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THE HYPONATREMIC HYPERTENSIVE SYNDROME: UNDERRATED DIAGNOSIS

Medicine

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ABSTRACT

The hypornatremic hypertensive syndrome is a rare and less recognized syndrome. The syndrome is characterized by hypertension and profound natriuriesis leading to sodium and water depletion. Hypertension is refractory to treatment. We report a 45 year old male with this syndrome with hyponatremia and confirmed renal artery stenosis. Syndrome should be suspected in patients in whom severe hypertension is associated with hyponaterima without apparent cause.

KEYWORDS:

Hyponatraemia, hypertension, atherosclerosis, renal artery stenosis

INTRODUCTION

The hyponatremic hypertensive syndrome is a rare complication of renal artery stenosis. Less number of cases have been reported so far. Features associated with syndrome includes hyponatremia, hypokalemia and elevated rennin activity. Patients with hyponatremic hypertensive syndrome may present with a spectrum of signs and symptoms including headache, confusion, postural dizziness, hypertensive neuropathy, polyuria, polydipsia, weight loss and salt craving [1,2]. The purpose of this report is to bring out the existence of this rare but yet underdiagnosed syndrome.

CASE HISTORY

An 45 year old male known case of hypertension, seizure disorder, chronic obstructive airway disease, ex smoker presented with complains of headache, giddiness and recurrent vomiting. He was admitted for workup and investigation. His blood pressure at the time of admission was 200/120mmhg. No such positive finding was found On examination.

On investigation the patient was found to be hyponatremic with a serum sodium concentration of 121mmol/l (normal range 135-145mmol/l). Serum urea was 55mg/dl and creatinine was elevated 3.4 mg/dl (normal range 0.6 to 1.2mg/dl). Serum potassium was 3.6 mEq/l (normal range 3.5-4.5 mEq/l). His GFR was 21ml/min. He weighed around 55 kg clinically. He had grade II hypertensive retinopathy.

The patient's blood pressure was poorly controlled despite adequate addition of drugs. Diuretic was stopped in view of persistent hyponatraemia. By the day 5 of admission patient's serum sodium concentration was fallen to <120mmol/l [table 1]. Patient was kept on NTG, calcium channel blockers, but his blood pressure did not lower down. With due course of admission sodium concentration remained low 118mmol/l and blood pressure remained elevated 190/110mmhg. He developed weakness, thirst and lethargy and recurrent vomiting and his weight fell to 52 kgs. His drugs for seizure and obstructive airway disease was continued.

Table 1: Serum electrolytes during admission.

Analyte	Units	Ref range	Day 1	Day 5	Day10	Day15	Day20	Day 25
Sodium	mmol/l	136-145	121	124	118	120	121	132
Potassium	mmol/l	3.5-4.5	3.6	4.2	3.6	4.5	3.8	4.4
Bld Urea	mg/dl	7-21	55	59	65	91	20	22
S. Creatinie	mg/dl	0.6-1.2	3.4	2.2	2.8	3.2	1.9	1.0

Number of investigations were done [Table2-3]. Carotid Doppler showed 64% stenosis at the bifurcation in left carotid artery. Ultrasound was done which showed bilateral MRD (medical renal diease). We got CT renal angiography which showed short segmental stenosis of left renal artery. A consultation from ctvs department was taken and patient was taken for stenting of renal artey.

Table 2: Other investigations:

Analyte	Units	Reference range	Result
HB	gm/dl	13-14	12.6
Wbc	Wbc/mm	4000-11000	5500
S. bilirubin	mg/dl	0.3-1.0	0.7
SGOT	IU	5-40	29
SGPT	IU	7-56	31
ALP	Units/l	44-147	116
TOTAL PROTEIN	g/100 ml	6-8.3	5.6
S. ALBUMIN	g/100ml	3.5 to 5.5	2.7
S CHOLESTROL	mg/dl	150-200	221
TRIGLYCERIDES	mg/dl	180-250	181
HDL	mg/dl	40-50	65
LDL	mg/dl	100-129	134
CALCIUM	mg/dl	8.5 to 10.2	8.9
URIC ACID	mg/dl	2.4-6.0	7.4
Urine osmolarity	mosmo/l	300-800	380
Spot urinary Na	mEq/l	0-20	54
Urine routine		2++ albumin	

Hb; heamoglobin, wbc; white blood cell, HDL; high density lipoprotein, lDL; low density lipoprotein.

Table 3: Analysis of 24 hour urinary collection:

Analyte	Units	Reference range	Result
Metanephrine	SI units	95-400	390

We defined hypertension either moderate to serve hypertension as a reading at least three consecutive occasions of greater than 165 mmhg systolic and >95 mmhg of diastolic. We defined significant hyponaterima as a serum sodium level of 130 mmol/l or less.

