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Acute limb ischaemia as an initial presentation of *Streptococcus viridans* infective endocarditis: a case report

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Abstract

Streptococcus viridans, a group of alpha-hemolytic streptococci, is a part of the normal oral flora typically linked to dental caries, pericoronitis and subacute infective endocarditis affecting native heart valves. We present a case of subacute infective endocarditis caused by *Streptococcus viridans* in a healthy man presenting with aortic valve involvement, large vegetation, septic embolism and acute limb ischaemia. Though acute limb ischemia is not a common initial presentation, it is crucial to consider infective endocarditis in the differential diagnosis when it is associated with a febrile illness, since delayed diagnosis leads to catastrophic complications.

Key words: acute limb ischemia, infective endocarditis, *Streptococcus viridans*, septic embolism

Introduction

Despite advancements in diagnostic and treatment modalities, infective endocarditis (IE) remains a significant cause of morbidity and mortality at an incidence of 1-7/100,000 people per-year.[1] Septic embolism is a well-known complication and can be life threatening if IE not detected and treated in timely manner. Systemic embolism occurs in 22%-50% of patients, predominantly affecting the central nervous system.[2] Peripheral arterial embolism involving the limbs can progress to acute limb ischaemia potentially causing limb loss. Primary risk factors for systemic embolism are large, floppy vegetations in the left heart with the peak risk at two to three weeks after the onset of endocarditis.[2,4] Early embolectomy is an option to relieve the occlusion and to prevent formation of infected aneurysms.[3]

Case presentation

A 20-year-old previously healthy university student presented with sudden onset pain and numbness of left lower limb for two hours. He was having high- grade intermittent fever for a period of eight days with constitutional symptoms such as headache, generalized weakness, fatigue, and loss of appetite. Two hours prior to admission he developed acute onset burning type pain in the left lower limb which was progressively getting worse and exacerbated by activities and bearing weight on left leg. It was accompanied by numbness and restricted movements of the left foot and loss of sensation below the left knee. There was no history of trauma to leg, any previous thrombotic disease or long period of immobilization. He was a teetotaler and denied any high-risk sexual behavior or illicit drug abuse.

On Examination, body temperature was 37.3°C and there were no peripheral stigmata of infective endocarditis. The pulse rate was 86 beats per minute, regular and collapsing in nature. Blood pressure was 110/50 mm Hg, respiratory rate was 15/minute and oxygen saturation on room air was 98%. Precordial examination revealed a grade three early diastolic murmur at left sternal edge with a non-displaced, thrusting apex. There were no signs of acute heart failure or pulmonary hypertension. The left lower limb

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was mottled and cold to touch below the knee and there was loss of tactile sensation. The left femoral pulse was present, but the left popliteal, posterior tibial and dorsalis pedis pulses were absent. He was able to flex and extend his left knee but was unable to move the left foot. The examination of the right lower limb was normal. Results of the initial investigations are shown in Table 1.

Full blood count revealed neutrophil leukocytosis and microcytic hypochromic anemia. Acute kidney injury and raised serum inflammatory markers were observed (Table 1). Screening tests for retroviral, syphilis (VDRL), viral hepatitis (HBs Ag and Hepatitis-c Ab) and autoimmune diseases (ANA) were negative. Transthoracic-echocardiogram (TTE) revealed a large aortic valve vegetation (19mmx18mm) and severe aortic regurgitation with a left ventricular ejection-fraction of 60% (Figure 1).

An urgent CT-arteriogram of lower limbs revealed a complete occlusion of the left distal common femoral, proximal superficial and deep femoral arteries and absence of distal flow. Based on these findings, a diagnosis of infective endocarditis of aortic valve with acute left lower limb ischemia secondary to septic embolization was made. Intravenous ampicillin, ceftriaxone and gentamicin were started empirically after obtaining blood cultures. He underwent an emergency embolectomy and emboli was sent for microbiological confirmation and histology.

