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Case Report

Sympathetic crashing acute pulmonary edema secondary to bilateral renal artery stenosis

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ABSTRACT

Bilateral renal artery stenosis (RAS) represents a notable etiology of renovascular hypertension, resulting in diverse organ complications. One of the rare complications associated with this condition is sympathetic crashing acute pulmonary edema (SCAPE). This is a case presentation of a man in the fifth decade presented with SCAPE in our ICU department.

Keywords: RAS, Pulmonary edema, Intensive care unit, Sympathetic crashing

INTRODUCTION

Renal artery stenosis (RAS) is the constriction of the one or both of renal arteries. It constitutes the predominant cause of hypertension in 1-10% of 50 million individuals in United States.[1] Atherosclerosis or fibromuscular dysplasia commonly leads to this condition, as end result it may lead to chronic kidney disease.[2,3]

Atherosclerotic blockage in the renal arteries worsens over time. Rapid development of complete blockage in both renal arteries can lead to acute kidney failure and sudden fluid overload causing rapid onset pulmonary edema.[4] We present a case of a SCAPE complicating an incidental finding of RAS.

CASE REPORT

A forty year old male with free medical background admitted To the ICU due symptoms and signs of pulmonary edema. The condition started 5 days prior to admission by high grade fever, hemoptysis, and mild S.O.B. Initial lab investigations showed a decrease in TWBCs and platelets, therefore he was provisionally diagnosed as dengue fever (which was meanwhile an endemic disease). Three days later he developed lower limb edema and became extremely dyspneic and desaturated on room air (80%), also he had severe productive cough, very high blood pressure 267/125, and RR 50.

Investigations

Further lab investigations showed high renal markers with both hypokalemia and hyponatremia, in addition to mild hypocalcemia, Urinalysis uncovered proteinuria and countable pus cells. In CBC, there was anemia and thrombocytopenia, high CRP and ESR. Blood clotting tests showed mildly elevated PT and INR but normal PTT. Parathyroid hormone markedly elevated (Table 1).

Chest X-ray showed diffuse pulmonary oedema. Pleural effusion was not detectable in the image (Figure 1).

USG revealed bilateral RAS with reno-vascular hypertension, bilateral pleural effusion, and features of acute-on-chronic kidney disease (Figure 2).

Management

The patient was put on continued BIPAP with high settings, aggressive diuretics, and IV nitroglycerin. After 24 hours the patient responded to the management showing improvement regarding oxygen requirements, control of blood pressure, and symptoms and signs of pulmonary disease and he was discharged from ICU after 5 days in good condition and arrangements for transfer to underwent bilateral stent placement was commenced.

Follow up

While most of the lab markers resolved to basal levels, renal profile and white cells count remained high. We noted that while blood urea was stepping up, the creatinine levels were fluctuating (Figure 3).

