISCUSSION

Classical polyarteritis nodosa is histologically characterized by inflammatory infiltrates rich in neutrophils associated with fibrinoid necrosis of the media of small and medium-caliber arteries [2]. The lesions observed can be segmental and of various ages [4]. At the initial stage, there is a thickening of the vascular walls, particularly the intima and the media, with edema, fibrinoid necrosis predominant in the internal part of the media and sometimes destruction of the internal elastic limit. It is associated with an inflammatory infiltrate with a majority of neutrophils and some eosinophils and lymphocytes [3]. In the most advanced lesions, the inflammatory infiltrate is predominantly mononuclear, mainly lymphocytic [3]. This was also the case with our patient, testifying to the progression of his disease.

HBV infection was the cause of polyarteritis nodosa in 36 to 50% of cases [5]. This prevalence is clearly declining today due to vaccination. HCV infection is more often complicated by cryoglobulinemic-type vasculitis. The association of PAN and HCV infection is poorly reported, in isolated cases. Debate Zoguéreh D et al [6] reported a case of PAN in a young drug addict who carried HBV and HCV. Bertchansky et al [7] reported a case of PAN associated with hepatitis C in a young 40-year-old patient also addicted to drugs. The prevalence of hepatitis C virus markers during ANP varies between 5 and 12% depending on

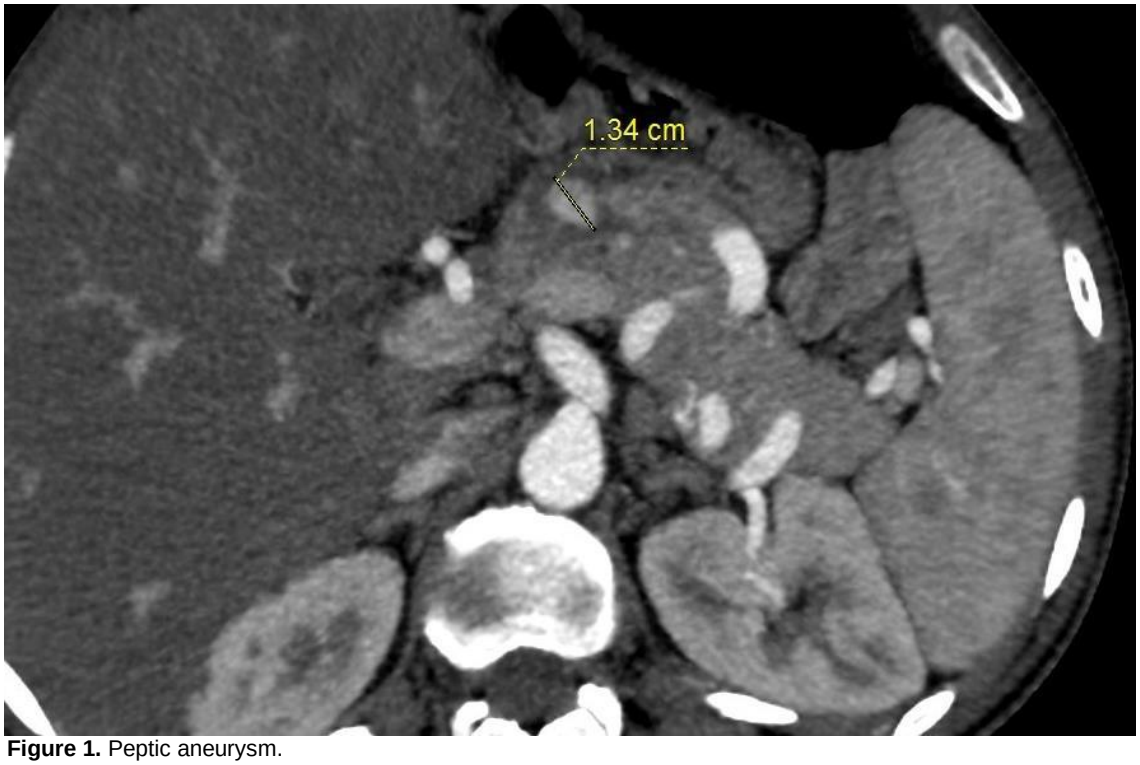

Figure 1. Peptic aneurysm.

