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Systemic Lupus Erythematosus Complicated by Pericarditis: A Case Report from the Guindo Clinic in Bamako, Mali

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Abstract

Juvenile systemic lupus erythematosus is a rare entity, affecting children under 16 years of age. Girls are more often affected than boys and the female predominance increases significantly with age. The initial manifestations are highly variable with an insidious and progressive onset. Non-specific symptoms include fever, anorexia, weight loss and asthenia. Pericarditis is the most common cardiac manifestation in systemic lupus erythematosus (SLE), occurring in 10% to 40% of cases. The biological elements of the diagnosis and follow-up of pediatric SLE are identical to those of adults and are based on regular measurement of complement, native anti-DNA antibodies, and inflammatory findings. Treatment is essentially based on corticosteroid therapy.

Keywords

Lupus, Child, Pericarditis, Clinic Guindo, Bamako Mali

1. Introduction

Pediatric-onset systemic lupus erythematosus (SLE) is a rare entity, with only 10% - 15% of cases diagnosed before the age of 16 years [1]. It is characterized by

its greater severity compared to adult-onset SLE due to the greater frequency of severe renal, hematological and neurological involvement [1].

The diagnosis of SLE in the pediatric age is not always easy and the management with heavy treatment in a growing being poses some specific problems [2].

In general, the onset of the disease is during adolescence and very rarely before the age of five. Girls are more often affected than boys and the female predominance increases significantly with age [2].

2. Observation

We report a case of lupus diagnosed in a 15-year-old girl.

This is an adolescent girl with a family history of lupus and diabetes of her father who is under treatment. She had been symptomatic for 2 to 3 days: marked by liquid diarrhea without mucus or blood in the stool, associated with chest and joint pain in the wrists, ankles and knees. She had headache and fever, which led to the use of an antipyretic analgesic before her admission to our center

On physical examination: The child was asthenic with pain rated at 8/10 according to VAS (Visual Analog Scale), temperature at 39˚C, weight at 55 kg, pulse at 120 bt/mn and blood pressure at 100/60 mm hg with conjunctival pallor. The abdomen was soft with no palpable mass. Heart sounds were regular with tachycardia at 120 beats per minute, no pericardial rub, no signs of heart failure, pulmonary examination was Normal. The neurological examination found no involvement of the cranial pairs and reactive pupils. The painful joints were not warm and not fluctuating. Erythematous angina, bilateral axillary adenopathies were noted. They were no visible skin lesions.

Paraclinical examinations:

Biological evaluation: blood ionogram was normal; creatinine level was normal at 54.6 umol/L. Normochromic microcytic anemia was noted with hemoglobin at 9.4/dl platelets at 120,000. CRP was 83 mg/L. Accelerated sedimentation rate at 1st and 2nd hour, blood glucose 5 mmol/L and ASLO was negative. TSH normal, martial status normal, cortisol level normal, hemoglobin electrophoresis normal, LDH 1584 IU/L, VRS negative, autoimmune assessment showed anti-native DNA antibodies greater than 300 IU/ml, and ANA-Screen 16.80 EU which are in favor of lupus.

Thoracic-abdominal-pelvic CT scans which showed mediastinal adenopathies.

Normal transthoracic echocardiography, except for a non-compressive pericardial circumferential effusion of 6 mm.

In total: the examination revealed: pallor, asthenia, angina, polyarthralgia, adenopathies, a non-compressive circumferential pericardial of 6 mm and native DNAs higher than 300 UI/L.

Treatment and evolution:

The evolution was favorable under antibiotic (amoxi-clavulanic acid) for pharyngitis, antipyretic (paracetamol), steroidal anti-inflammatory (Prednisone 60 mg/m^2 per day) after deworming, gastric dressing and rest. The patient was

treated with high-dose prednisone (60 mg/m^2/d) for 2 months, followed by a tapering dose and maintenance treatment.

3. Discussion

Juvenile systemic lupus erythematosus is a rare entity, affecting children under 16 years of age, estimated at 10% - 17% of cases, and whose diagnosis is not always easy and whose course is generally more severe than that of adults according to most of the few published pediatric series [2] [3]. The greater severity of pediatric SLE compared to adult SLE may be due to a greater involvement of genetic factors. A recent study of the number of susceptibility alleles for the occurrence of SLE, identified by an approach based on the detection of polymorphic markers such as single nucleotide polymorphisms (SNPs), found a higher number of SLE risk alleles before the age of 18 years in African-American patients [4]. The prevalence rate of pediatric SLE is significantly lower than that of adults. The annual incidence rate of SLE in children < 16 years of age is less than 1 per 100,000 people in studies from Europe and North America. In Taiwan, the prevalence of childhood SLE has been estimated to be 6.3 per 100,000 [1].

