CRITICAL LIMB ISCHEMIA; A SEQUEL OF VITAMIN B-12 DEFICIENCY ANEMIA ENDING UP IN AMPUTATION

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ABSTRACT

A 35-year-old serving military soldier serving in Siachin area reported for provision of prostheses. He had a left transfemoral amputation due to critical limb ischemia (CLI). On evaluation, he had severe anemia with impaired position sense and proprioception and was unable to use the prosthesis. On further exploration, he was found to be deficient in vitamin B12 that had presumably predisposed him to develop CLI. After indoor intravenous vitamin B12 administration along with high protein and calorie diet, he improved remarkably and started using the prosthesis provided to him. He was discharged on oral mecobalamin therapy and is now on regular follow up with the internist and hematologist. Before exposure to extreme cold weather, military personnel must undergo screening for anemia in addition to other pertinent medical examination to prevent complications.

INTRODUCTION

Critical limb ischemia (CLI) is described as a reduction in tissue perfusion to an extent that surpasses the limit of the obligatory oxygen delivery as well as the compensatory mechanisms such as vasodilation and collateral circulation[1]. Predisposing factors include advanced age, smoking, diabetes, dyslipidemia, hypertension, obesity and cold exposure[2,3]. Exposure to cold may cause inadequate blood circulation, endothelial injury, local thrombosis and hypoxia when the ambient temperature is below freezing point[4]. Augmenting factors in this scenario are tight clothing or boots, cramped positions, fatigue, certain medications, smoking, alcohol use and diabetes mellitus[4,5].

Megaloblastic anemia is defined as an anemic state characterized by the presence of abnormally large red blood cells in the peripheral blood. The spectrum of etiologies associated with macrocytic anemia includes nutritional deficiencies (e.g. vitamin B12 and folate), drugs, primary bone marrow disorders and other chronic illnesses[6]. Past regional medical literature has not reported development of CLI in a patient of vitamin B12 deficiency anemia to the best of our knowledge.

Among patients with CLI 25% die and 30% undergo amputation by one year of onset[5]. Amputation is preventable if impending CLI is timely identified and managed appropriately[7]. We present here, report of a military soldier with vitamin B12 deficiency anemia who developed CLI on exposure to cold weather in Siachin eventually resulting in transfemoral amputation on the left side.

CASE REPORT

A 35 year old serving military soldier presented to the amputee indoor rehabilitation department of armed forces institute of rehabilitation medicine (AFIRM) eight months after a unilateral transfemoral amputation for provision of prosthesis. Patient was performing his duties at Siachin area where he developed discoloration of his left foot along with pain. Over the next five days, the problem progressively intensified involving left leg upto the knee joint. Due to inclement weather conditions patient was evacuated on eighth day to combined military hospital Rawalpindi where

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he was diagnosed to have developed irreversible CLI and his transfemoral amputation was carried out on an urgent basis. During pre-operative evaluation, he was found to have severe anemia with a hemoglobin level of 6 g/dL. The mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) were increased (116 fL and 40 pg respectively) (Normal: 80-100 fL and 26–34 pg respectively) with a mean corpuscular hemoglobin concentration (MCHC) within the reference range. During operation, he was transfused with four pint of whole blood, but his red cell morphology was overlooked and not further investigated.

The patient had history of poor dietary habits with little consumption of meat, poultary or dairy products. Patient was non-smoker. He reported using tight clothing and resting in awkward positions. He disclosed that he had a history of severe anemia in 2005 when his hemoglobin was found to be 6 g/dL. He was investigated thoroughly at that time and given some intravenous vitamin B12. He continued taking oral vitamin B12 for next seven years until he was posted to the Siachin. Before ascend, his medical evaluation had revealed a hemoglobin level of 11 g/dL with normal red cell morphology. His family history was negative for any cardiovascular event or peripheral vascular disease.

On physical examination at AFIRM, he was pale and had a pulse rate of 100 beats per minute. He had a transfemoral amputation on left side (figure-1) and had poor proprioception and vibration sensations. His hemoglobin was 7.2 g/dL and total leucocyte count was 2.7 x 109/L (Normal: 4 – 11 x 109/L). The platelet count was normal. The MCV and MCH were increased (120fL and 41pg respectively) with a MCHC within the reference range. The reticulocyte count was raised i.e. 2.3%(Normal: 0.5–1.5%). The serum lactate dehydrogenase level was markedly raised to 2309 units per liter (U/L) (Normal: 140-280 U/L). The serum cobalamin level was low i.e. 100 pg/mL (normal: 200 - 900 pg/mL) while serum folic acid and ferritin levels were within standard parameters. His thyroid profile was also normal. Serum protein electrophoresis revealed normal albumin with diffuse staining in the gamma region. Bence Jones proteins were not detected in the urine and the urinary protein electrophoresis was normal. Ultrasound of the abdomen did not reveal any abnormality. Serum anti tissue transglutaminase antibodies were absent and bone marrow trephine biopsy suggested megaloblastic anemia.