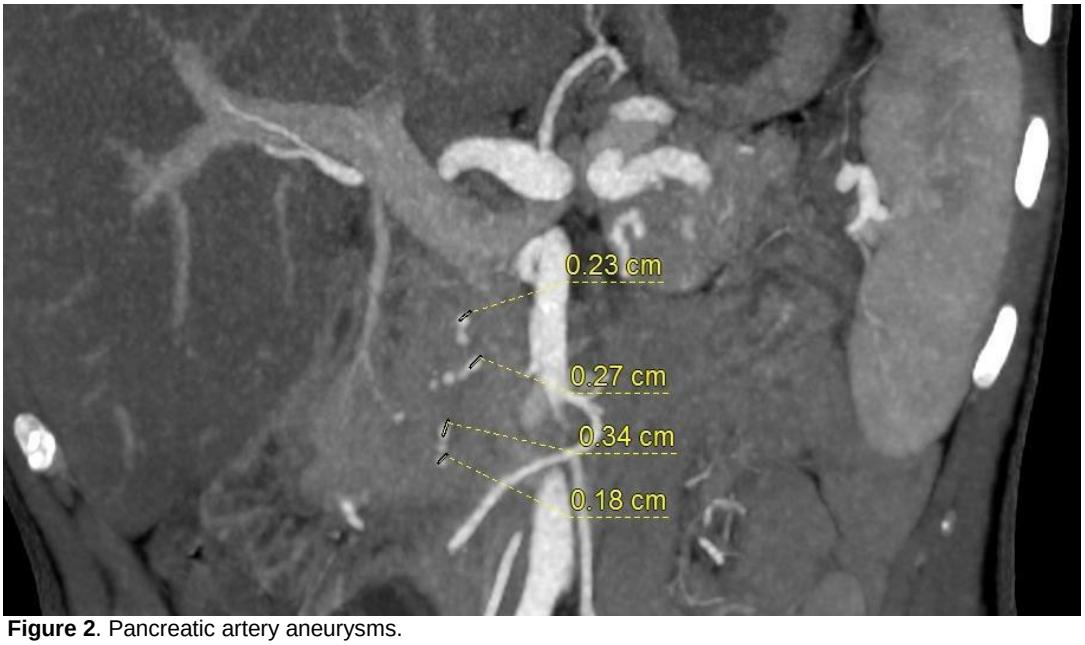

Figure 2. Pancreatic artery aneurysms.

the study [8]. The mechanisms leading patients infected with HCV to develop PAN-type vasculitis, which has become exceptional in France [9], remain unclear [10]. Our patient presented 6 of the 10 ACR criteria of 1990 for ANP. The presence of 3 of these 10 criteria allows the classification of vasculitis as PAN with a sensitivity of 82.2% and a specificity of 86% [11]. The criteria met by our patient are: weight loss of more than 4kg, diffuse myalgia, peripheral neuropathy, diastolic arterial hypertension, micro-aneurysms and anatomo-pathological appearance.

