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Hypokalemic Tetraparesis Revealing Sjögren's Syndrome: A Case Report

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Abstract:
Background:
Sjögren's syndrome (SS) is a systemic autoimmune disease that may be associated with various extra-glandular manifestations, particularly renal and neurological involvement. Renal tubulointerstitial involvement may lead to distal renal tubular acidosis, resulting in severe hypokalemia and potentially acute neuromuscular complications.
Methods:
We report the case of a young female patient presenting with an unusual neurological manifestation revealing Sjögren's syndrome. Clinical, biological, immunological, and histopathological findings were analyzed.
Results:
The patient was admitted for rapidly progressive acute flaccid tetraparesis in the setting of hypokalemic paralysis associated with renal involvement and sicca symptoms. The diagnosis of Sjögren's syndrome was established based on a combination of clinical, immunological, and histopathological findings. Clinical outcome was favorable following correction of hydro-electrolytic disturbances.
Conclusion:
This case highlights that Sjögren's syndrome should be considered in the diagnostic workup of unexplained hypokalemic paralysis, even in the absence of initially obvious glandular manifestations.

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I. Introduction:

Sjögren's syndrome (SS) is a systemic autoimmune disease characterized by lymphocytic infiltration of exocrine glands, mainly resulting in sicca symptoms (1). Beyond glandular involvement, several systemic manifestations may occur, particularly renal involvement predominantly represented by tubulointerstitial nephritis and distal renal tubular acidosis (2). The latter may lead to severe hypokalemia which, in rare cases, can reveal the disease through acute hypokalemic tetraparesis (2). We report the case of a patient in whom hypokalemic tetraparesis was the presenting manifestation of Sjögren's syndrome.

II. Methods:

This was a retrospective descriptive study reporting a clinical case collected in the Neurology Department. Clinical, biological, immunological, and radiological data were retrospectively obtained from the patient's medical records and analyzed to assess the diagnostic, therapeutic, and outcome characteristics of the case. A targeted literature review was also performed to discuss the pathophysiological, diagnostic, and therapeutic aspects of this unusual clinical presentation.

III. Observation:

A 33-year-old woman was admitted for rapidly progressive weakness of all four limbs evolving in an afebrile setting. Neurological examination revealed flaccid tetraparesis predominantly affecting the lower limbs, graded as 2/5 in the upper limbs and 1/5 in the lower limbs according to the Medical Research Council (MRC) scale, with diffuse abolition of deep tendon reflexes, without cranial nerve involvement or objective sensory deficits.

General examination revealed multiple erythema nodosum lesions at different stages of evolution (Figure 1), associated with oral and ocular dryness suggestive of sicca syndrome. Laboratory investigations demonstrated severe hypokalemia at 2.6 mEq/L associated with renal involvement, characterized by impaired renal function with an estimated creatinine clearance of 60 mL/min/1.73 m² and a 24-hour proteinuria of 1.08 g/24 h. Cerebrospinal fluid analysis was unremarkable.

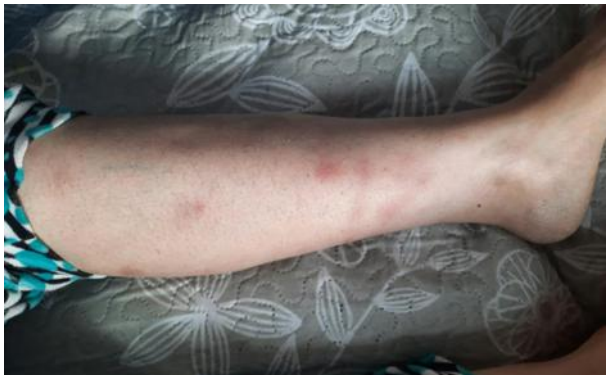

Figure 1: Multiple erythema nodosum lesions of different evolutionary stages involving the leg.

Correction of hypokalemia resulted in a rapid and complete recovery of motor deficits. Given the association of hypokalemic tetraparesis, renal involvement, sicca syndrome, and erythema nodosum, Sjögren's syndrome was suspected and subsequently confirmed by positive anti-SSA/SSB antibodies and a minor salivary gland biopsy demonstrating grade IV chronic lymphocytic sialadenitis according to the Chisholm and Mason classification.

IV. Discussion:

Sjögren's syndrome (SS) is a systemic autoimmune disease characterized by lymphocytic infiltration of the exocrine glands leading to sicca symptoms, but it may also be associated with numerous extra-glandular manifestations (1,2). It predominantly affects women and can involve several organs, particularly the kidneys and nervous system.

Renal involvement in SS is mainly represented by tubulointerstitial lesions, especially distal renal tubular acidosis, whereas glomerular involvement is less common (2,3). This condition may lead to urinary potassium wasting resulting in sometimes severe hypokalemia. Clinical manifestations secondary to hypokalemia are variable, ranging from muscle weakness to acute flaccid paralysis that may exceptionally become life-threatening (4,5).

Neurological manifestations of SS are most frequently peripheral, predominantly consisting of axonal sensorimotor polyneuropathies. However, presentation with hypokalemic tetraparesis remains rare, although several cases have been reported in the literature (4,6). In some cases, muscular symptoms may precede sicca manifestations, which can delay diagnosis (6). Acute management relies on prompt correction of hydro-electrolytic disturbances, particularly potassium supplementation, generally resulting in favorable clinical recovery (2).

V. Conclusion:

The presentation of Sjögren's syndrome (SS) as hypokalemic tetraparesis is a rare but well-documented clinical entity. This diagnosis should be considered in any case of unexplained hypokalemic paralysis, particularly when findings suggestive of renal tubular involvement are present, even in the absence of initially obvious ocular or oral sicca symptoms. Early recognition allows prompt and appropriate management and may help prevent complications related to the underlying systemic disease.

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