After diagnosing vitamin B12 deficiency, 1000 µg of vitamin B12 was started intravenously on alternate day basis. High protein and calorie diet was provided to the patient. His hemoglobin levels rose to 10 g/dL in about four weeks. For limb amputation, he was provided a modular transfemoral prosthesis with polycentric knee joint and a multi-axial foot. (Figure-2) The patient was discharged on oral mecobalamin therapy in a dose of 500µg thrice daily and he is now on regular follow up with the internist and hematologist.

DISCUSSION

CLI presents with resting leg or foot pain and breakdown of the skin of the leg or foot resulting from disease of the peripheral arteries. Approximately 500-1000 individuals in a million people develop CLI each year[6,8] . Gangrene and sepsis are fatal complications if not treated promptly[9].

Exposure to cold indirectly potentiates development of CLI by producing circulatory stasis and vaso-constriction. At high altitude, in anemic patients a thinner oxygen column makes the circumstances even graver.

The presentation of CLI is dominated by severe pain that typically occurs at night in usually the distal part of the foot and is relieved by hanging the foot by the bedside or sleeping upright in a chair[9]. For diagnosis ankle-brachial indices, segmental limb pressures and wave form analysis and transcutaneous oxygen and carbon dioxide saturations should be a part of routine initial evaluation[9].

The management of CLI requires a multi disciplinary team comprising of experts in vascular imaging, atherosclerosis, wound care and physical therapy. Therapeutic goals in these patients is getting rid of pain, healing ulcers, avoiding major amputation, reducing cardiovascular risk factors, enhancing quality of life and prolonging survival[10]. These goals may be achieved through medical therapy, revascularization or amputation. Medical therapy involves adequate analgesia, pressure relief, treatment of infection, local care of wounds and modification of atherosclerotic risk factors[10]. Patients with non-ambulatory status, severe comorbidities, lesions not responsive to revascularization, extensive tissue necrosis and life-threatening infections are potential candidates for primary amputation[11,12]. Intermittent pneumatic compression and spinal cord stimulation are alternative choices for those who are either unwilling or unable to undergo amputation and may stimulate wound healing and provide symptom relief[8].

Either revascularizations or amputation is chosen for treating CLI; good nutrition and goal-oriented rehabilitation are needed for maximal independence. Close monitoring for coronary artery and cerebrovascular disease is generally required life-long [13].

CONCLUSION

CLI is a deleterious outcome of many factors including cold and anemia. Before exposure to extreme cold weather, military personnel must undergo screening for anemia in addition to other pertinent medical examination to prevent complications.

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

REFERENCES

1. Dawson DL, Mills JL. Critical limb ischemia. Curr Treat Options Cardiovasc Med 2007; 9(2): 159-70.
2. Gordon T, Kannel WB. Predisposition to atherosclerosis in the head, heart, and legs. The Framingham study. JAMA 1972; 221(7): 661-6.
3. Brown AL, Juergens JL. Arteriosclerosis and atherosclerosis. In: Allen EV, et al. Peripheral vascular disease. Philadelphia: Saunders, 1972; 57–66.
4. Harirchi I, Arvin A, Vash JH, Zafarmand V. Frostbite: incidence and predisposing factors in mountaineers. Br J Sports Med 2005; 39(12): 898-901;discussion 901.
5. Reamy BV. Frostbite: review and current concepts. J Am Board FamPract 1998; 11(1): 34-40.
6. .Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FGR, on behalf of the TASC II Working Group. Inter- society consensus for the management of peripheral arterial disease (TASC II). J VascSurg 2007; 45(1 Suppl): S5-S67
7. Feiring AJ, Krahn M, Nelson L, Wesolowski A, Eastwood D, Szabo A. Preventing leg amputations in critical limb ischemia with below-the-knee drug-eluting stents: the PaRADISE (PReventing Amputations using Drug eluting StEnts) trial. J Am CollCardiol 2010; 55(15): 1580-9.
8. Dormandy JA, Rutherford RB. Management of peripheral arterial disease (PAD). TASC Working Group. TransAtlantic Inter-Society Consensus (TASC). J VascSurg 2000; 31(1 Pt 2): S1–S296.
9. Lumsden AB, Davies MG, Peden EK. Medical and endovascular management of critical limb ischemia. J EndovascTher 2009; 16(2 Suppl 2): II31-62.
10. Slovut DP, Sullivan TM. Critical limb ischemia: medical and surgical management. Vasc Med 2008; 13(3): 281-91.
11. Varu VN, Hogg ME, Kibbe MR. Critical limb ischemia. J VascSurg 2010; 51(1): 230-41.
12. Santilli JD, Santilli SM. Chronic critical limb ischemia: diagnosis, treatment and prognosis. Am Fam Physician 1999; 59(7): 1899-908.
13. Taylor LM Jr, Edwards JM, Porter JM. Present status of reversed vein bypass grafting: five-year results of a modern series. J VascSurg 1990; 11(2): 193-205; discussion 205-6.