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A Case of Trigger-Point Injection-Induced Hypokalemic Paralysis

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Patient: Male, 39
Final Diagnosis: Trigger-point induced hypokalemia
Symptoms: Bilateral lower extremity weakness
Medication: Epinephrine • Bupivacaine • Methylprednisolone
Clinical Procedure: Trigger-point Injection
Specialty: Nephrology and Radiology

Objective: Unknown etiology

Background: Trigger-point injection (TPI) therapy is an effective modality for symptomatic treatment of myofascial pain. Serious adverse effects are rarely observed. In this report, we present the case of a 39-year-old man who experienced severe, transient hypokalemic paralysis in the context of TPI therapy with methylprednisolone, bupivacaine, and epinephrine. He was successfully treated with electrolyte replacement in a closely monitored setting.

Case Report: A 39-year-old man with no past medical history except for chronic left hip pain from a work-related injury received a TPI with methylprednisolone and bupivacaine. The TPI targeted the left iliopsoas tendon and was administered using ultrasound guidance. There were no immediately perceived complications, but within 12 h he presented with severe hypokalemic paralysis with a serum potassium 1.7 mmol/L. Judicious potassium repletion was initiated. Repeated tests after 6 h consistently showed normal potassium levels of 4.5 mmol/L.

Conclusions: Severe hypokalemic paralysis in the context of trigger-point injection is an incredibly rare occurrence and this is the first case report in English literature. A high index of clinical suspicion and a systematic approach are therefore required for prompt diagnosis and management of this obscure iatrogenic entity. Clinicians can enhance patient safety by allowing the primary pathology to guide them.

MeSH Keywords: Hypokalemia • Hypokalemic Periodic Paralysis • Trigger Points

Full-text PDF: http://www.amjcaserep.com/abstract/index/idArt/903139

Background

Trigger points are symptomatic irritable foci in taut bands of a skeletal muscle or its fascia [1]. They are the hallmark physical examination signs of myofascial pain [2,3]. Trigger-point injection (TPI) is an effective modality for symptomatic treatment of myofascial pain syndrome [4–6]. Steroids and anesthetics are thought to inactivate tight muscular bands, thereby relieving nerve irritation and referred pain [7]. The use of ultrasound-guided injection improves efficacy of drug delivery and enhances safety [8,9]. Site injury is common, but serious complications are rare [8,10]. We present a case of TPI therapy-induced hypokalemic paralysis. To the best of our knowledge, this is the first such case report in the English literature [11].

Case Report

A 39-year-old man, with no known past medical history except for chronic left hip pain from a work- related injury, received a TPI with methylprednisolone, bupivacaine, and epinephrine. He had received the same drug cocktail 3 months prior without incident, and the intervention was effective in relieving his hip pain. The TPI targeted the left iliopsoas tendon and was administered using ultrasound guidance. He went to sleep that night without perceived complications. Approximately 12 h after therapy, he awoke with shortness of breath and functional quadriplegia. His mental faculties were intact and he did not experience chest pressure, vomiting, abdominal discomfort, or diarrhea.

He was brought to the Emergency Department with the following vital signs: BP 162/92 mmhg, HR 94 bpm, RR 22 cpm, T 97.9°F (36.6°C), and SaO_2 95% on room air. He appeared calm and conversational. He was in mild respiratory distress with shallow respirations. Results of cardiovascular, pulmonary, and gastrointestinal examinations were unremarkable. A neurologic exam revealed intact cerebral and cognitive function, GCS 15, normal pupillary light reflex and extraocular muscle movements, and no nystagmus. Cranial nerves and cerebellar function test results were also normal. He had severe paresis of the upper extremities, 1/5 bilateral motor strength, and nearly choreiform in movement. Both lower limbs were paraplegic. Sensory perception and deep-tendon reflexes were normal in all extremities.

Electrocardiography (EKG) demonstrated sinus arrhythmia, flat T waves, and small U waves, with a normal QT interval. He was in metabolic acidosis with a base deficit of -7 (pH 7.31, HCO_3 19 mmol/L, PCO_2 38 mmHg). The patient's serum potassium was 1.7 mmol/L, magnesium 2.1 mg/dl, and calcium 9.8 mg/dl. Urine K/Cr ratio was 0.067, creatinine kinase levels were moderately elevated (523 IU/L), and thyroid function was normal (Table 1).

