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Case Study
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Lupus Pancreatitis in City Rheumatological Consultation in Bamako (Mali)

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Abstract

Lupus pancreatitis is a rare but potentially severe entity. It is a visceral complication of multifactorial and poorly elucidated pathogenesis. The diagnosis combines two of the three criteria: Typical pain, the elevation of pancreatic enzymes above three time's normal, and imaging. Improved prognosis depends on early diagnosis and efficient treatment. We describe the diagnostic approach and clinical features of a 19-year-old Melanoderma patient.

Keywords: Pancreatitis; Systemic Lupus; Mali

Introduction

Described for the first time in 1939 by Reifeinstein et al. [1], pancreatitis is a rare visceral manifestation during Systemic Lupus Erythematosus (SLE). Its incidence varies from 0.4 to 1.1 cases per 1000 lupus per year [2]. Early diagnosis is pledge of an efficient therapy (corticosteroids and immunosuppressants) to ensure a good prognosis. We report our first observation in a *Melanoderma* subject suffering from SLE in severe flare [3,4].

Observation

A 19-year-old girl had been followed for 45 days for SLE and chronic endoscopic gastritis. The diagnosis of SLE was based on the EULAR/ACR 2019 classification criteria (presence of antinuclear antibodies, malar rash, alopecia, synovitis, fever, leuco-neutropenia). The therapy included prednisone (10 mg/day) and azathioprine (100 mg/day). She is hospitalized in emergency for transfixing epigastric pain, abdominal pain, diarrhea, incoercible vomiting and fever. The physical examination noted patient lying in trunk's anteflexion, feverish at 40°C, epigastric defense and distended abdomen, with much rumbling. SLE activity was very high with a SLEDAI score of 24. Biological assessment revealed inflammatory syndrome (CRP at 150 mg/L, ESR at 110 mm), amylasemia at 392 IU/L and lipasemia at 853 IU/L. Liver tests and stool examinations were normal. The chest-abdominal-pelvic CT-scan was normal. The diagnosis adopted is lupus pancreatitis after having eliminated other causes (biliary lithiasis, toxic, traumatic, drug and neoplastic). She received a bolus of methylprednisone, parenteral analgesics, isocoagulation and rehydration. She underwent a strict 48 h diet. The evolution was favourable to 5th day hospitalization with apyrexia and pain amendment. The relay by oral corticosteroids and hydroxychloroquine was instituted.

Discussion

The occurrence of pancreatitis can complicate the evolution of connectivitis, vasculitis and granulomatosis [1]. Pancreatic involvement during SLE is rare [5,6]. Its incidence varies from 0.4 to 1.1 cases per 1000 lupus per year [2,5]. It can be concomitant with other lupus disorders in 50% of cases [5] inaugural and revealing in 11% of cases [5], or a potentially serious complication [6]. It was subsequent in our patient, which is a particularity. The pathogenesis of this pancreatitis is not well understood [1]. It is multifactorial; difficult to separate what amounts to vasculitis, thrombosis in the context of anti-phospholipid syndrome, or iatrogenic or concomitant complications [7]. The diagnosis is based on the association of two of the following three criteria:

- Typical pain
- Increased pancreatic enzymes above three times normal

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• Computed tomography (CT) imaging, renography (MRI) or ultrasound [7]. Pancreatic pain is relieved by anteflexion of the trunk (pancreatic position) and aspirin [8]. In our patient, epigastric pain incorrectly labeled as gastritis by digestive endoscopy could lead to mistake. The classic aspirin therapy test was not done for fear of a hypothetical gastric perforation. However, any abdominal pain syndrome and/or vomiting in a lupus context suggest lupus pancreatitis [2]. The elevation of lipasemia is of a better diagnostic specificity because lipase is exclusively pancreatic [9]. The elevation of protein C Reactive has an interest in prognosis but she suggested looking for infectious etiology in the patient [9]. CT-scan has proved to be the reference examination in the diagnosis but the pancreas can be normal in 14% to 29% of cases as in our patient [9]. The drug toxicity in this case of azathioprine and prednisone can be invoked initially acute pancreatitis [1,9]. However, the chronology of evident clinical signs in our patient minimizes iatrogenia. Many observations in the literature raise the difficulty of specifying the exact etiology of lupus pancreatitis, even autopsy studies are often non-contributory [10]. Most authors proceed by excluding other possible etiological factors and improving symptomatology with anti-inflammatory treatment to indirectly retain, responsibility for SLE [7]. Efficient therapeutic management depends on early diagnosis for a good prognosis [6]. Methylprednisone bolus having improved the patient, a relay with oral corticosteroids and substitution of azathioprine with synthetic antimalarial (hydroxychloroquine) was decided.

Conclusion

Most lupus pancreatitis have been described in leukoderma subjects. However, our first observation in melanoderma does not suggest any singularity. In all cases, the best prognosis depends on early diagnosis and efficient management.

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