Table 1. Results of initial investigations

Investigations	*Results*	*Reference range*
White blood cell	17.6×10^9/l	4-10
Neutrophil	76.8%	50-70
Lymphocyte	17.2%	20-40
Hemoglobin	8.8g/dl	11-15
Mean corpuscular volume	79fl	80-100
Mean hemoglobin concentration	25pg	27-34
Red cell distribution width	14%	11-16
Platelets	355×10^9/l	150-450
Erythrocyte sedimentation rate	96 mm/hour	<20
C-reactive protein	148mg/l	0-3
Serum creatinine	157micromol/l	49-90
Serum potassium	5.4mmol/l	3.5-5.1
Serum sodium	140mmol/l	136-145
Alanine transaminase	16 IU/L	16-63
Aspartate transaminase	25 IU/L	15-37
Urine full report	Trace proteinuria RBC- 30-35/hpf WBC- 3-5/hpf	

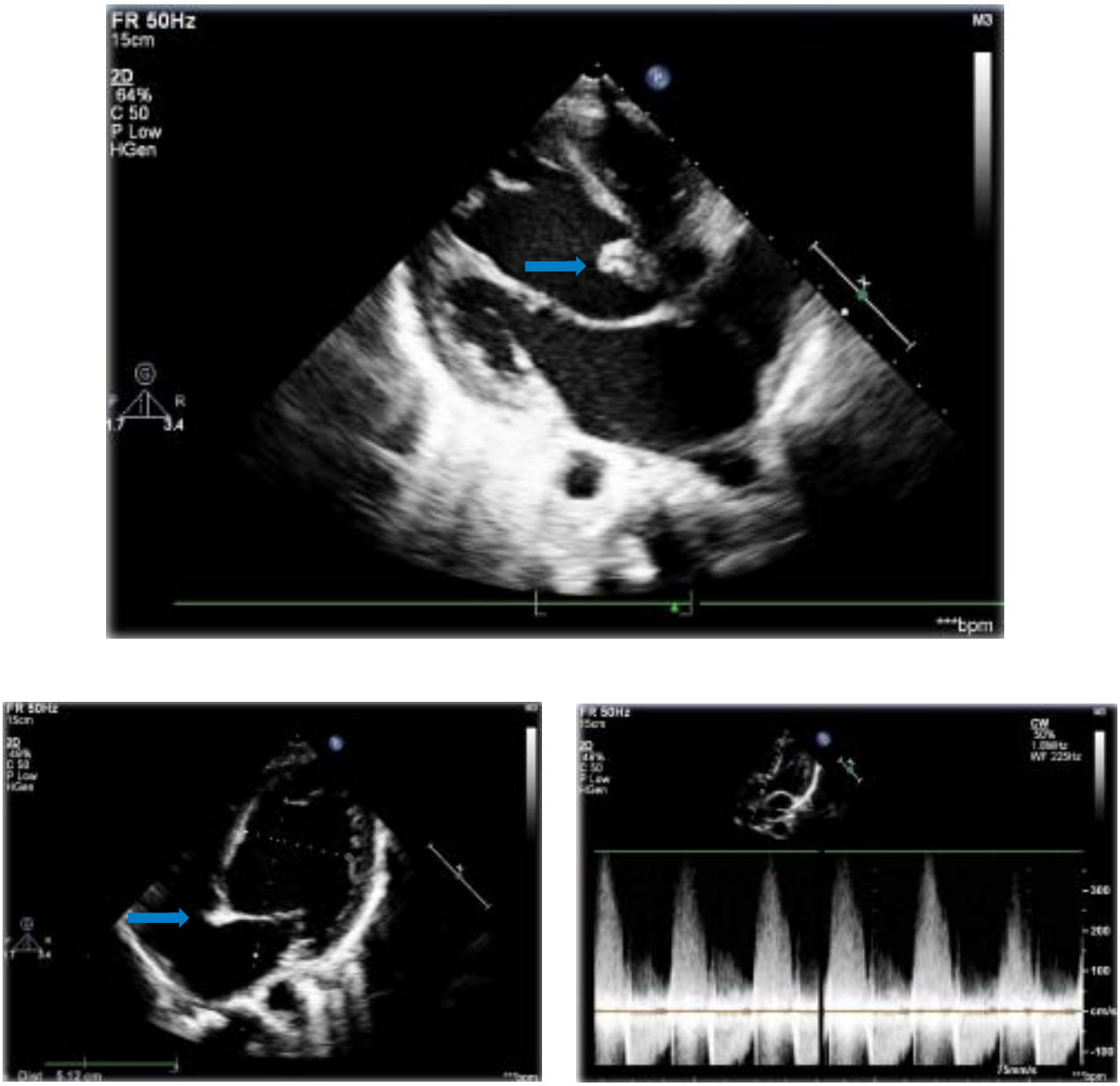

Figure 1. Transthoracic echocardiography (blue arrow heads denote the vegetation of aortic valve).

Post-operatively, good volume dorsalis pedis and posterior tibial pulses, and improved motor and sensory functions of the left leg were noted. Anticoagulation was initiated with enoxaparin. All three blood cultures were negative, embolic tissue culture isolated growth of *Streptococcus viridans*. On day 10 of admission, he underwent excision of vegetation along with infected valve and aortic valve replacement with 21mm On-X mechanical prosthetic valve. Histology revealed evidence of endocarditis, degenerative myxoid changes with fibrinoid necrosis. 2D-echocardiography two weeks after surgery revealed well-functioning prosthetic aortic valve with para-valvular leak, no obvious vegetation. Later, warfarin was initiated and bridged with enoxaparin to achieve target INR of 2.0 to 3.0. After 26 days of hospital stay, he was transferred to a local health care center to complete six weeks course of antibiotics and titration of warfarin. At follow-up visit after two weeks, he was able to walk normally, has gained full function of left leg and surgical wounds healed completely.

Discussion

The predominant aetiological agents of IE are staphylococci (especially in acute IE) and streptococci (commonly subacute IE). Among streptococci, *Streptococcus viridans* is the most frequent organism associated with subacute bacterial native valve endocarditis (NVE) and late prosthetic valve endocarditis (PVE).[4] Cardiac murmurs are detected in 85% of cases, with new murmurs occurring in 48% of IE.