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Severe Epistaxis, Atypical Presentation of Lupus Hepatitis

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Abstract

Lupus hepatitis is a rare manifestation of systemic lupus erythematosus. When present, it manifests as frustrating clinical signs and moderate disturbance of liver function. We describe a case of lupus hepatitis revealed by massive epistaxis secondary to hepatocellular failure in a 35-year-old female patient whose diagnosis of lupus was made secondarily. Management consisted of azathioprine and corticosteroids, with good resolution of clinical and laboratory signs.

Keywords

Lupus Hepatitis, Systemic Lupus, Azathioprine, Corticoids, Epistaxis

1. Introduction

Systemic lupus erythematosus (SLE) is a chronic inflammatory rheumatic disease, a connective tissue disorder that progresses in relapses and remissions, with the production of autoantibodies and immune complexes affecting all tissues. This non-organ-specific autoimmune disease can present in several forms due to the diversity of its symptoms and clinical signs, sometimes delaying diagnosis [1]. SLE can affect the liver, but hepatic involvement is rare, and its manifestations are highly unspecific, generally limited to disturbed liver function and rarely hepato-cellular failure [2]. Coagulopathies with the haemorrhagic syndrome can be a manifestation of hepatocellular failure, while connectivities tissue diseases are rarely among the leading etiologies considered. We report a case of lupus hepatitis, which was revealed by profuse epistaxis.

2. Case Presentation

This is the case of a 35-year-old female patient with 3 parities in the post-partum period (35th day post-partum), with no personal or family history of inflammatory rheumatism. She is not an alcoholic or a smoker, is not hypertensive, and has no known history of miscarriage or liver disease. She presented with a sudden onset of profuse anterior epistaxis and an unstable hemodynamic state. After emergency measures (peripheral venous access, nose-blowing, bidigital compression, anterior tamponade with haemostatic packing). Emergency laboratory tests showed anaemia at 6 g/dl, severe thrombocytopenia at 58,000/mm^3, with a normal leucocyte count. Transaminases (ALT, AST were 6 times the normal value), with a collapsed PT of 39%. The patient was transfused with whole blood and fresh frozen plasma. In the etiological search for her epistaxis, questioning revealed a long-standing history of polyarthralgia associated with oral ulcerations and photosensitivity; she also described abdominal bloating with no urinary or pulmonary complaints. The investigation did not reveal any exposure to toxic or ionizing radiation, nor any use of anticoagulants or hepatotoxic drugs. Clinical examination revealed a deteriorated general condition, clinical anemia, noncholestatic jaundice, petechiae, discoid lupus on the auricle and scalp associated with nonscarring alopecia. Transudative ascites were also present, with a protein level of 22 g/l. Biological tests revealed anemia at 10 g/dl, platelets at 160,000/mm^3, and leukopenia at 4200 white blood cells/mm^3 (after transfusion). Viral serologies were negative, as were anti-Hbc and antiHbe, and alfafetoprotein was negative. Lipidogram was undisturbed, sedimentation rate elevated to 75 mm/h, CRP 8 mg/dl, serum protein electrophoresis showing polyclonal hypergammaglobulinemia and hypoalbuminemia (30 g/dl), proteinuria 0.6 g/l with no cylindruria and no hematuria. Immunological tests were negative for rheumatoid factor, anti-CCP and ANCA. FAN 1600 and speckled type, anti native DNA negative, anti-U1RNP 124, anti-SM 64, anti-Scl negative. Antibody anti smooth muscle, anti KLM1 and anti mitochondria were negative. Abdominal ultrasonography revealed non-specific homogeneous hepatomegaly with a liver span of 18 cm, in addition to ascites. Cardiac ultrasonography, renal ultrasonography and chest radiography were normal. A liver biopsy confirmed non-specific acute hepatitis (acute hepatitis of the peribiliary zones without signs of portal involvement). The patient had been treated with azathioprine 50 mg daily, a bolus of methylprednisolone 120 mg daily for three days (a slightly lower dose than the total dose in case of lupus flare), followed by a decrease to 60 mg daily and then 40 mg combined with hydroxychloroquine (200 mg twice daily after reassuring ophthalmological examination).

3. Discussion

The coexistence of autoimmune hepatitis with lupus represents around 1% of cases in the literature [3]. The diagnosis of lupus hepatitis is often difficult to make; it is a diagnosis of exclusion since toxic, viral, drug-induced and even autoimmune hepatitis must be ruled out. Manifestations are often minimal, consisting of hepa-

tomegaly, jaundice, liver enzyme disturbance and sometimes cholestasis [4]. A Moroccan series showed a 2.92% prevalence of lupus hepatitis in 616 cases of lupus, and less than 1% of these subjects presented with hepatocellular insufficiency, which was asymptomatic [2]. Occasionally, the presence of liver damage is also a poor prognostic factor [5]. In our case, it was a severe coagulation disturbance associated with a drop in prothrombin levels, leading to a bleeding syndrome.

In the literature review we conducted, we did not find any case of lupus hepatitis revealed by massive epistaxis, as in our patient. We did find one case of death due to hepatic encephalopathy, but without hemorrhagic syndrome. Most treatments consist of azathioprine and corticosteroids. In refractory forms, bellimumab has been used, with spectacular regression of the disease in some cases [6]. In our patient, the evolution was favorable after initiation of azathioprine (50 mg per day) coupled with hydroxychloroquine (400 mg per day) and consequent corticosteroid therapy. After 15 days (two weeks), transaminases improved significantly, reaching twice the normal values. The prothrombin rate rose to 73%, and albumin normalized. After six weeks, liver function was fully restored.

4. Conclusion

Lupus hepatitis is rare and, when present, is usually only mildly symptomatic. Rarely does it lead to hepatocellular failure. However, when investigating hepatocellular insufficiency, this rare etiology should be sought out. Azathioprine and corticosteroids are effective in its management.

Consent

Informed consent was obtained from the patient for publication of this case in a scientific journal.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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