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Sydenham's Chorea in an 11-Year-Old Girl at the Bouaké University Hospital (Côte d'Ivoire)

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Abstract

Sydenham's chorea is a neurological complication of acute rheumatic fever (ARF), frequently encountered in developing countries. It presents with involuntary movements, psychiatric disturbances, and muscular hypotonia, typically occurring in a post-streptococcal infection context. The diagnosis is primarily clinical. We report the case of an 11-year-old adolescent admitted for abnormal movements, in whom clinical and paraclinical evaluation led to the diagnosis of Sydenham's chorea. The outcome was favorable under treatment combining antibiotics, non-steroidal anti-inflammatory drugs, and valproic acid. The aim of this case report is to highlight the epidemiological, diagnostic, and therapeutic features of this condition through a clinical case and a review of the literature.

Keywords

Sydenham's Chorea, Involuntary Movements, Acute Rheumatic Fever, ASLO, Côte d'Ivoire

1. Introduction

Sydenham's chorea, also known as post-streptococcal chorea or Saint Vitus' dance, is the most common form of acquired chorea in children. It was first described by Thomas Sydenham in 1686 [1]. It is a major manifestation of acute rheumatic fever (ARF), occurring in up to 40% of affected patients. The clinical presentation is characterized by brief, rapid, involuntary movements of variable amplitude, of-

ten associated with psychiatric disturbances and muscular hypotonia. The pathogenesis is believed to be autoimmune in nature [2]. Its incidence has significantly declined in developed countries in parallel with that of ARF, following the widespread use of antibiotics to treat streptococcal pharyngitis [3]. In contrast, in developing countries, ARF and Sydenham's chorea remain significant public health concerns [4] [5].

We report a case of Sydenham's chorea in an 11-year-old girl admitted to the Pediatrics Department of the Bouaké University Hospital.

The objective of this case report is to highlight the epidemiological, diagnostic, and therapeutic features of this condition based on a review of the literature.

2. Case report

K.S., an 11-year-old girl living with her father in a rural area, was brought to the pediatric emergency department for abnormal movements. The history revealed a progressive onset of symptoms, including involuntary abnormal movements, incoherent speech, and muscle weakness in the lower limbs. Her past medical history included several episodes of untreated sore throat, the last occurring about three months before chorea onset, and an incomplete vaccination record. There was no recent use of neuroleptics, antiemetics, or antiepileptic medications.

On physical examination at admission, she weighed 25 kg, appeared generally well, had a body temperature of 37˚C, and her conjunctivae were normally colored. Neurological assessment showed a Glasgow Coma Score of 15/15, supple neck, and no signs of Kernig or Brudzinski. She exhibited broad, rotatory, and chaotic involuntary movements, predominantly affecting the upper and lower limbs. Muscle strength in the right lower limb was graded at 4/5. Osteoarticular examination revealed tenderness in the knees and elbows. The throat was non-inflammatory, and the tonsils were not enlarged. Examination of the respiratory, cardiovascular, and digestive systems was unremarkable.

Paraclinical investigations showed a white blood cell count of 4000/mm^3, hemoglobin level of 12.3 g/dL, and platelet count of 254,000/mm^3. Erythrocyte sedimentation rate (ESR) was elevated at 70 mm in the first hour and 100 mm in the second hour. The antistreptolysin O (ASLO) titer was high at 3200 mg/L. C-reactive protein (CRP) was 2.8 mg/L. Urea was 0.32 g/L, creatinine 9.3 mg/L, calcium 118 mg/L, potassium 4.44 mEq/L, and sodium 148 mEq/L. Electroencephalogram, electrocardiogram, cardiac ultrasound, and hemoglobin electrophoresis were all normal.

Based on these findings, the diagnosis of Sydenham's chorea was made. The patient was hospitalized and treated with intravenous antibiotics (amoxicillin-clavulanic acid 850 mg three times daily), a non-steroidal anti-inflammatory drug (ketoprofen 25 mg diluted in 100 cc of 5% dextrose administered slowly IV), and an antiepileptic (valproic acid 200 mg three times daily orally).

Five days later, clinical improvement was observed with resolution of the abnormal movements and normalization of inflammatory markers. Discharge was au-

thorized after 10 days of hospitalization. Prophylactic antibiotic therapy with benzathine penicillin (1.2 million IU every three weeks) was initiated. NSAIDs were continued for 4 weeks, and valproic acid was maintained for 6 months, with gradual withdrawal in the absence of recurrence. Monthly outpatient follow-up for the first three months showed complete remission without recurrence. At six months, the patient remained asymptomatic with no evidence of cardiac involvement on clinical examination and echocardiography.

The outcome was favorable, marked by the absence of symptom recurrence and the resumption of school activities after two months.

3. Discussion

The objective of this case report was to highlight the epidemiological, diagnostic, and therapeutic characteristics of this condition through a review of the literature. Sydenham's chorea is the most frequent infectious chorea in children [6]. Improvements in living standards, the advent of antibiotics, and prophylactic measures have made this condition rare in developed countries [5] [7]. However, its prevalence remains high in developing countries, likely due to poor hygiene conditions and low socioeconomic status. In the case we presented, the adolescent lived in a rural area where hygiene and sociodemographic conditions are often unfavorable.

