

Carpopedal Spasm in an Elderly Woman

Ranjan K. Singh

1.

Corresponding author: Ranjan K. Singh, dr_ranjankumarsingh@yahoo.com

Abstract

An elderly woman developed carpopedal spasm following antihypertensive drugs--losartan and hydrochlorothiazide. Substitution of losartan and hydrochlorothiazide with another drug, s-amlodipine, ameliorated spasm. A case is discussed with potential mechanism of development of carpopedal spasm. Although such a complication has not been reported before, carpopedal spasm has been reported when hydrochlorothiazide given concurrently with omeprazole.

Categories: Internal Medicine

Keywords: hypokalemia, hypocalcemia, losartan, hydrochlorothiazide, carpopedal spasm, hypertension

Introduction

An elderly woman in all probability develops hypocalcemia due to vitamin D deficiency in the absence of anticonvulsant drugs or chronic renal failure [1]. The development of neuromuscular excitability, such as carpopedal spasm, depends on absolute concentration of calcium [1]. Concurrent presence of hypokalemia and hypomagnesemia can potentiate the neurological abnormalities associated with hypocalcemia [2]. Ault and Geiderman [3] have reported association of carpopedal spasm and hypokalemia. A combination of angiotensin receptor blocker - losartan and thiazide diuretic hydrochlorothiazide started in an elderly hypocalcemic woman, precipitated carpopedal spasm by causing hypokalemia. A case is discussed here.

Case Presentation

In May 2011, a 65-year-old female patient was started on antihypertensive drugs (losartan 50 mg and hydrochlorothiazide 12.5 mg q.d.) for Stage II hypertension (JNC 7 classification). She was non-diabetic. Her renal function and serum electrolytes were normal before starting the antihypertensive drug. Although she became normotensive within a fortnight, she developed involuntary rigid painful muscle contraction over hands and feet (carpopedal spasm). It further progressed to cramps in muscles of back and face in a week time. She received IV 10% calcium gluconate for severe truncal spasm. She had neither seizures nor a history of taking an anticonvulsant drug. Clinical examination revealed positive Chvostek's sign. Respiratory rate was 18/minute. Pulse was 100 bpm, and blood pressure was 130/80. CBC was normal. Urinalysis yielded no abnormality. Corrected calcium level was 2.0mmol/l (2.2-2.6) with PTH 71.48ng/L (ref. value - 10-60). Serum Na⁺ and K⁺ were 135mmol/l and 2.7mmol/l, respectively. Serum creatinine was 53.04μmol/L (ref. 53.04 - 106.08) 0.6mg/dl and serum aldosterone was 0.28nmol/L (ref. value supine .08 - .044, standing 0.19-0.83). Thyroid function was normal. ECG showed sinus tachycardia with prolonged QTc interval (70ms). CXR and USG of abdomen were normal. Supplementation of vitamin D analogue and hydroxycalciferol with calcium did not ameliorate spasmodic episodes even after 15 days. Level of corrected calcium and potassium remained 2.0mmol/l with potassium 2.8mmol/l, respectively. Substitution of losartan and hydrochlorothiazide by another antihypertensive drug, s-amlodipine, led to gradual subsidence of spasmodic episodes in a week time. Serum potassium and corrected calcium rose to 3.6mmol/L and 2.2mmol/L, respectively. PTH level was within normal range.

Discussion

Hypocalcemia from deficient vitamin D production or absorption stimulates the release of parathyroid hormone (PTH), resulting in secondary hyperparathyroidism [4]. The attendant decrease in serum calcium triggers rise in PTH in an attempt to maintain calcium level in the blood. Raised PTH level also activates Renin Angiotensin Aldosterone System (RAAS) and contributes to development of hypertension. Hydrochlorothiazide causes hypokalemia because of increased potassium excretion in urine and redistribution of potassium inside the cells following hypochloremic metabolic alkalosis. Hypokalemia is probably an over-feared complication of low dose thiazide diuretic, but hydrochlorothiazide in a dose of 12.5 mg/day may induce potassium wastage and hypokalemia [5-6]. Elderly people remain at risk of developing hypomagnesemia following hydrochlorothiazide [7]. Losartan may attenuate hypokalemia in response to decrease in renal perfusion, distal tubular function, and aldosterone level [8]. However, losartan in a dose of 50mg/day does not alter potassium level in normal functioning kidney, except in renal failure, diabetes, renal artery stenosis, or along with other potassium sparing diuretics [9]. In addition, aldosterone levels remain normal because decreases in plasma aldosterone levels have been found only with high dose of losartan (120 mg/day) [9]. Hydrochlorothiazide-generated hypokalemia can lead to carpopedal spasm unresponsive to calcium and vitamin D analogue. Moreover, muscle cramp as an adverse event during

Received 02/03/2013
Review began 02/03/2013
Published 02/18/2013

© Copyright 2013

Singh. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 3.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

losartan therapy exacerbates spasmodic event. Withdrawal of combination drug (losartan and hydrochlorothiazide) ameliorates carpopedal spasm.

