Renal calculi in primary hyperaldosteronism

Udaya M Kabadi

Summary

Increased urinary calcium (Ca^{++}) excretion and the presence of negative Ca^{++} balance is well documented in primary hyperaldosteronism. However, renal calculi as a major manifestation of this disorder has not previously been described. This report describes a patient who presented with renal calculi in association with primary hyperaldosteronism. We believe that primary hyperaldosteronism was a major pathogenetic factor in the formation of renal calculi since the increased urinary excretion of Ca^{++} and uric acid noted at onset declined following a short-term spironolactone administration and remission from renal calculi has persisted following initial nephrolithotomy and continued spironolactone therapy, which also corrected hypertension and hypokalemia, a hallmark of this disorder.

Keywords: renal calculi, hyperaldosteronism

Primary hyperaldosteronism is reported to facilitate urinary calcium excretion and induce a negative calcium balance.¹ However, the occurrence of renal calculi in this disorder has not been reported. Herein, we describe a patient, in whom renal calculi were present for several years prior to diagnosis of primary hyperaldosteronism and continuous therapy with spironolactone (Searle, Skokie, IL, USA), following initial nephrolithotomy, prevented subsequent stone formation during the follow-up period of 12 years.

Case report

A 47-year-old white man presented for evaluation of hypertension and persistent hypokalemia (serum K^+ , $<3.0 \text{ mM/l}$) while not receiving therapy with diuretics. The patient related presence of hypertension for 10 years, initially detected at the time of hospitalisation for management of subarachnoid haemorrhage secondary to a rupture of an intracranial arteriovenous malformation. Several regimens using different drugs were not optimally effective in controlling blood pressure with recordings ranging from 140/85 to 170/110 mmHg. He also related recurrent passage of stones and gravel in the urine with several short hospitalisations and visits to emergency rooms over the previous 3–4 years. The diagnosis of idiopathic primary hyperaldosteronism

was established by documentation of low renin ($<0.2 \text{ ng/ml/h}$) and elevated plasma aldosterone ($42\text{--}54 \text{ ng/dl}$; normal $3\text{--}10 \text{ ng/dl}$) on several occasions as well as the lack of suppression following a high sodium (300 mM/day) diet for three days and further confirmed by abdominal computed tomography (CT) scan, showing bilateral adrenal enlargement, arteriography with no unilateral localisation, ie, tumour blush and raised aldosterone concentrations in bilateral adrenal venous blood samples without a significant gradient between two sides, as well as a lack of suppression on oral administration of 8 mg cyproheptadine (Merk, Sharp & Dohme Inc, West Point, PA, USA), as described previously.² Intravenous pyelography revealed bilateral multiple renal calculi. Spironolactone therapy in gradually increasing dosage with a final daily dose of 300 mg permitted withdrawal of other antihypertensive drugs and normalised serum K^+ level between $4.2\text{--}4.6 \text{ mM/l}$ as well as 24-h urinary excretion of Ca^{++} and uric acid (table). Subsequently, the patient underwent elective bilateral nephrolithotomy in two stages in 1981 and 1982. The chemical analyses of the calculi demonstrated both calcium urate and pyrophosphate crystals. During the follow-up period of about 12 years, the patient reported no recurrence of renal colic, nor gross haematuria and a recent intravenous pyelography revealed no renal calculi. Finally, 24-h urinary Ca^{++} and uric acid excretion have also remained normal (table).

Discussion

Chronic mineralocorticoid administration and primary hyperaldosteronism are both known to facilitate renal Ca^{++} excretion.^{1,3–5} Furthermore, excretion of acidic urine due to increased tubular reabsorption of bicarbonate induced by elevated aldosterone may also enhance urate crystallisation and promote formation of urate stones. Finally, normalisation of 24-h urinary Ca^{++} and uric acid excretion as well as total remission from new stone formation with spironolactone therapy in our patient definitely indicate primary hyperaldosteronism to be responsible for formation of renal calculi and not just a coincidental occurrence. We believe that the use of spironolactone or other agents (eg, ACE inhibitors) causing hypoaldosteronism may be extended also to patients with mixed renal calculi who receive a multiple drug regimen, eg, thiazides, allopurinol, and/or alkalinising solutions.

The author would like to thank Marcia Gregory for secretarial assistance.