DISCUSSION

In this case we report that, hyponatremic-hypertensive syndrome following renal artery ischemia/stenosis. The combination of hyponatremia and severe hypertension in patients with underlying renal ischemia has been known for many years.

Criteria for diagnosis of the hyponatremic-hypertension syndrome caused by renal ischemia were as follows: (1) Hyponatremia, plasma sodium concentration below the lower limit of normal for our hospital (,136 mmol/L), (2) Hypertension, systolic blood pressure 165 mm Hg and diastolic blood pressure 95 mmhg in the sitting or supine

position in the presence or absence of antihypertensive drug treatment, (3) Renal ischemia, evidence of renal ischemia (80% stenosis or total occlusion of a major renal artery) on selective angiography or strongly suggestive evidence of severe ischemia[1].

The hyponatremic hypertensive syndrome is a consequence of multiple factors promoting sodium depletion in the presence of unilateral renal artery stenosis. Decreased perfusion of the glomerular apparatus distal to a serve renal artery stenosis leads to increased rennin and angiotensin II production, causing hypertension. Angiotensin II influences fluid and sodium reabsorption in the renal tubule in a biphasic dose dependent manner. Normal concentration of angiotensin II stimulate reabsorption. High concentration promote water and electrolyte loss [3].

Other causes of hyponatremia in hypertensive patients, include use of thiazide diuretics[4], rennin-secreting tumors[5], acute intermittent porphyria[6], malignant hypertension and chronic renal failures[7]. It is likely that hyponatremic- hypertensive syndrome evolves into malignant hypertension in some cases with development of pathological hallmark, arteriolar fibrinoid necrosis and clinical papilledema, retinal exudates and hemorrhages.

In our case the patient was a heavy smoker, Nicholson et al have shown an association between cigarette smoking and renal artery stenosis wheather the pathology was atherosclerotic or fibromuscular renovascular dieases[8]. Nicotine can be a potent stimulus to ADH release and may contribute to hyponatremia. Therefore smoking was central to the pathophysiology of hyponatremic-hypertensive syndrome, most obviously through development of atherosclerotic renovascular diseases[9].

The incidence of hyponatremia-hypertension syndrome with renal ischemia is not readily apparent from literature. Response to surgery, angioplasty, and drug therapy was not well documented. It is our hope that this report leads to, precareful documentation and long term follow up.

To summaries, we report 45 year old male as hyponatremic-hypertensive syndrome with renal ischemia with atherosclerotic disease of renal artery. Patient was a heavy smoker. The pathophysiology appears to center on intense activation of the rennin-angiotensin system with angiotensin II in concert with volume depletion stimulating thirst and ADH release and enchancing aldosterone secretion resulting in hyponatremia. The syndromes needs to be better recognized and worked up.

REFERENCES

1. Agarwal M, Lynn LK, Richards AM, Nicholls MG, Hyponatrmic-hypertensive syndrome with renal ischemia: An under recognized disorder, Hypertension 1999;33:1020-4.
2. Atkinson AB, Davies DL, Leckie B morton JJ, Brown JJ, Fraser R, et al. hyponatraemic hypertensive syndrome with renal artery occlusion corrected by captopril, lancet 1979;2: 606-9.
3. Wang T, Chan YL. Mechanism of angiotensin II action on proximal tubular transport. J Pharmacol Exp Ther 1990; 252: 689-95.
4. Fichman MP, Vorherr H, Kleeman CR, Telfer N. Diuretic-induced hyponatremia. Ann Intern Med. 1971;75:853– 863.
5. Misiani R, Sonzogni A, Poletti EM, Cassinelli G, Gualandris L, PericottiS, Ghislandi R, Agazzi R, Belair M-F, Bruneval P. Hyponatremic hypertensivesyndrome and massive proteinuria in a patient with reninproducing leiomyosarcoma. Am J Kidney Dis. 1994;24:83– 88.
6. Qadiri MR, Church SE, McColl KEL, Moore MR, Youngs GR. Chester porphyria: a clinical study of a new form of acute porphyria. Br Med J.1986;292:455– 459.
7. Gennari FJ. Hypo-hypernatraemia: disorders of water balance. In:Davison AM, Cameron JS, Gru¨nfeld J-P, Kerr DNS, Ritz E, WinearlsCG, eds. Oxford Textbook of Clinical Nephrology. Oxford, UK: Oxford University Press; 1998:175–200.
8. Nicholson JP, Alderman MH, Pickering TG, Teichman SL, Sos TA,Laragh JH. Cigarette smoking and renovascular hypertension. Lancet.1983;2:765–766.
9. Husain K, Frantz AG, Ciarochi F, Robinson AG. Nicotine-stimulatedrelease of neurophysin and vasopressin in humans. J Clin Endocrinol Metab. 1975;41:1113–1117.