[5] Echocardiographic findings include vegetations, abscess formation, new dehiscence of a prosthetic valve, or newly observed valvular regurgitation which are considered to be major diagnostic criteria. Endocarditis caused by *Streptococcus viridans* is associated with larger vegetations, greater valve destruction, more frequent embolic events, and a higher mortality rate compared to other forms of streptococcal infective endocarditis. Vascular complications in left sided endocarditis are associated with peripheral embolism, cerebral infarctions being the most frequent. Thromboembolism of the extremity arteries are uncommon, with an incidence of 4%-5% in those with native valve endocarditis.[6,7] Acute coronary syndrome associated with IE due to extrinsic compression of coronary arteries by vegetations are being reported rather than embolic phenomena.[8,9] Our patient did not have any symptoms or electro-cardiographic changes of cardiac ischaemia.

The general approach to treatment involves initial stabilization, prompt collection of blood cultures, and definitive medical and/or surgical intervention. Embolectomy and systemic antibiotic administration followed by replacement of damaged valve are the mainstays of treatment. The mortality rate was more than 80% in the past,[10] but reduced to 25-30% with the current treatment approaches.[11] The choice of antibiotic depends on whether the affected valve is native or prosthetic, as well as the causative microorganism and its antibiotic susceptibilities. After a definitive diagnosis of endocarditis in our patient, a course of empirical antibiotic therapy was started with ampicillin, ceftriaxone and gentamicin which was later changed to penicillin G and ceftriaxone as culture-guided antibiotics. Surgical intervention is indicated for most patients with PVE, *Staphylococcus aureus* endo-carditis, fungal endocarditis, and endocarditis associated with large vegetations (≥10mm). Early surgical intervention is recommended for patients with >15mm vegetations as in our patient, and high mobility of the vegetations irrespective of the degree of valve destruction. Presence of heart failure and/or poor response to antibiotics[1] are other indications. Presence of an invasive pathogen, extensive destructive vegetation, left-sided valve involvement, large septic embolus and acute lower limb ischaemia were poor prognostic factors in our patient. Paravalvular abscess, peri-annular extension of infection, PVE, septic shock, advanced age and comorbidities like diabetes, hypertension, heart failure, renal failure are the other poor prognostic factors.