Sydenham's chorea typically occurs in late childhood or around puberty, most commonly between the ages of 5 and 15 years, with a marked female predominance [8]-[11]. This case of an 11-year-old girl fits that typical profile. The onset is usually gradual and insidious [3], often occurring 1 to 6 months after a streptococcal pharyngitis episode [2]. Our patient's onset occurred about three months after the last sore throat, reinforcing the post-streptococcal link. The pathophysiology involves an autoimmune mechanism triggered by antigenic mimicry between group A streptococcus and the basal ganglia. This cross-reaction disrupts the balance of excitatory dopaminergic, intrastriatal cholinergic, and inhibitory GABAergic pathways, leading to the clinical manifestations [2].

Chorea is often localized, affecting one side of the body (hemichorea) or a limb segment (monochorea), in 10% - 20% of cases [7] [11]. In this case, the chorea affected all four limbs, a less common presentation that can lead to diagnostic confusion. The motor agitation in Sydenham's chorea is almost always accompanied by prominent psychiatric disturbances [1] [11], such as emotional lability, irritability, obsessive-compulsive symptoms, anxiety, sleep and attention disorders, and general nervousness. Some authors even consider these symptoms to be constant [1] [7].

Muscle strength is often normal but can sometimes be reduced, evidenced by the inability to keep the eyes closed or to maintain tongue protrusion [1] [3] [7] [11] [12]. In other cases, weakness may affect other body parts, particularly the lower limbs, potentially causing functional impairment. In our observation, segmental and global muscle weakness was noted on the right side, with a muscle strength score of 4/5.

Diagnosis is primarily clinical, supported by paraclinical findings such as elevated antistreptolysin O (ASLO) titers or other evidence of streptococcal infection, increased ESR, and C-reactive protein. The blood count may be normal or show mild leukocytosis. An ECG may reveal PR interval prolongation. Brain MRI is often normal but may show nonspecific abnormalities of the basal ganglia. EEG may sometimes show diffuse, nonspecific slowing. Echocardiography is performed to detect valvular involvement [13]. However, in our context, technical limitations can hinder the realization of all these exams.

In our case, ESR was elevated, CRP was normal, ASLO was high, and ECG, echocardiography, and EEG were normal. The presence of large, rotatory, chaotic involuntary movements in an adolescent could raise suspicion for drug-induced chorea, lupus chorea, Huntington's disease, Wilson's disease, or motor epilepsy. However, the absence of recent exposure to pro-choreic drugs, the young age of the patient, absence of cognitive decline or pyramidal signs, the post-streptococcal context, biological signs of inflammation, and normal EEG made these diagnoses less likely [14]. Brain MRI was not performed, and serum copper/ceruloplasmin levels, as well as autoimmune antibody screening, were difficult to access in our setting. The exclusion of these conditions was therefore based on the clinical presentation, patient history, and absence of suggestive findings on routine laboratory tests and neurological examination.

Treatment of Sydenham's chorea includes both symptomatic and specific components. Various symptomatic treatments are available. Neuroleptics such as haloperidol, pimozide, and chlorpromazine have proven efficacy, with haloperidol being the most effective. However, side effects are more frequent with haloperidol [8]. Antiepileptic drugs like carbamazepine and sodium valproate are also effective and generally better tolerated than neuroleptics. In this case, the patient received sodium valproate with good clinical improvement [8] [15].

Given the autoimmune nature of Sydenham's chorea, anti-inflammatory agents like salicylates and corticosteroids can effectively reduce abnormal movements [1] [16]-[18]. As for specific treatment, antibiotic therapy is essential. It is recommended in all cases, even when a definitive rheumatic etiology is not confirmed, to reduce the pathogenic potential of group A beta-hemolytic streptococcus in the pharynx and to prevent the spread of virulent strains [1] [3] [4]. Penicillin remains the antibiotic of choice for treating streptococcal infections and can be administered orally (Penicillin V) or intramuscularly (Penicillin G); in case of penicillin allergy, erythromycin may be used. Long-term secondary prophylaxis consists of intramuscular injections of benzathine penicillin to prevent recurrence of rheumatic fever, in line with WHO 2024 guidelines, with emphasis on adherence and prompt treatment of new streptococcal infections [18] [19]. Although amoxicillin-clavulanic acid is not the standard prophylactic treatment for Sydenham's chorea or acute rheumatic fever, its use in this case was aimed at reducing any residual streptococcal colonization while awaiting long-term benzathine penicillin prophylaxis.

Preventive measures include proper management of ENT and skin infections, keeping vaccinations up to date, and early diagnosis and treatment of chorea. As in this case and those reported in the literature, the outcome is usually favorable with treatment. Relapses or persistent chorea is rare and is often due to noncompliance or the presence of an underlying cause [8].

4. Conclusion

Sydenham's chorea, although less frequent than in the past, is still observed in our country. Its clinical diagnosis is based on the identification of involuntary abnormal movements associated with psychiatric disturbances and muscular hypotonia. There is no standardized therapeutic protocol. The course of the disease may be marked by relapses, cardiac involvement, and residual psychiatric disorders, which can cause disruptions in family and social life. Only primary prophylaxis through systematic treatment of streptococcal pharyngitis can reduce its incidence and related complications.

Authors' Contributions

All authors intellectually participated in the preparation and revision of the manuscript prior to its submission.

Consent

Consent for publication was obtained from the patient's parent.

Conflicts of Interest

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

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