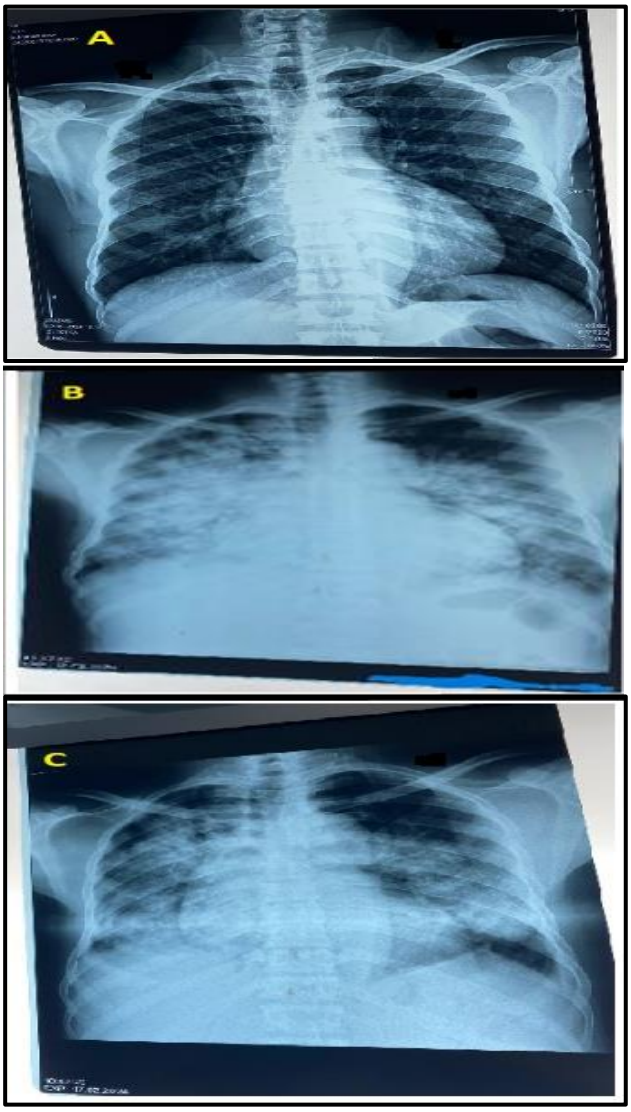

Figure 1 (A-C): The chest x-ray of the patient at presentation. After one week, after 12 days post admission and the increase in pulmonary congestion. The improvement after commencing the GTN infusion in addition to other medication.

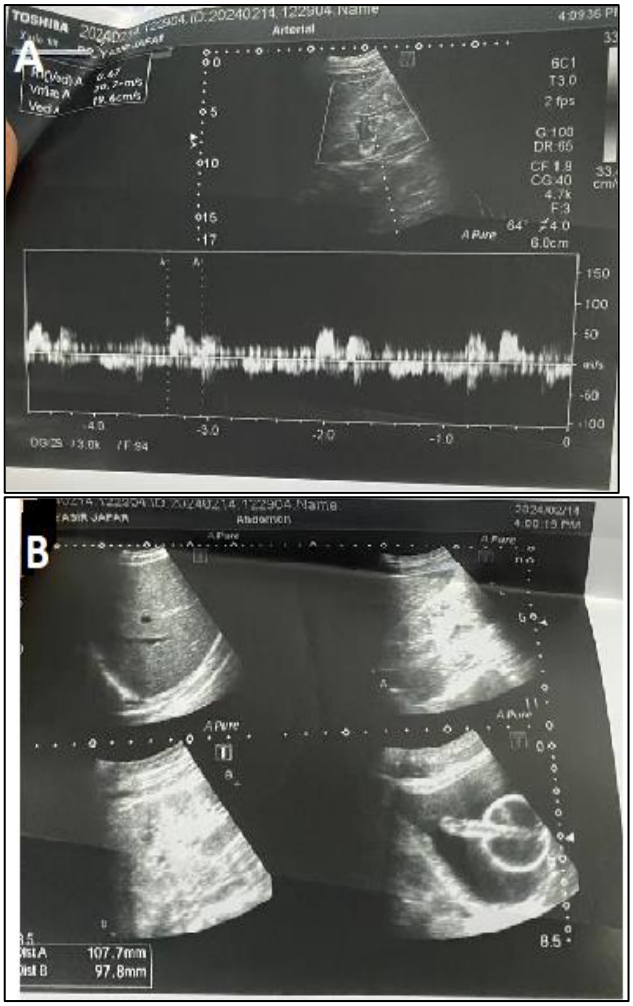

Figure 2 (A and B): Doppler ultrasound at presentation.

A Doppler ultrasound of the patient showing decrease vascular perfusion with a triphasic spectrum waveform and increase resistivity indices in both kidneys.

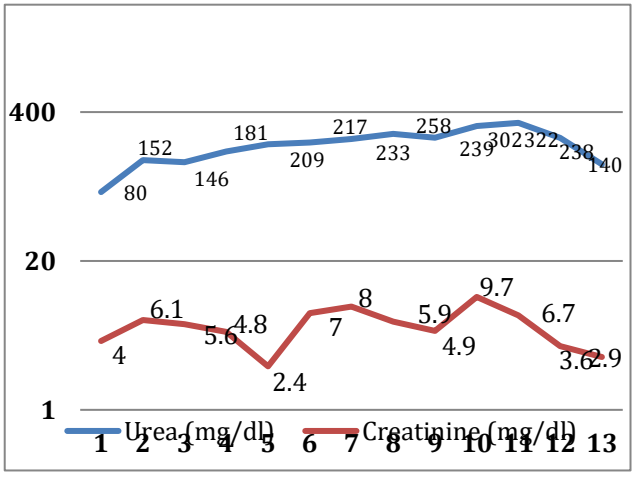

Figure 3: Daily comparative chart of the patient's serum urea and creatinine.

Urea and creatinine daily chart. Horizontal axis represents the day intervals of ICU admission. *Some data were missing before downgrading to the ward.

