within normal limits. Differential diagnoses of acute inflammatory During the current admission, the patient's vital signs were and had normal potassium levels. There was no family history of muscle weakness, her serum potassium levels were found to be low. Between episodes of severe flaccid muscle paralysis involving both proximal and distal muscle groups, in the last 10 years. During each episode of she complained of sudden onset weakness of all four limbs (without sensory involvement) since the previous day. On further elicitation of her past history, it was revealed that the patient had had four episodes of severe flaccid muscle paralysis involving both proximal and distal muscle groups, in the last 10 years. During each episode of weakness, her serum potassium levels were found to be low. Between the paralytic episodes, the patient functioned well without any therapy and had normal potassium levels. There was no family history of muscle diseases.

During the current admission, the patient's vital signs were within normal limits. Differential diagnoses of acute inflammatory demyelinating polyradiculoneuropathy and HPP were under consideration. The patient's serum potassium level was 2.4 mmol/L, which is well below normal. Furthermore, her electrocardiography (ECG) showed prominent U-waves, which is classically seen in cases of hypokalemia. Her serum sodium level was normal (141 mmol/L). Further, an arterial blood gas analysis revealed a blood pH of 7.1, signifying acidosis. This decrease in pH, along with an elevated transtubular potassium gradient ushered in the diagnosis of distal RTA, with HPP as its complication.

On further evaluation, it was discovered that the patient had a mild sicca component associated, in the form of mild keratoconjunctivitis sicca and xerostomia. The data presented in this report support the view that the paralytic episodes were due to hypokalemia secondary to RTA associated with Sjogren's syndrome. A final diagnosis of complicated Sjogren's syndrome was made. The patient was started on low dose oral corticosteroids and potassium supplements, following which she has improved symptomatically.

DISCUSSION

In our patient, the presentation was classical of HPP, as evidenced by the serum levels, ECG and response to potassium supplementation. The etiology of HPP was the crux in the case. Hypokalemia paralysis is a well-known, albeit rare, complication of severe distal RTA from any cause [6,7]. Distal RTA may be primary or more commonly secondary to paraproteinemia, medullary sponge kidney, nephrocalcinosis, obstructive uropathy, and autoimmune disease. Hypokalemia due to distal RTA is usually a late manifestation [8].

Sjogren's syndrome manifesting for the first time as HPP caused by distal RTA is a very rare presentation but has been reported in <2% cases of Sjogren's syndrome as a presenting manifestation in the available literature. Management includes potassium supplements, corticosteroids, and cytotoxic agents, if necessary [3,9,10].

CONCLUSION

To conclude, we would like to emphasize on the fact that hypokalemia should not just be corrected but also investigated for the cause. Sjogren's syndrome presenting as HPP is very rare, but it should be borne in mind when investigating a case of HPP secondary to distal RTA.

**ABSTRACT**

The most common presentations of Sjogren's syndrome include dryness of eyes, oral cavity, and features of systemic scleroderma. When a patient walks in with such classical features, it becomes easy for the clinician to diagnose and treat the patient. However, Sjogren's syndrome may present atypically as experienced in the present case. Here, the authors present a case of Sjogren's syndrome, which presented as hypokalemic periodic palsy, secondary to distal renal tubular acidosis.

**Keywords**: Scleroderma, Potassium, Sicca syndrome, Renal tubular acidosis.
REFERENCES