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Case Report

A curious case of upper limb ischemia associated with deep vein thrombosis following cardioversion for ventricular tachycardia

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ABSTRACT

Acute upper limb ischaemia is an uncommon vascular emergency that may result in irreversible limb loss. Thromboembolic complications following cardioversion for ventricular tachycardia remain poorly characterised. We report the case of a 70-year-old woman with type 2 diabetes mellitus who underwent direct-current cardioversion for haemodynamically unstable ventricular tachycardia. Subsequently, she developed acute pain, numbness, and bluish discolouration of the left upper limb, with absent brachial and radial pulses and complete loss of distal motor and sensory function. Duplex ultrasonography demonstrated absent flow in the brachial, radial, and ulnar arteries. Echocardiography revealed global left ventricular hypokinesia with an ejection fraction of 30%, without intracardiac thrombus. Concurrent right lower limb deep vein thrombosis was identified. The limb was classified as Rutherford class III acute limb ischaemia, necessitating above-elbow amputation. This case highlights a severe vascular complication temporally associated with cardioversion for ventricular tachycardia, emphasising early recognition, prompt vascular assessment, and timely intervention to prevent irreversible ischaemic injury.

Keywords: Acute upper limb ischemia, Ventricular tachycardia, Cardioversion, Deep vein thrombosis, Cardioembolism

INTRODUCTION

Peripheral vascular diseases are a group of disorders that primarily affect arterial blood flow in the peripheral regions, due to a variety of causes. Of this, acute upper limb ischemia (ALI) is a significant vascular emergency that arises in patients with structural heart disease and arrhythmias, often resulting in morbidity and lifelong disability.[1,2]

The resulting ischemia is usually a consequence of thromboembolism that arises in the setting of the above said defect or abnormal electrophysiological rhythm. In patients with atrial fibrillation, cardioversion may be followed by thromboembolic complications, including stroke, particularly when risk factors for thrombus formation are present.[3] However, the evidence regarding thromboembolic risk following cardioversion for ventricular tachycardia remains extremely limited.

In this light, upper limb involvement of ALI (known as acute upper limb ischemia or AULI) is an exceedingly rare entity due to the number of factors that promote the lodging and thereby occlusion of the vessel in areas such as the lower limbs; upper limb cases account for only 17% of all ALIs, them being rare entities in themselves due to improvement in treatment protocols.[1]

The rarity of this condition is highlighted in the fact that diagnostic delays are more common in resource limited settings where advanced vascular interventions are not feasible, underscoring the importance in recognising this

condition. With this, we describe a case of a left upper limb claudication after cardioversion for ventricular tachycardia.

CASE REPORT

A 70-year-old woman with a known history of type 2 diabetes mellitus was referred to our hospital with pain and numbness in the left upper limb. She initially presented to the referring hospital with chest pain, giddiness, and breathlessness. Electrocardiography revealed ventricular tachycardia associated with unrecordable blood pressure, for which two cycles of direct current cardioversion were performed, successfully restoring sinus rhythm (Figure 1). She was subsequently initiated on inotropic support. Later that day, she developed pain in the left forearm, followed by bluish discoloration of the limb. Local examination revealed absent brachial and radial pulses, with loss of movements and sensation below the elbow. Anticoagulation was initiated, and she was referred to our hospital for further management. The echocardiographic findings before cardioversion at the referring hospital were unavailable.

On arrival, the patient was conscious and afebrile, with a regular pulse rate of 102 beats/min and a blood pressure of 100/60 mmHg while receiving inotropic support. Her oxygen saturation was 98% on supplemental oxygen at 10 L/min. Examination of the left upper limb revealed bluish discoloration and relative coldness below the elbow, absent brachial and radial pulses, complete loss of movements and sensation distal to the elbow, and wrist drop (Figure 2). Cardiovascular examination revealed a regular rhythm without murmurs, while bilateral crepitations were heard on respiratory examination.

Initial investigations revealed neutrophilic leukocytosis, mild anaemia, elevated blood urea, and markedly elevated liver transaminases. Electrocardiography demonstrated poor R-wave progression, while transthoracic echocardiography revealed global left ventricular hypokinesia with an ejection fraction of 30%, without intracardiac thrombus. Doppler ultrasonography of the left upper limb demonstrated absent flow in the brachial, radial, and ulnar arteries, while bilateral lower limb Doppler revealed deep vein thrombosis involving the right common femoral, saphenofemoral, and popliteal veins. Chest radiography showed cardiomegaly with increased bronchovascular markings. The laboratory investigation results are summarised in Table 1.

