

CASE REPORTS

RENAL VEIN THROMBOSIS AT HIGH ALTITUDE

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ABSTRACT

Though risk of venous thromboembolism is increased at high altitude, involvement of renal veins is rare. A 27-year-old soldier was evacuated from a height of 18000 feet after developing flank pain and haematuria. Ultrasound showed an enlarged left kidney and lack of flow in proximal renal vein. CT scan confirmed the presence of renal vein thrombus. Serum IgM Anti β -2 Glycoprotein I antibodies, IgG Anti β -2 Glycoprotein I antibodies and IgM anticardiolipin antibodies were detectable and the former two were present after three months also. Secondary causes of antiphospholipid syndrome were excluded. He was started on low molecular weight heparin and is now on lifelong oral anticoagulation with warfarin.

Keywords: Antiphospholipid syndrome, Haematuria, Thrombosis.

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INTRODUCTION

Prolonged presence at high altitudes increases risk of innumerable health hazards. There is a high burden of diseases such as frost bite, acute mountain sickness, high altitude cerebral edema and high altitude pulmonary edema, attributed to the height and cold environment. Venous thrombosis is also more common at high altitude, with several pathophysiological changes predisposing to this. Initially, there is a transient increase in platelet count, increased factor X and XII activity, shortening of prothrombin time, impaired clot retraction and platelet dysfunction. In the later stages, there is an increase in haematocrit, factor VIII levels, platelet adhesiveness and thromboxane activity¹. This usually manifests as deep vein thrombosis in legs and less frequently as cerebral venous sinus thrombosis². Other sites are less frequently involved. We are reporting an unusual case of renal vein thrombosis precipitated by exposure to high altitude with underlying predisposition.

CASE REPORT

A 24-year-old patient was evacuated to Combined Military Hospital Skardu from a height of

18000 feet, with a history of abdominal pain for a week and gross haematuria of two days' duration. There was no history of fever, anorexia, nausea, vomiting, dyspnea, cough, swelling of feet or reduced urine output. He did not report any reduction in physical activity or immobility. He was not known to have nephrolithiasis and never had similar pain in the past. He had been deployed at this altitude for two months. Prior to this, he had been serving in the northern areas for 18 months, though he resided at lower altitudes. Family and drug histories were not contributory. Physical examination was unremarkable. Initial investigations revealed haemoglobin 13.2g/dl and platelets 493,000/ul. Total leukocyte count and serum urea/creatinine were normal. Urine routine examination showed numerous red blood cells per high power field and trace proteinuria. Spot urinary protein: creatinine ratio was 44 mg/mmol. Ultrasound Kidneys Ureter and Bladder (KUB) showed an enlarged left kidney with hypoechoic thickened cortex (fig-1). Doppler scan revealed a dilated left renal vein with internal echoes. Flow could be seen in distal segment only and there was no clear flow in renal vein tributaries. Contrast enhanced CT scan KUB demonstrated non-enhancing parenchyma in mid/lower pole with sparing of cortex in upper pole and the renal capsule (cortical rim sign). A filling defect

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(thrombus) was seen in the left renal vein (fig-2). A diagnosis of left renal vein thrombosis was made, and the patient was started on warfarin (overlapped with enoxaparin sodium initially). He was then transferred to Pak Emirates Military Hospital Rawalpindi for further workup. Serum IgM Anti β -2 Glycoprotein I antibodies were positive (11 U/ml), as were IgG Anti β -2 Glycoprotein I antibodies (63 U/l) and IgM anticardiolipin antibodies (15 U/ml). Serum IgG anticardiolipin

ring 6.95 cm with irregular contours, cortical thinning and mild perinephric fat stranding. Based on one clinical feature and one laboratory feature of Sydney Classification Criteria, a definitive diagnosis of left renal vein thrombosis secondary to primary antiphospholipid syndrome was established. The patient was counseled and advised to use warfarin for the rest of his life.

DISCUSSION

Nephrotic syndrome and dehydration are the most common causes of renal vein thrombosis in adults and infants respectively. However, many other conditions are also associated with renal vein thrombosis, including renal cell cancer, renal transplantation, Behçet syndrome, extrinsic compression (e.g. lymph nodes, tumor, retroperitoneal fibrosis, aortic aneurysm), trauma and oral contraceptive pills. Prothrombotic conditions, including Antiphospholipid Syndrome (APS), may also be responsible. This syndrome is also named after Dr Hughes, who first described it in 1983³. It may either be primary or secondary to a broad range of causative factors, including systemic autoimmune diseases, infections, drugs, malignancies and haematological disorders described elsewhere⁴.

Antiphospholipid antibodies stimulate platelet aggregation. They also activate endothelial cells, resulting in release of proinflammatory cytokines. APA binding to β 2-glycoprotein, prothrombin and protein C and S also interferes with coagulation cascade. All these processes are thrombogenic, but a second hit is often required for clot formation. This could be in the form of trauma, infection or nonimmune procoagulant factors⁵. In our patient, the second hit was most probably dehydration at high altitude. We initially thought of high altitude as the only cause for developing thrombosis, but his normal haemoglobin and hematocrit levels went against this hypothesis.

We did not test for lupus anticoagulant because the patient was already on anticoagulants and the diagnosis was otherwise confidently established based on presence of other criteria mentioned in the Sydney classification criteria.



Figure-1: Ultrasound KUB.



Figure-2: Non-contrast enhanced CT scan KUB.

antibodies, anti nuclear antibodies, extractable nuclear antibodies, anti-ds DNA antibodies, rheumatoid factor, VDRL and TPHA were negative, while serum CRP, chest X ray and ECG were normal. The patient remained under treatment for the next three months, during which period his renal function tests worsened progressively, rising to serum urea 7.8 mmol/l and creatinine 133 μ mol/l. Repeat testing after three months revealed positive IgG Anti β -2 Glycoprotein I antibodies (58 U/l) and borderline IgM Anti β -2 Glycoprotein I antibodies (6 U/l). Plain CT scan KUB at that time showed a shrunken left kidney measu-

APS involves kidneys in about 25% cases. This is generally due to thrombotic phenomena. Patient may develop renal artery or vein thrombosis. More commonly, they present with APS nephropathy, characterized by acute thrombosis in glomeruli and/or arterioles and chronic vascular lesions such as fibrous intimal hyperplasia of arterioles, organized thrombi with or without recanalization, and fibrous arterial occlusions or focal cortical atrophy⁶. Non-thrombotic complications such as minimal change disease, membranous nephropathy and pauci-immune glomerulonephritis have also been described⁷.

CONCLUSION

Though high altitude predisposes to venous thromboembolism, renal vein thrombosis is rare. Antiphospholipid syndrome is one of the causes of venous thrombosis, and the propensity is increased by exposure to high altitude.

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CONFLICT OF INTEREST

There is no conflict of interest to be declared by any author.

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