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# Adrenal Insufficiency Presenting with Hyperkalemic Quadriparesis

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**ABSTRACT**

*Secondary hyperkalemic muscle weakness and flaccid paralysis are rare presentations of Primary Adrenal Insufficiency (PAI). The clinical picture is that of ascending muscle weakness, similar to Guillain Barre Syndrome, in the presence of hyperkalemia. We report on a patient known to have PAI, taking dexamethasone and fludrocortisone supplements, but who stopped taking the latter. A month later, there was a progressive ascending weakness of his legs, trunk, and arms. On examination, he was shown to have 3/5 muscle weakness in both legs and arms. His initial potassium level was 7.4 meq/l, Electrocardiogram (ECG) revealed peaked T waves. He was successfully treated for hyperkalemia. Within 6 hours, however, he was able to regain full power of his muscles. PAI should be considered as a differential diagnosis of secondary hyperkalemic paralysis. Mineralocorticoid replacement is an essential component of PAI treatment to avoid this potential neurological complication and life-threatening electrolyte disorder.*

**Keywords:** Secondary hyperkalemic, Adrenal insufficiency, Hyperkalemic paralysis

## INTRODUCTION

Severe muscle weakness and flaccid paralysis are recognized consequences of hyperkalemia [1,2]. Typically, patients have underlying renal insufficiency or drug-induced hyperkalemia [3,4]. Adrenal insufficiency presenting with hyperkalemic paralysis has rarely been reported [5,6]. Here, we report a case of quadriparesis, caused by severe hyperkalemia in a known patient with PAI.

## CASE PRESENTATION

A 35-year-old man was diagnosed with PAI 15 years earlier. He was on dexamethasone 1 mg twice daily, as he showed a suboptimal response on hydrocortisone replacement in terms of the persistence of his symptoms and hyperpigmentation. In addition to dexamethasone, he was on fludrocortisone 0.1 mg daily. He ran out of fludrocortisone a month before this presentation. One week beforehand, though, he started to have progressive muscle weakness, and difficulty in walking and climbing stairs, standing from a sitting position and getting out of bed. He later developed weakness of his trunk, followed one day later with difficulty in carrying heavy loads. He also had tingling sensations in his feet. On examination, he had bilateral 3/5 muscle weakness in his legs and arms. His initial electrolytes revealed a serum potassium level of 7.4 mmol/l, sodium 123 mmol/l, chloride 95 mmol/l, urea 12.3 mmol/l, creatinine 80 mmol/l, and calcium 2.4 mmol/l. The ECG (Figure 1) showed tall broad T waves. The patient was initially treated with 1 g of calcium gluconate over 10 minutes, 10 units of regular insulin, with 50 cc of 50% dextrose, and hydrocortisone 100 mg intravenously. He also received salbutamol nebulization. In two hours, his potassium dropped to 6.3 meq/l, in 4 hours, it was 5.8 and, in 12 hours it dropped to 5.3 meq/l. Repeat ECG showed improvement of T waves (Figure 2).

## DISCUSSION

The neurological presentation and ECG abnormalities in our patient were related to severe hyperkalemia, which can cause significant muscle weakness and even flaccid paralysis [1,7]. The muscle weakness is typically ascending in progression as in our patient starting in the legs and progressing to the trunk and arms. Generally, potassium levels are ≥ 7.0 meq/l and could reach near 10 meq/l, reflecting the slow chronic progression of hyperkalemia [1,8].

Secondary hyperkalemic paralysis has been reported about a wide spectrum of disorders and medications, particularly angiotensin-converting enzyme inhibitors and potassium-sparing diuretics resulting in severe hyperkalemia especially when renal insufficiency is present [1-3]. The precise mechanism of muscle weakness is not completely understood; it appears different than channelopathy, which occurs in hyperkalemic periodic paralysis [9]. It has a similar clinical pattern to Guillain-Barre syndrome [2,9]. Also, electrophysiological studies show a comparable situation to GBS. Clinical and electrophysiological recovery is typical with the correction of hyperkalemia [2,7,8].

PAI is a disease with a gradual onset, which may be unnoticed by the patient for many years. Its clinical presentation results from deficiencies of corticosteroids, mineralocorticoids, and adrenal androgens. The impact of mineralocorticoid loss could result in postural dizziness, weakness, and hypotension. These symptoms are related to volume depletion caused by mineralocorticoid deficiency. The usual electrolyte abnormalities include hyponatremia (most common) and hyperkalemia. Hyperkalemia is often described as mild and is found in 50% to 60% of patients [10]. The impaired potassium excretion can also be exacerbated by a reduced glomerular filtration rate.

Hyperkalemic paralysis is reported as the presenting complaint of PAI [5,6]. Clinical presentation is comparable to other causes of hyperkalemic-induced paresis/paralysis. Our patient was known to have adrenal insufficiency, and was on a potent corticosteroid, without mineralocorticoid activity. Yet, he was vulnerable to manifestations of mineralocorticoid deficiency, as he ran out of fludrocortisone.

## CONCLUSION

This case demonstrates the importance of mineralocorticoid replacement in patients with PAI, in addition to corticosteroids. This is important when patients are given replacement corticosteroids, which have little or no mineralocorticoid activity. In these patients, a dose of fludrocortisone up to 0.2 mg daily may be needed to normalize their electrolyte imbalance and suppress their plasma renin activity.

## DECLARATIONS

### Conflicts of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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