He immediately received judicious potassium repletion with a combined total of 150 milliequivalents of IV and PO KCl administered, which resolved the EKG abnormalities and muscle weakness within 2 h. Repeated potassium tests after 6 h consistently showed normal levels of 4.5 mmol/L

Upon revisiting the patient's history, he knew of no family members with periodic paralyses, muscle defects, or kidney disease. Personal history is negative for adrenal or thyroid disorder.

Discussion

The etiology of hypokalemia can often be gleaned from the patient history. Diuretic use and gastrointestinal losses from

Table 1. Laboratory evaluation.

Serum labs		Urinary labs	
Na	140 (136–145meq/L)	Osmolality	637 (300–1000 mosmkg)
K	1.7 (3.5–4.5 meq/L)	pH	6.0 (5.0–7.0)
Cl	110 (98–107 meq/L)	Na	131 mmol/L
HCO3	20 (22–29 mmol/L)	K	12 mmol/L
Glucose	210 (70–105 mg/dl)	Cl	176 mmol/L
BUN	20 (8–26 mg/dl)	U. Crea	66.3 mg/dl
S. Crea	1.03 (0.72–1.25 mg/dl)	U.K/U. Crea ratio	0.18 meq/mmol
Mag	2.1 (1.6–2.6 mg/dl)		
Anion gap	10 (8–16)		
eGFR	106		
TSH	0.61 (0.34–5.6 uIU/ml)		
Venous blood gas			
pH	7.31 (7.35–7.45)		
pCO2	38 (15–45 mmhg)		
HCO3	19 (18–26 mmol/L)		

U. K/U. Crea Ratio (<1.5 suggests poor intake, GI loss, transcellular shifts).

vomiting and diarrhea are among the common causes [12]. Although a decrease in intake can lower serum potassium levels, it is rarely sufficient to cause significant hypokalemia. A study by Gallen et al. (1998) restricted potassium intake to 20 meq per day, resulting in only a 0.6 meq/L reduction of potassium levels from the baseline [13].

In cases where the cause of hypokalemia is not readily apparent, assessment of renal potassium excretion is the next step in evaluation. The most accurate method is a 24-h urine collection. However, in the clinical setting, this is not the best test, as patients will need immediate repletion, which will invariably alter the results. The urine potassium-to-creatinine ratio (U. K-Cr) from a single random sample supplies comparable point-of-care information. Since more potassium is excreted in relation to constant creatinine excretion, higher U. K-Cr values reflect renal potassium wasting. Values less than 1.5 meq/mmol creatinine (13 meq/g creatinine) are seen in transcellular potassium shifts, gastrointestinal losses, diuretic use, and poor intake [14,15].

A study of 43 patients with severe hypokalemia (range, 1.5–2.6 mmol/L) associated with paralysis demonstrated the efficacy of U.K-Cr in distinguishing between hypokalemia from renal potassium wasting and that from potassium redistribution. The 30 patients with periodic paralysis (whose hypokalemia was caused by transcellular shifts) had significantly lower levels than the 13 patients with hypokalemia, due mostly to renal potassium wasting (11 *vs.* 36 meq/g creatinine or 1.3 *vs.* 4.1 meq/mmol creatinine) [15].

The trans-tubular potassium gradient (TTKG) gauges the renal potassium secretion by the cortical collecting duct.

Although this measurement cannot be made in humans, it was proposed that potassium concentration at this site could be estimated clinically [16–18]. However, in a later publication, the authors of the original studies found that these assumptions were not valid [19]. It was concluded that TTKG is not reliable in evaluating this condition.

In our patient, the U. K-Cr ratio was normal and did not reflect renal potassium wasting syndromes such as Bartter, Gittelman's, or renal tubular acidosis. Normal renal potassium handling, the absence of extra-renal losses, and rapid return to normal-high potassium levels with minimal repletion all support that the effect was transient and redistributive. The only remaining explanation is a "relative" decrease in serum potassium secondary to transcellular shifting.

