PARIPEX - INDIAN JOURNAL OF RESEARCH | Volume-9 | Issue-6 | June - 2020 | PRINT ISSN No. 2250 - 1991 | DOI : 10.36106/paripex

# nalo **ORIGINAL RESEARCH PAPER** Surgery **EXPOSURE TO HIGH ALTITUDE : A RISK** KEY WORDS: Thrombosis, FACTOR FOR SPONTANEOUS RADIAL ARTERY High Altitude, Radial Artery, **THROMBOSIS** Hypercoaguability Lt Col (Dr) Graded Specialist (Surgery), 153 General Hospital, Leh Yogesh Kukreja Col (Dr) Animesh Senior Advisor (Surgery) and Prosthetic Surgeon, 153 General Hospital, Leh. Vatsa\* \*Corresponding Author Maj (Dr) S S Graded Specialist (Radiodiagnosis), 153 General Hospital, Leh. Choudhary Maj (Dr) Zenith Graded Specialist (Surgery), 153 General Hospital, Leh.

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ABSTRACT

Spontaneous vascular thrombosis may occur after a short climb or prolonged stay at high altitude. Upper extremity arterial thrombosis is rare and is more prevalent on the ulnar side of the circulation. Radial artery thrombosis is even rarer and is usually iatrogenic. We present three cases of spontaneous Radial Artery thrombosis in low landers staying in high altitude area.

# INTRODUCTION

Spontaneous vascular thrombosis, both venous and arterial, is reported after a short climb or prolonged stay at high altitude causing significant morbidity and mortality (1,2), Thrombosis in lower limbs is six times more common than upper limbs (3). In upper limb it is frequently seen in the ulnar artery secondary to single or repeated blunt trauma to the hypothenar eminence also known as 'hypothenar hammer syndrome' with relative sparing of the radial artery (4,5). Radial artery thrombosis may arise due to a wide variety of causes including iatrogenic cannulation, emboli, atherosclerosis or blunt and penetrating trauma (6,7,8). Spontaneous Radial artery thrombosis is a rare entity and is not routinely reported. We present three cases of spontaneous Radial Artery thrombosis in low landers staying in High Altitude Area.

## **Case Report**

49 year old male non smoker with no comorbidities in High Altitude Area for 18 months presented with with pain and numbness in left forearm and wrist of 08 days duration. No history of trauma. On examination patient had tenderness in left forearm with no change in local temperature. Radial pulse was not palpable, all other peripheral pulses were palpable. There was no weakness of upper limb. CDFI showed thrombosis is left radial artery (Figure 1). Patient was managed with Low Molecular Weight Heparin and underwent elective thrombectomy. Postoperatively, his symptoms improved.



Figure 1 Left Radial Artery Thrombosis

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49 year old male non smoker with no comorbidities in High Altitude Area for 10months presented with with pain and numbness in left forearm and wrist of 01 month duration. No history of trauma. On examination patient had tenderness in left forearm with no change in local temperature. Radial pulse was not palpable, all other peripheral pulses were palpable. There was no weakness of upper limb. CDFI showed thrombosis is left radial artery (Figure 2). Patient was managed with Low Molecular Weight Heparin.





50 year old male non smoker with no comorbidities in High Altitude Area for 01 month presented with pain and numbness and loss of power in left forearm and wrist of 02 days duration. No history of trauma. On examination patient had tenderness in left forearm with cold extremity. Radial and ulnar pulse were not palpable, all other peripheral pulses were palpable. There was weakness of upper limb. CDFI showed thrombosis is left radial and ulnar artery (Figure 3 & 4). Patient was managed with Low Molecular Weight Heparin and underwent emergency thrombectomy. Postoperatively, his symptoms improved.



Figure 3 : Thrombosis In Left Radial Artery With Patent

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**Right Radial Artery** 



Figure 4: Thrombosis In Left Ulnar Artery With Patent Right Ulnar Artery

All haematological and biochemical parameters in this patient were within normal limits. All these patients were screened for procoagulant state and thrombophilia work up was unremarkable. The cause of thrombosis in these cases could be attributable to their stay in high altitude.

#### DISCUSSION

Thrombosis has a multifactorial etiology with simultaneous interaction of several acquired and genetic risk factors with environmental and behavioral risk factors (9). Various researches have ascertained a number of hematological changes on ascent to High Altitude and even have reproduced them on simulation in hypobaric chambers, these changes varies with the duration of stay (10,11,12). Individuals show initial transient phase of hypercoagulability usually lasting a week. In contrast to individuals with prolonged stay at high altitude late hypercoagulability develops which peaks at 05 months and persists for as long as the individual stays at High Altitude (10).

High altitude exposure or stay has 30 times more risk of having thrombotic events with individuals having presexisting prothrombotic states, such as hereditary thrombophilia, are more predisposed (13). Other suggested mechanisms include dehydration, secondary polycythemia – one of the major cause, hypoxia-induced hemostatic changes such as increased platelet activity, immobility, inflammatory changes secondary to endothelial injury, hyperhomocysteinemia, exercise, and coagulation pathway triggered by hypothermia (14).

Hypercoagulability is demonstrated in high altitude by increased D-dimer levels, prothrombin time, and activated protein C resistance (15).

Suggested full hematological workup in these cases includes hemoglobin, packed cell volume, prothrombin time, activated partial thromboplastin time. Fibrinogen, d-Dimers, lipid profile, homocysteine levels, antiphospholipid antibodies, lupus anticoagulants, anticardiolipin antibodies, coagulation factors (II, V, VIII, IX, X), protein C, protein S, antithrombin, factor V Leiden mutation, plasminogen activator inhibitor-I, platelet activation factors.(16,17,18,19). A limited procoagulant work up was done in these cases which was unremarkable and has a high probability of being attributable to stay in High Altitude.

#### CONCLUSION

Thrombosis at High Altitude is a reality with variable manifestations and affects the recreational climber with short stay as well be an occupational hazard for those deployed there with prolonged stay. Experimental evidence is limited and trials to prove thrombogenecity technically challenging. A greater understanding and awareness of its varied presentations and management is required by all those engaged in treating such patients.

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