Conclusions

Acute limb ischaemia is a rare vascular medical emergency encountered in clinical practice. IE is a well-known cause for acute limb ischaemia and should be suspected when associated with a prolonged febrile illness. Clinical examination of the heart should be an integral part of assessment even though often overlooked in the emergency care of limb ischaemia. Timely interventions and medical treatment play a vital role in managing embolic and valvular complications of infective endocarditis.

Author declaration

Consent

Written informed consent for publication was obtained from the patient.

Conflict of interests

The authors have declared that no competing interests exist.

Author contribution

Concept, design, acquisition, analysis, inter-pretation, critical review and supervision by AP, NS, TK, SG and BT. All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

References

1. Sabe MA, Shrestha NK, Menon V. Contemporary drug treatment of infective endocarditis. *Am J Cardiovasc Drugs.* 2013; **13**(4): 251-8. doi:10.1007/s40256-013-0015-6
2. Mocchegiani R, Nataloni M. Complications of infective endocarditis. *Cardiovasc Haematol Disord Drug Targets.* 2009; **9**(4): 240-8. doi:10.2174/1871529x10909040240
3. Nakayama DK, O'Neill Jr JA, Wagner H, et al. Management of vascular complications of bacterial endocarditis. *J Pediatr Surg.* 1986; **21**(7): 636-9. doi: 10.1016/s0022-3468 (86)80421-3
4. De Gennes C, Souilhem J, Chapelon C, et al. Arterial embolism of the limbs in infectious endocarditis of the heart valves. *Presse Medicale*. 1990; **19**(25): 1177-81. PMID: 2142282
5. Gould FK, Denning DW, Elliott TS, et al. Guidelines for the diagnosis and antibiotic treatment of endocarditis in adults:

a report of the Working Party of the British Society for Antimicrobial Chemotherapy. *J Antimicrob Chemother.* 2012; **67**(2): 269-89. doi: 10.1093/jac/dkr450.

6. Galyfos G, Giannakakis S, Kerasidis S, et al. Infective endocarditis as a rare cause for acute limb ischemia. World *J Emerg Med.* 2016; **7**(3): 231. doi:10.5847/wjem.j.1920-8642.2016.03.012
7. Uglov AI, Diuzhikov AA. Surgery of arterial embolism in patients with infective endocarditis. *Angiol Sosud Khir.* 2004; **10**(3): 97-103. PMID: 15622400.
8. Ngonge AL, Amah C, Udongwo N, et al. Infective endocarditis presenting as acute coronary syndrome. *Chest.* 2024; **166**(4): A542-3. doi: 10.1016/j.chest.2024.06.376
9. Manzano MC, Vilacosta I, San Román JA, et al. Acute coronary syndrome in infective endocarditis. *Cardiologia.* 2007; **60**(1): 24-31.doi:10.1016/S1885-5857(07)60102-9
10. Lozano P, Flores D, Blanes I, et al. Acute lower limb ischemia complicating endocarditis due to Candida parapsilosis in a drug abuser. *Annals of Vascular Surgery.* 1994; **8**: 591-4.
11. Singh SP, Singh D. Infective endocarditis: A case series. *Journal of Cardiac Critical Care.* 2020; 4(2):156-60. doi:10.1055/s-0040-1716610