The patient was initially managed with broad-spectrum intravenous antibiotics, intravenous heparin at a dose of 5,000 units four times daily, and noradrenaline support for haemodynamic instability. Vascular surgery consultation was obtained, and the left upper limb was classified as Rutherford class III acute limb ischaemia, indicating irreversible ischaemic injury. In view of the extensive vascular compromise and the absence of distal motor and sensory function, left above-elbow amputation was planned. The procedure was performed and was uneventful.

Table 1: Summary of laboratory investigations.

Parameters	Values	Reference range/ impression
Total leukocyte count (10^3/µl)	17.3	4.0-11.0
Hemoglobin	10	12.0-16.0
Platelet count (10^3/µl)	230	150-450
Urea (mg/dl)	65	15-40
Creatinine (mg/dl)	1.0	0.6-1.1
Sodium (mmol/l)	136	135-145
Potassium (mmol/l)	4.2	3.5-5.1
Chloride (mmol/l)	97	98-107
Calcium (mg/dl)	9.1	8.5-10.5
Magnesium (mg/dl)	1.9	1.7-2.4
PT (seconds)	16.0	11-13.5
APTT (seconds)	20.9	25-35
INR	1.3	0.8-1.2
Troponin I	Negative	

PT – prothrombin time, APTT – activated partial thromboplastin time, INR – international normalized ratio

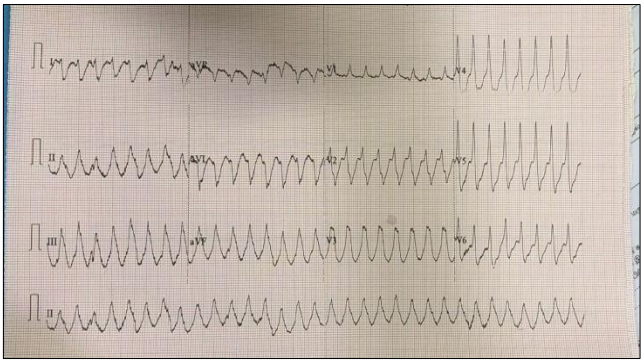


Figure 1: The electrocardiograph in the above image taken at the peripheral hospital prior to presentation, demonstrates absent P waves and broad based regular QRS complexes suggestive of monomorphic ventricular tachycardia.



Figure 2: The left upper limb of the patient, with a noticeable bluish discolouration of the limb distal to the elbow joint.

Postoperatively, the patient continued to receive inotropic support and intravenous heparin, followed by bridging to oral warfarin. The target international normalised ratio (INR) of 2–3 was achieved. Dual antiplatelet therapy with aspirin and clopidogrel was also initiated. During the subsequent hospital stay, the patient demonstrated clinical improvement, with her heart rate stabilising at approximately 80 beats/min, and was successfully weaned off supplemental oxygen. She was discharged on oral warfarin, aspirin, clopidogrel, and heart failure medications and advised for repeated follow up.

DISCUSSION

AULI is an uncommon but potentially devastating vascular emergency that requires prompt recognition and appropriate intervention. This case is notable for the sudden onset of severe left upper limb ischaemia following direct-current cardioversion for haemodynamically unstable ventricular tachycardia (VT), which is an exceedingly rare arrhythmia that this condition is followed by. Although thromboembolism is a major cause of AULI, the temporal relationship with cardioversion, in the presence of severe left ventricular dysfunction, raises important questions regarding the underlying mechanism and the challenges of preventing irreversible limb injury that may result in amputation.

Cardioembolism is an important cause of AULI, particularly in patients with structural heart disease and cardiac arrhythmias. James et al demonstrated that embolic occlusion accounted for 47.2% of cases in their series of 36 patients with acute upper limb ischaemia, although primary arterial thrombosis and iatrogenic injury were also identified.[4] Similarly, a systematic review reported that 92% of 1,326 upper limb arterial occlusions were attributable to thromboembolic disease.[2] These findings support the importance of considering an embolic mechanism in patients presenting with sudden limb pain, discoloration, absent pulses, and neurological deficits. However, unlike the predominantly atrial fibrillation-associated cases described in the literature, our patient developed ischaemia following cardioversion for VT, making the underlying mechanism less straightforward.

