Type A aortic dissection into brachial artery causing upper limb ischaemia: A case report

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**Abstract**

INTRODUCTION: Acute type A aortic dissection (AAAD) is a surgical emergency with high operative mortality. Distal propagation of the dissecting flap can lead to malperfusion of territory supplied by the aorta including axillary and brachial arteries causing ischaemia of the upper limb.

CASE PRESENTATION: We present a case of a 67 year old gentleman who had AAAD and developed upper limb malperfusion after repair. Despite adequate repair, the residual dissecting flap propagated distally in the upper arm vasculature causing thrombosis of the brachial artery. The patient subsequently underwent brachial artery cut-down and embolectomy but revascularization was not achieved. He ultimately required an above-elbow amputation.

CONCLUSION: Upper limb ischaemia from AAAD is a rare phenomenon that is mainly due to malperfusion. Majority of malperfusion resolve after aortic dissection repair. This is an unusual case of persistent upper limb ischaemia despite adequate repair due to the direct extension of the residual dissection flap from the aortic root into the brachial artery.

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1. Introduction

Aortic dissection occurs when an intimalmedial tear allows blood flow to enter the aortic wall; thereby creating a new secondary false lumen that may propagate proximally or distally in a spiraled or straight manner. The true lumen may become compressed by the pressurized false lumen to the point of collapse that can lead to ischemic complications such as malperfusion. The presence of malperfusion, and other factors such as prompt treatment, medical comorbidities, extent of aortic repair, have been found to affect overall survival. Limb and other end-organ ischemia affect 15%–40% of patients [1,2]. Majority of cases of aortic dissection with malperfusion can be treated with urgent resection of the area of primary intimal tear to achieve occlusion of the false lumen. Persistent malperfusion after aortic repair can be treated with revascularization techniques. This is an unusual case study depicting a case of progressive static malperfusion from a dissecting flap despite adequate repair and attempted revascularization. The case has been reported in line with the SCARE criteria [3].

2. Case report

A 67-year-old man with a past medical history of hypertension, ischaemic heart disease, end-stage renal failure, and bleeding gastrointestinal tract from helicobacter pylori gastritis was presented to the emergency department with severe central stabbing chest pain that radiated to his back. Family and social history were non-contributory and he was found to be non-compliant to his anti-hypertensive medications. His physical examination was significant for haemodynamic instability. Neurological, peripheral vascular and abdominal examination were unremarkable. Electrocardiogram revealed deep biphasic T wave inversion over anterol leads with Wellen’s syndrome features. Cardiac enzymes level was normal. Urgent cardiac catheterization was performed which revealed a tight calcified lesion (90–99% critical stenosis) in the mid left anterior descending artery (LAD). He was referred to cardiothoracic surgery for consideration of coronary artery bypass grafting (CABG) for single vessel disease. Further investigations with echocardiogram and computed tomography (CT) aortogram confirmed the presence of a Stanford acute type A aortic dissection (AAAD) involving the aortic root with extension into the aortic arch vessels including the right brachiocephalic and right subclavian artery (Fig. 1), and into the rest of the thoracic and abdominal aorta to the level of the left iliac artery. He underwent an emergency ascending aorta replacement, resuspension of aortic valves and CABG of the left internal mammary artery to LAD. Intraoperatively we noted the presence of a dissection flap extending from the level of the sinotubular junction to the aortic arch. A 5 cm large

**Abbreviations:** LAD, left anterior descending artery; CABG, coronary artery bypass grafting; CT, computed tomography; AAAD, acute type A aortic dissection.

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entry tear was seen at the distal ascending aorta about one-third circumference of aorta anteriorly with multiple smaller entry tears at aortic arch over the lesser curvature.

On postoperative day 5, his right hand was noted to be hypothermic without appreciable radial and ulnar arteries Doppler signals. There were no signs of compartment syndrome. A repeated CT angiogram revealed satisfactory repair of the aortic dissection but noted further propagation of the residual dissection flap down into the right axillary artery with acute occlusion of the radial and ulnar arteries (Fig. 2). He underwent a right brachial artery cutdown, radial and ulnar artery thrombolysis and embolectomy twice but was inundated with recurrent thrombosis despite being on heparin infusion. Distal to the antecubital fossa, his right forearm developed fixed mottling and blisters formation without signs of compartment syndrome (Fig. 3). Another CT angiogram was performed and reported a patent brachial artery with further tracking of the residual dissection flap down into the brachial artery with recurring occlusion of both radial and ulnar arteries (Fig. 4). As he was haemodynamically unstable, his right forearm was deemed unsalvageable and he underwent an above-elbow amputation performed by a senior consultant surgeon in the Hand and Microsurgery Department. Intraoperatively, the brachial artery dissection flap was appreciated at the level of the amputation (Fig. 5). Post-operative CT angiogram revealed no evidence of residual dissection and the patient became more haemodynamically stable after the amputation.