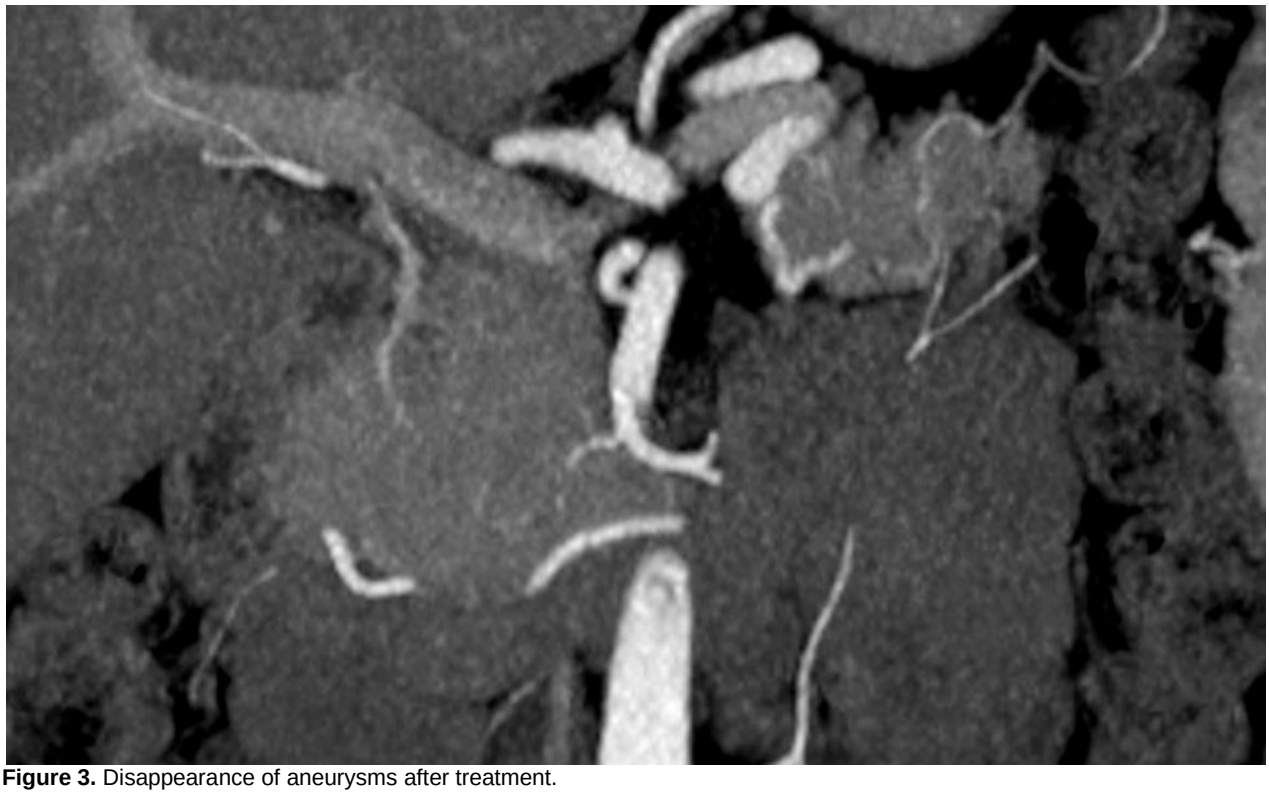

Figure 3. Disappearance of aneurysms after treatment.

Following these ACR criteria, Saadoun et al [12] made a diagnosis of PAN in 31 patients (19.3%) among a cohort of 161 patients with HCV-associated vasculitis. Type III cryoglobulinemia was present in our patient. Mixed cryoglobulinemia represents the prototype of autoimmune diseases linked to HCV [13].
Therapeutically, cyclophosphamide, in combination with corticosteroid therapy, was the classic treatment for ANP [14]. When PAN is associated with HCV infection, antiviral therapy should be combined. Under this combination, the evolution was very satisfactory in our patient with remission of PAN and disappearance of HCV. It also benefited from plasma exchanges which led to a good improvement in his peripheral neuropathy. Once the remission is obtained, relapses are rare during PAN [15].

CONCLUSION

PAN, described by Küssmaul and Maier in 1866, is necrotizing vasculitis that can result from infection with HBV. Its association with HCV is poorly described, but despite its infrequency, testing for HCV infection must be part of the work-up for PAN.

Conflict of interest: None

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