Electrical cardioversion can be associated with systemic embolisation when pre-existing intracardiac thrombi are present, although the thromboembolic risk is best established in atrial fibrillation and cannot be directly extrapolated to VT.[3] A recent case report by Alkoheji et al described coronary artery embolism and acute myocardial infarction following cardioversion for VT, demonstrating that embolic complications may occur in this clinical setting.[5] Although these reports provide clinically relevant parallels, they do not establish that cardioversion directly caused the ischaemia in our patient. The absence of an intracardiac thrombus on subsequent transthoracic echocardiography does not exclude a transient embolic source, while the unavailability of pre-cardioversion echocardiographic findings limits further interpretation. Therefore, a cardioembolic event remains plausible but unconfirmed.

Another noteworthy finding was the coexistence of right lower limb deep vein thrombosis (DVT). Although venous thrombosis does not ordinarily cause arterial occlusion, concurrent venous and arterial thromboses raise the possibility of an underlying prothrombotic state or, less commonly, paradoxical embolisation through a right-to-left shunt. However, no such shunt was documented, and the available findings do not establish a common mechanism. Severe left ventricular dysfunction, diabetes mellitus, and acute physiological stress may have contributed to the thrombotic risk, but their individual roles remain uncertain.

The patient had a presentation that supported the classification of Rutherford class III acute limb ischaemia.[6] The European society for vascular surgery guidelines recommend primary amputation when the limb is irreversibly ischaemic, as revascularisation is unlikely to restore meaningful function and may expose the patient to complications associated with reperfusion injury.[7] Accordingly, the decision to proceed with above-elbow amputation was consistent with established current management principles.

In contrast, Wong et al reviewed nonoperative management of acute upper limb ischaemia and highlighted the limitations of the available evidence for conservative treatment, including concerns regarding persistent symptoms and functional outcomes.[2] Another study reported immediate reperfusion following brachial embolectomy in 16 patients with acute upper limb ischaemia, with no limb loss or residual limb disability among the survivors.[8] In more recent studies, El-Sayed et al reported a 2.1% amputation rate among 96 patients undergoing surgical revascularisation, although substantial perioperative complications were observed.[9] Together, these studies emphasise that limb salvage remains achievable when revascularisation is undertaken before irreversible neuromuscular injury develops. In our patient, the advanced neurological deficit at assessment substantially limited the possibility of functional recovery.

Duplex ultrasonography established the absence of flow in the brachial, radial, and ulnar arteries, facilitating prompt clinical assessment in our case. Similarly, Huang et al. demonstrated the utility of point-of-care ultrasonography in rapidly identifying acute brachial artery occlusion, facilitating expedited vascular consultation and successful embolectomy.[10] Although computed tomography angiography provides valuable anatomical information, imaging should not delay definitive treatment when irreversible ischaemia is clinically evident.[7] Following surgery, anticoagulation was continued, particularly given the concurrent DVT. Nevertheless, the indication for combining warfarin with aspirin and clopidogrel warrants careful reassessment because triple antithrombotic therapy

increases bleeding risk and is not routinely indicated solely for acute limb ischaemia.[11]

The strengths of the case lie in its rarity and the noteworthy complication that may result from such a common setting; a detailed review of literature yielded possibly less than five documented cases in literature with these exact sequelae. Caution must be taken by the physician to avoid such untoward consequences that, in spite of being non-fatal in this case, resulted in the loss of a limb and thus affects the daily life of the patient. Further research may establish the exact mechanistic causes and improve our understanding to avoid future complications as such.

CONCLUSION

This case highlights acute upper limb ischaemia following direct-current cardioversion for haemodynamically unstable ventricular tachycardia, culminating in above-elbow amputation. The coexistence of severe left ventricular dysfunction and deep vein thrombosis raises the possibility of an underlying thromboembolic or prothrombotic mechanism, although a direct causal relationship with cardioversion remains unproven.

Early recognition of acute limb ischaemia, prompt vascular assessment, and timely intervention are essential to optimise limb salvage. This case emphasises the importance of vigilant peripheral vascular monitoring following cardioversion and highlights the need for further research into thromboembolic complications associated with cardioversion for ventricular arrhythmias.

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