3. Discussion

Aortic dissections is formed by an intimal tear which allows pulsatile flow within the medial layer of the aortic wall. Pressurized blood cleaves a dissection plane within the media, with propagation along the aorta. The dominant direction of propagation of the dissection is antegrade but retrograde propagation also occurs. The separation of the aortic layers creates two aortic lumens. The true lumen is surrounded by intima and the false lumen is surrounded by the intimal–medial dissection flap and a weak medial–adventitia outer wall [4,5]. It frequently occurs in a previously dilated section of aorta. Other proposed pathogeneses include atherosclerotic intimal destruction and haemorrhage or occlusion of the vasa vasorum with consequent ischaemia. The strongest risk factors are arterial hypertension and atherosclerosis. Once dissection has occurred, acute or chronic dilatation of the false luminal channel follows. Historically, acute dissection has been defined as occurring within 2 weeks of symptom onset, with chronic dissection occurring beyond the second week [6].

DeBakey et al. introduced a classification of aortic dissections, distinguishing three subtypes based on the origin of the intimal tear and the extent of the dissection [7]. Subsequently, Daily et al. introduced the more commonly adopted Stanford classification [8]. Dissections involving the ascending aorta proximal to the brachiocephalic artery were termed type A, whereas those not involving the ascending aorta were referred to as type B dissections. This classification system disregards the site of primary intimal tear but is clinically relevant as prognosis and management depend on whether or not the ascending aorta is involved.

In addition to acute thoracic and back pain, the clinical presentation of AAAD depends on the pathological anatomy and resulting complications. The pain is usually located retrosternally and may propagate distally or proximally as the dissection evolves. The aortic adventitia is innervated with autonomic afferent nerve fibres, and the intimal tear and disruption of the media results in intense visceral pain [9].

AAAD is a life-threatening emergency. The incidence is estimated at 2.9 per 100,000 per year [10]. Historically, the mortality rate was 1–2% per hour for the first 48 h from the onset of symptoms [11]. Patients with AAAD admitted to hospital alive but treated conservatively, the mortality rate approaches 60% while in-hospital mortality rate treated surgically was found to be 17–28% [12,13].

Open surgery remains the gold standard for AAAD [14]. The main surgical principle is to reduce risk of aortic rupture or proximal extension of the dissection to prevent complications. In patients with preoperative malperfusion, restoration of blood flow should be assessed immediately after completion of the aortic repair.
With advanced age and hemodynamic instability on arrival, malperfusion constitutes a major risk factor for mortality in AAAD [15]. Malperfusion may result in coronary, brain, spinal cord, visceral organ or limb ischemia. Approximately a quarter of cases with AAAD will have limb malperfusion at time of initial presentation [16]. The prevalence of upper limb ischemia is a rare phenomenon being reported in only 1.6–3% of aortic dissection cases [17]. A high index of suspicion for malperfusion is critical to identify it both on clinical examination and imaging studies. In particular for limb ischemia, pulseless extremities and absence of arterial perfusion on arteriogram is indicative of malperfusion. Limb malperfusion is association with malperfusion of other organ system but it does not predict higher mortality or renal injury [17].

Malperfusion can be classified as dynamic, static or mixed with dynamic malperfusion being the most frequent type of malperfusion. For dynamic malperfusion, the overpressurized false lumen pushes the septum towards the true lumen and collapse the true lumen and obstruct the origin of arterial branches. Symptomatology is dynamic due to alterations in the position of the dissection flap, further antegrade or retrograde propagation, or expansion of the false lumen and subsequent compression of the true lumen. Limb ischemia may result from dynamic compression of the lumen by the false lumen. Dynamic malperfusion is treated by resecting...
the primary intimal tear and reestablish the flow towards the true lumen. Repair of aortic dissection may allow true lumen to reexpand and ameliorate dynamic malperfusion. Proceeding firstly with aortic repair avoids unnecessary peripheral revascularization. After adequate repair, post-operative limb ischaemia requiring surgery is fairly uncommon [18].

On the other hand, static malperfusion results from stenosis or occlusion of an organ arterial branch due to dissection, intramural hematoma or thrombosis. Unlike dynamic occlusion, static malperfusion will persist despite resection of the primary intimal tear and restoration of arterial flow in the true lumen. Majority of static occlusion can be treated endovascularly [19]. Rarely, interventional revascularization is unsuccessful and extra-anatomical peripheral bypass surgery is required to salvage limb perfusion emergently [17].

4. Conclusion

Though the majority of limb malperfusion resolve after repair of aortic dissection, the presence of limb malperfusion should be continually assessed as it may happen during the postoperative period due to possible propagating of dissecting flap resulting in static malperfusion. While majority of static malperfusion are amenable of endovascular or open revascularization, refractory cases should be treated with bypass expeditiously to avoid limb loss.

Declaration of Competing Interest

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Ethical approval

Case report ethical approval is exempted.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of written consent is available for review by Editor-in-Chief of this journal on request.

Author contribution

All the above authors have participated in the writing of the paper. The authors have reviewed the manuscript and approved it for submission.

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