Table 1: Laboratory results of the CBC, ESR, CRP, blood clotting profile, and serum parathyroid hormone that had been taken from the patient's blood. Apart from the complete blood count, the other tests shown were taken sporadically.

Dates	CBC									Blood clotting tests			
	Hb gm/dl	RBCS million/mm^3	TWBC S/mm^3	Neutrophil (%)	Lymphocytes (%)	Eosinophil (%)	Platelet/mm^3	CRP mg/dl	ESR mm/hr	PT (sec)	PTT (sec)	INR ratio	PTH pg/ml
5th Feb	10.0	3.540*	8800	74%*#	17%	2%	83000$		60*				
8th Feb	10.1	3.660*	17700*	75%*#	16%	2%	181000						
11th Feb	9.3*	3.200*	14900*	81%*	12%	1%	149000$	57.7*		16.9*	30.3	1.4*	
12th Feb	8.4*	3.100*	14500*	94%*	3%	1%	144000$						
13th Feb	9.5*	3.290*	18600*	82%*	7%	1%	224000			18.4*	33.8	1.6*	
15th Feb	8.7*	3.050*	11100*	87%*	6%	2%	248000						
16th Feb	9.1*	3.430*	11600*	90%*	4%	1%	339000						
17th Feb	8.8*	3.230*	12100*	83%*	8%	2%	394000						
18th Feb	9.0*	3.190*	11600*	81%*	10%	2%	354000	36.0*					
21st Feb	9.8*	3.420*	19300*	88%*	7%	1%	381000						193*
23rd Feb	10.0	3.560*	12000*	79%*	12%	1%	339000						
24th Feb	10.5	3.460*	10400	80%*	11%	1%	349000						
25th Feb	10.5	3.750*	11200*	80%*	11%	1%	330000						

*Results above normal and $ below normal. #Some laboratory references consider these results as normal values.

DISCUSSION

In patients with bilateral RAS, the disruption of normal renal function and regulation of fluid balance can result in a significant activation of the sympathetic nervous system. This exaggerated sympathetic response is a key feature of SCAPE.[5]

Bilateral RAS can cause SCAPE by three main mechanisms: Impaired sodium excretion, rise hemodynamic load resulting in diastolic dysfunction, and disruption of pulmonary capillary blood-gas barrier.[6]

Renal ischemia caused by RAS results in activation of the renin-angiotensin system, leading to stimulation of the sympathetic nervous system, which causes LV wall stress and an imbalance between increased oxygen demand and supply to the myocardium. This worsens the LV diastolic dysfunction leading to a further rise in LV end diastolic pressure.[7,8]

What sets SCAPE apart from other types of decompensated heart failure is the sudden elevation of left ventricular end diastolic pressure that quickly fills the alveolar space, leading to a critical and life-threatening emergency.[9]

Diagnosis of SCAPE is based purely on clinical findings. Individuals who suffer from SCAPE experience a sudden beginning of difficulty breathing that worsens within minutes to hours, leading to severe pulmonary edema.[10]

In SCAPE, unlike other acute heart failure syndromes, the primary goal is to quickly reduce afterload to interrupt the vicious cycle caused by the sympathetic surge, leading to enhancement in cardiac output and diastolic dysfunction. Hence, it is recommended to start nitroglycerin at a high dose with vasodilator effects instead of slowly increasing the dose.[11]

Loop diuretics like furosemide have been mainly utilized for treating acute pulmonary edema. Still, even with its widespread use, there is limited evidence backing its effectiveness in treating SCAPE patients.[12]

Non-invasive ventilation (NIV) is a successful treatment option that offers oxygenation and reduces the workload of the heart by lowering preload and afterload. It is linked to lower rates of invasive mechanical ventilation and reduced mortality in patients with cardiogenic pulmonary edema, thus lowering the occurrence of related complications.[13,14]

Regarding RAS treatment options include: medical management, surgical management, or percutaneous therapy.[15]

The degree of RAS that would justify any intervention attempt is greater than 80% in patients with bilateral stenosis or stenosis in a solitary functioning kidney regardless of whether they have renal insufficiency or not.[16]

CONCLUSION

SCAPE is a serious complication of bilateral RAS that requires prompt recognition and a tailored management approach. Understanding the pathophysiology, clinical presentation, and management strategies for this condition is crucial for providing appropriate care to patients with this life-threatening complication of reno-vascular hypertension.

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