Medical Service, VA Medical Center, Phoenix, and College of Medicine, University of Arizona, Tucson, Arizona, USA
UM Kabadi

Correspondence to
Udaya M Kabadi, Chief, Endocrine Section, Medical Service (111E), VA Medical Center, 650 East Indian School Road, Phoenix, AZ 85012, USA

Accepted 15 March 1995

Table 24-h urinary excretion of calcium (Ca^{++}) and uric acid prior to (pre Rx), and during therapy (post Rx) with spironolactone at 2 months and again at 12 years in a patient with primary hyperaldosteronism and renal calculi. Figures are given as mean $\pm$ SEM of four values at each time point

	Calcium (mM/day)	Uric acid (mM/day)
pre Rx	104.5 $\pm$ 3.7	5.10 $\pm$ 0.13
post Rx (2 months)	55.5 $\pm$ 2.0	3.18 $\pm$ 0.10
post Rx (12 years)	54.30 $\pm$ 2.31	3.25 $\pm$ 0.08
normal value	62.5–75.0	3.87–4.16

Summary points

- renal calculi may occur in primary hyperaldosteronism
- raised urinary excretion of calcium and uric acid may occur in primary hyperaldosteronism
- spironolactone therapy reduces urinary excretion of calcium and uric acid, and helps prevent recurrent renal calculi in primary hyperaldosteronism

- 1 Resnick LM, Largh JH. Calcium metabolism and parathyroid function in primary aldosteronism. *Am J Med* 1985; 78: 385–90.
- 2 Gross MD, Grekin RJ, Gniadek TC, Villareal J. Suppression of aldosterone by cyproheptadine in idiopathic aldosteronism. *N Engl J Med* 1981; 305: 181–5.
- 3 Luft R, Sjogren B. Some aspects of the metabolic effect of desoxycorticosterone acetate. *Metabolism* 1953; 2: 313–21.

- 4 Massry S, Coburn JW, Chapman LW, Kleenman CR. The effect of long-term desoxycorticosterone acetate administration on the renal excretion of calcium and magnesium. *J Lab Clin Med* 1968; 71: 212–9.
- 5 Wright GL, Rankin GD. Concentrations of ionic and total calcium in plasma of four models of hypertension. *Am J Physiol* 1982; 243: H365–70.

Giant cell carcinoma of the lung

Shiekh Aejaaz Aziz, Mushtaq Ahmad, Azra Shah, Gul Mohammad Bhat, Ajaz Lone, Khursheed Ahmad, Hajra Bano

Summary

A 50-year-old non-smoking, hypertensive female, presenting with superior vena caval compression, was found to have giant cell carcinoma of the lung. She received intensive combination chemotherapy. However she died in the following 36 hours, as a consequence of refractory hypotension.

Keywords: giant cell carcinoma, superior vena caval obstruction

Giant cell carcinoma is a rare, distinctive lethal variant of large cell cancer of the lung.¹ The tumour is quite extensive at diagnosis and survival for more than one year is exceptional.²

Case report

A 50-year-old, non-smoking, hypertensive woman was admitted to our hospital on 19 October 1994, with a non-productive cough and breathlessness of one month duration. Clinical examination revealed features suggestive of superior vena caval compression, with partial collapse of the right upper lobe of lung. Her blood pressure was 150/100 mmHg (on calcium channel blockers); the rest of her cardiovascular and other systemic examination was non-contributory. Fundus oculi were normal. Her investigations showed a normal haemogram and biochemical parameters. Electrocardiogram (ECG) was normal. Chest X-ray revealed a mass shadow in the right upper and middle zone with mediastinal invasion.

Abdominal ultrasonography was normal. Computed tomography (CT) scan of the chest showed a homogenous mass of soft-tissue density in the anterior and middle mediastinum, impressing on the arch of aorta and causing displacement and occlusion of the superior vena cava. There was compression of the right upper lobe bronchus with extension into the chest wall and pleural effusion on the right side (figure). A clinical diagnosis of bronchogenic carcinoma (T4N2MO-IIIB) was made. She was put on decompressive treatment and a Trucut biopsy was done, which subsequently revealed features suggestive of giant cell carcinoma of the lung. She was put on combination chemotherapy consisting of intravenous cyclophosphamide 750 mg/m², adriamycin 50 mg/m² and cisplatin 100 mg/m² on day 1; the

Figure CT scan of the chest showing a homogenous mass of soft density in the anterior and middle mediastinum with impression on arch of aorta, displacement and occlusion of superior vena cava with pleural effusion

Departments of
Medical Oncology &
Pathology,
Sher-i-Kashmir
Institute of Medical
Sciences, Srinagar,
India

SA Aziz
M Ahmad
A Shah
GM Bhat
A Lone
K Ahmad
H Bano

Correspondence to
Dr Shiekh Aejaaz Aziz,
Consultant, Department of
Medical Oncology,
Sher-i-Kashmir Institute of
Medical Sciences, Post Bag
No 27, Soura, Srinagar 190
011, India

Accepted 15 March 1995