The normal potassium distribution between and cells and the extracellular fluid is primarily maintained by the Na-K-ATPase pump in the cell membrane. Factors that increase the pump's activity and potassium entry into the cells are outlined in Table 2. The exact trigger in this case is unclear. The proposed mechanism is an exaggerated response to the epinephrine component of the TPI. Another theory is that the increased stress of the procedure lead to epinephrine release. In trauma patients, high levels of circulating epinephrine are negatively correlated with serum potassium [20], which is known as post-traumatic hypokalemia.

Table 2. Factors causing intracellular potassium translocation.

Factors causing intracellular potassium translocation
• Increased availability of insulin
• Elevated β-adrenergic activity – stress or administration of beta agonists
• Acidemia
• Hypokalemic periodic paralysis (HPP)
• Increased blood cell production
• Hypothermia
• Intoxication of barium, cesium, chloroquine, and some antipsychotics

The Naranjo nomogram estimates the causality of this event as a probable adverse drug reaction with a score of 6 [21]. A clinical review of the medications administered in the TPI show that methylprednisolone and epinephrine have the potential to cause hypokalemia, via mineralocorticoid effects by the former, and the latter through B2 adrenoreceptors linked to Na-K ATPase upregulation. The prevalence of hypokalemia in methylprednisolone therapy is 18% (95% CI 10.5–25.8) and causes only a mild reduction in serum potassium [22]. Bupivacaine, when used alone, does not cause significant hypokalemia. In 1994, Lofgren and Hahn studied the effects on plasma potassium levels when adding epinephrine to the local anesthetic solution for intercostal nerve block in patients undergoing upper abdominal surgery. When used in combination, bupivacaine and epinephrine produced a modest decrease in plasma potassium compared to bupivacaine used alone, especially after surgery (0.58 mmol/L *vs.* 0.10 mmol/L $P<0.03$) [23].

Uncommon causes of sporadic (periodic) paralyses were also considered. Patients with hypokalemic periodic paralysis (HPP) generally have a family history of the disorder. Its onset appears early (during childhood and late teenage years) and is triggered by stress and high-carbohydrate meals. Thyrotoxic paralysis presents with abnormal thyroid hormone levels during the acute attack. Lastly, Andersen-Tawil syndrome and para-myotonia congenita are rare genetic disorders evident during early childhood. Our patient's case profile was inconsistent with all of these possibilities. The diagnosis of hypokalemia secondary to TPI medications was made after excluding all other possible etiologies.

Rapid onset of severe symptomatic hypokalemia warrants urgent investigation and requires immediate treatment [14]. It is critical to distinguish hypokalemia caused by redistribution from that caused by depletion. In contrast to those with marked depletion, patients with hypokalemia due to potassium redistribution have little or no potassium deficit and are at risk for rebound hyperkalemia after minimal potassium repletion [24,25]. As seen in a study by Lin et al. (2004), 19 out of 30 patients with periodic paralysis developed rebound hyperkalemia (serum potassium greater than 5 meq/L) after administration of a mean of 63 meq of potassium chloride [15].

The importance of prompt treatment cannot be overstated. Apart from life-threatening arrhythmias, "judicious" early repletion precludes the clinician from having to face the potential diagnostic and therapeutic dilemma once ischemic rhabdomyolysis has set in. The release of potassium from injured myocytes will mask the severity of the underlying hypokalemia or even lead to normal or high values. Although treatment for hypokalemia seems straightforward, keeping in mind the primary pathology will appropriately guide the clinician and enhance patient safety.

Conclusions

Transcellular shifting is a common cause of hypokalemia. Severe hypokalemic paralysis in the context of trigger-point injection is an incredibly rare occurrence and this is the first case report in English literature. A high index of clinical suspicion and a systematic approach are therefore required for prompt diagnosis and management of this obscure iatrogenic entity.