Girls are more often affected than boys and the female predominance increases significantly with age [2]. In a Swiss study, in a group of 59 patients aged 5 to 16 years, the ratio of girls to boys was 6 to 4 before the age of 9, 24 to 7 between the ages of 10 and 14, and 18 out of 0 between 15 and 16 years of age, just as in our case where the sex is female. This increase in the ratio of girls to boys with age could be due to the key role played by hormonal factors in the female preponderance [1].

The initial manifestations are highly variable with an insidious and progressive onset. The non-specific symptoms are fever, anorexia, weight loss and asthenia. They are particularly deceptive in adolescence. Early in the disease, a single organ may be affected, but the systemic form is the usual form of revelation. Arthritis, rash, and renal involvement are the most common involvement in the pediatric form [5]. Cardiac involvement during systemic lupus erythematosus is part of the 11 diagnostic criteria established by the American Rheumatism Association (ARA) in 1982, through pericarditis [6].

Pericarditis is the most frequent cardiac manifestation of systemic lupus erythematosus (SLE), occurring in 10% to 40% of cases. Its clinical expression is no specific, and presents with chest pain typically accentuated in the sitting and lying position, relieved in the sitting and bending forward position, of a respiro-dependent nature with or without dyspnea, and may be accompanied by fever. Auscultation may reveal decreased heart sounds or pericardial rub. The effusion is usually small, and the rarity of tamponade explains the limited data available on the pericardial fluid, which is most often highly inflammatory, with a protide level greater than 50 g/L, and a glucose level is usually low. Pleural effusion is frequently associated. Ultrasound demonstrates pericardial effusion

and/or thickening [7].

The presence of arthralgias in SLE is one of the most common osteoarticular symptoms, present in 88% of cases at the time of diagnosis. These are most often asymmetric, transient and sometimes migratory arthralgias [8] [9]. The joints affected are, in decreasing order, the small joints of the hands, fingers, wrists then the feet, elbows, knees and shoulders. Hip involvement is rare. Prolonged morning discomfort is often noted (in 50% of patients).

These joint manifestations may be brief and transient or progress to chronicity. Arthritis is present initially in 69% of cases and during the course of the disease in 84% of cases [8].

About 50% of patients with lupus have polyadenopathies. They are more associated with exacerbations. These adenopathies are often discrete and localized in the cervical, axilla and inguinal regions [10] [11].

Hematological signs are a major severity factor in SLE. In particular, autoimmune anemia and thrombocytopenia (50%) require prolonged treatment with immunosuppressants. Inflammatory anemia affects up to 70% of children with SLE. Lymphopenia may be related to lupus or to treatments [12].

The biological elements of the diagnosis and follow-up of pediatric SLE are identical to those of adults and are based on regular measurement of complement, native anti-DNA antibodies and elements of the inflammatory balance. Antiphospholipid antibodies are also routinely tested at diagnosis and during thrombotic episodes. A urine dipstick is systematically performed at each consultation and during each attack, or 24-hour proteinuria is performed to look for a renal complication [12].

The management of this condition requires multidisciplinary collaboration. It is essential to educate the child and his family [13].

In fact, corticosteroid therapy, which is usually the basis of treatment, is used in high doses. This is likely to induce several damages, in particular those concerning bone mineralization during a crucial period for the acquisition of bone mass, as well as other deleterious effects likely to worsen the prognosis, in this case obesity, infections, statural delay, diabetes, cataract and osteoporosis [2] [3] [14]. It is used in high doses (1 - 2 mg/kg/day), with venous corticosteroid therapy depending on the indication. The duration of the attack is 6 weeks to 3 months, followed by a slow progressive degression after the relapse has been stopped [13].

4. Conclusion

Pediatric onset systemic lupus erythematosus (SLE) has a clinical presentation similar to that of adults. However, the diagnosis of this pediatric entity is not always easy (no specific sign), the course is generally more severe, and the management often requires long-term corticosteroid therapy and education of the child and his family.

Conflicts of Interest

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

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