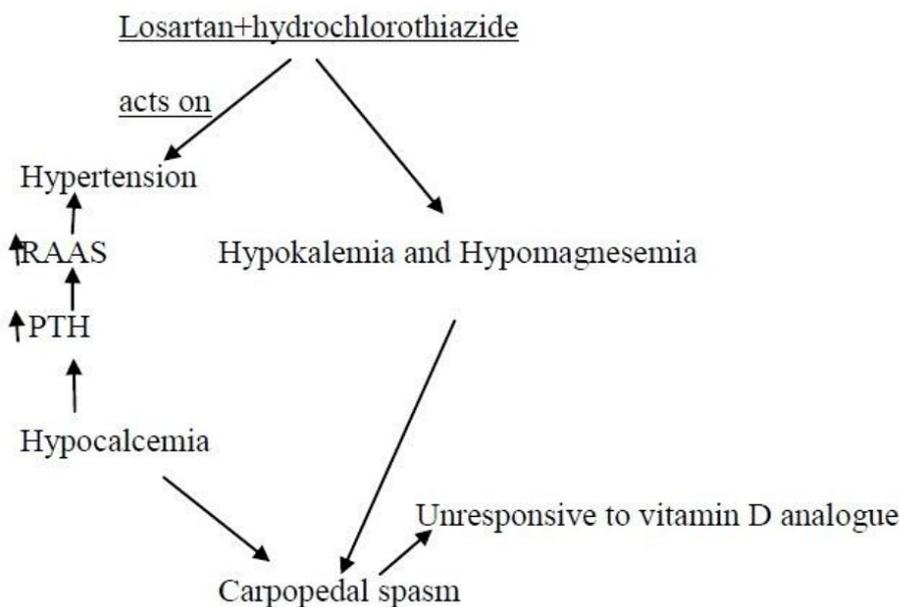


FIGURE 1: Potential Mechanism of Carpopedal Spasm in Elderly Receiving Losartan And Hydrochlorothiazide Drugs

RAAS-Renin angiotensin aldosterone system, PTH-Parathyroid hormone

Conclusions

An elderly hypertensive woman developed carpopedal spasm following a combination of antihypertensive drugs (hydrochlorothiazide and losartan). Carpopedal spasm was unresponsive to vitamin D analogue unless both drugs were restricted.

Additional Information

Disclosures

Human subjects: Consent was obtained by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

- Cooper MS: Diagnosis & management of hypocalcemia . Br Med J . 2008, 336:1298-1302. [10.1136/bmj.39582.589433.BE](https://doi.org/10.1136/bmj.39582.589433.BE)
- Norma M. Metheny: Calcium imbalances. Fluid and Electrolyte Balance. Jones & Bartlett Learning; 2012. 91-109.
- Ault MJ, Geiderman J: Hypokalemia as a Cause of Tetany . Western J Med . 1992, 157:65- 66.
- Thomas MK, Demay MB: Vitamin D deficiency and disorders of Vitamin D metabolism . Endocrinol Metab Clin North Am. 2000, 29:611-27. [10.1016/S0889-8529\(05\)70153-5](https://doi.org/10.1016/S0889-8529(05)70153-5)
- Franse LV, Pahor M, Di Bari M, Somes GW, Cushman WC, Applegate WB: Hypokalemia associated with diuretic use in cardiovascular events in the Systolic Hypertension in the Elderly Program. Hypertension . 2000, 35:1025-30. [10.1161/01.HYP.35.5.1025](https://doi.org/10.1161/01.HYP.35.5.1025)
- Zillich AJ, Garg J, Basu S, Bakris GL, Carter BL: Thiazide diuretics, potassium, and the development of diabetes: a quantitative review. Hypertension . 2006, 48:219-224.
- Hydrochlorothiazide. <http://www.rxmed.com/>.
- Palmer BF: Managing hyperkalemia caused by inhibitors of the renin angiotensin system . N Eng J Med . 2004, 351:585-592. [10.1056/NEJMr035279](https://doi.org/10.1056/NEJMr035279)
- Burnier M, Waeber B, Brenner HR: Clinical pharmacology of the angiotensin II receptor antagonist losartan potassium in healthy subjects. Hypertension . 1995, 13 :523-8. [10.1097/00004872-199507001-00003](https://doi.org/10.1097/00004872-199507001-00003)