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References:

1. Tough EA, White AR, Richards S, Campbell J: Variability of criteria used to diagnose myofascial trigger point pain syndrome – evidence from a review of the literature. Clin J Pain, 2007; 23(3): 278–86
2. Simons DG, Travell JG, Simons LS: Travell & Simons' myofascial pain and dysfunction: The trigger point manual. v. 1. Upper half of body. 2nd ed. Baltimore: Williams & Wilkins, 1999
3. Sorrell MR: The physical examination of migraine. Curr Pain Headache Rep, 2006; 10(5): 350–54
4. Borg-stein J, Iaccarino MA: Myofascial pain syndrome treatments. Phys Med Rehabil Clin N Am, 2014; 25(2): 357–74
5. Scott NA, Guo B, Barton PM, Gerwin RD: Trigger point injections for chronic non-malignant musculoskeletal pain: A systematic review. Pain Med, 2009; 10(1): 54–69
6. Venâncio Rde A, Alencar FG, Zamperini C: Different substances and dry-needling injections in patients with myofascial pain and headaches. Cranio, 2008; 26(2): 96–103
7. Certa H, Fedtke N, Wiegand HJ et al: Toxicokinetics of p-tert-octylphenol in male Wistar rats. Arch Toxicol, 1996; 71(1–2): 112–22
8. Wong CS, Wong SH: A new look at trigger point injections. Anesthesiol Res Pract, 2012; 2012: 492452
9. Payne JM: Ultrasound-guided hip procedures. Phys Med Rehabil Clin N Am, 2016; 27(3): 607–29
10. Cheng J, Abdi S: Complications of joint, tendon, and muscle injections. Tech Reg Anesth Pain Manag, 2007; 11(3): 141–47
11. Kim D, Kim O: A case of hypokalemic paralysis provoked after trigger point injection with dexamethasone and lidocaine. J Korean Neurol Assoc, 2007; 25(2): 225–28
12. Weiner ID, Wingo CS: Hypokalemia – consequences, causes, and correction. J Am Soc Nephrol, 1997; 8(7): 1179–88
13. Gallen IW, Rosa RM, Esparaz DY et al: On the mechanism of the effects of potassium restriction on blood pressure and renal sodium retention. Am J Kidney Dis, 1998; 31(1): 19–27
14. Lin SH, Lin YF, Halperin ML: Hypokalaemia and paralysis. QJM, 2001; 94(3): 133–39
15. Lin SH, Lin YF, Chen DT et al: Laboratory tests to determine the cause of hypokalemia and paralysis. Arch Intern Med, 2004; 164(14): 1561–66
16. West ML, Marsden PA, Richardson RM et al: New clinical approach to evaluate disorders of potassium excretion. Miner Electrolyte Metab, 1986; 12(4): 234–38
17. Ethier JH, Kamel KS, Magner PO et al: The transtubular potassium concentration in patients with hypokalemia and hyperkalemia. Am J Kidney Dis, 1990; 15(4): 309–15
18. Choi MJ, Ziyadeh FN: The utility of the transtubular potassium gradient in the evaluation of hyperkalemia. J Am Soc Nephrol, 2008; 19(3): 424–26
19. Kamel KS, Halperin ML: Intrarenal urea recycling leads to a higher rate of renal excretion of potassium: an hypothesis with clinical implications. Curr Opin Nephrol Hypertens, 2011; 20(5): 547–54
20. Beal AL, Deuser WE, Beilman GJ: A role for epinephrine in post-traumatic hypokalemia. Shock, 2007; 27(4): 358–63
21. Naranjo CA, Busto U, Sellers EM et al: A method for estimating the probability of adverse drug reactions. Clin Pharmacol Ther, 1981; 30(2): 239–45
22. Tamez-Pérez HE, Cisneros-Pérez V, Cedillo-Rodríguez JA et al: [Prevalence of hypokalemia in patients with methylprednisolone pulse therapy]. Rev Invest Clin, 2009; 61(3): 194–97 [in Spanish]
23. Löfgren A, Hahn RG: Hypokalemia from intercostal nerve block. Reg Anesth, 1994; 19(4): 247–54
24. Schaefer M, Link J, Hannemann L, Rudolph KH: Excessive hypokalemia and hyperkalemia following head injury. Intensive Care Med, 1995; 21(3): 235–37
25. Hsiao YH, Fang YW, Leu JG, Tsai MH: Hypokalemic paralysis complicated by concurrent hyperthyroidism and hyperaldosternoism: A case report. Am J Case Rep, 2017; 18: 12–16