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Case Report

Treatment of Acute Type A Aortic Dissection Involving Upper Extremity Reperfusion Injury

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A 70-year-old man underwent emergent primary central repair for acute type A aortic dissection (AAAD) with right upper extremity ischemia. Ascending aorta and hemi-arch replacement concomitant with additional right upper peripheral bypass was performed for persistent right upper arm ischemia. The early reperfusion injury (RI) of the right upper extremity was defined the next day, and managed by continuous hemodialysis (CHD) and infusion therapy, resulting in the arm being salvaged. This is an extremely rare adverse phenomenon, and we herein described its successful treatment with perioperative intensive management following central repair of AAAD.

Keywords: acute type A aortic dissection, upper extremity ischemia, reperfusion injury

Introduction

Most cases of limb ischemia caused by malperfusion (MP) associated with acute type A aortic dissection (AAAD) are generally resolved by the recanalization of blood supply to the true lumen following emergent or urgent aortic central repair without postoperative renal dysfunction or significant mortality.[1)] Although the prevalence of upper arm alone ischemia is a rare phenomenon, being reported in only 1.6%–3.0% of aortic dissection cases,[1,2)] to the best of our knowledge, there have been no previous reports on upper extremity early reperfusion injury (RI) as an uncommon adverse complication following AAAD repair. Therefore, we herein describe a successful surgical case with intensive care that developed upper limb ischemia with early RI caused by MP of AAAD.

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Case Report

A 70-year-old man was referred to our hospital with initial symptoms of right upper arm hemiplegia and severe progressive upper back pain around the right scapula. He was medium height and build, with 173 cm of height, 63 kg of weight, and 21.3 of body mass index, respectively. He was independent of preoperative activity of daily living, not sarcopenic and he has no frailty. He was not blue-collar. AAAD was confirmed by thoracoabdominal enhanced computed tomography (CT), which showed aortic dissection with primary entry at the enlarged ascending aorta (diameter of 54 mm) that extended from the brachiocephalic artery to the right axillary artery (**Fig. 1**). His consciousness was clear; however, the right brachial artery was not palpable and low arterial pressure was not measurable by right-side arterial monitoring. Four hours after the onset of his symptoms, emergent central repair by ascending aorta and hemi-arch replacement with a 28-mm Triplex prosthetic Dacron vascular conduit with a side-arm (Vascutek Terumo, Tokyo, Japan) and concomitant aortic valve resuspension for all three commissures following primary entry resection at the ascending aorta were

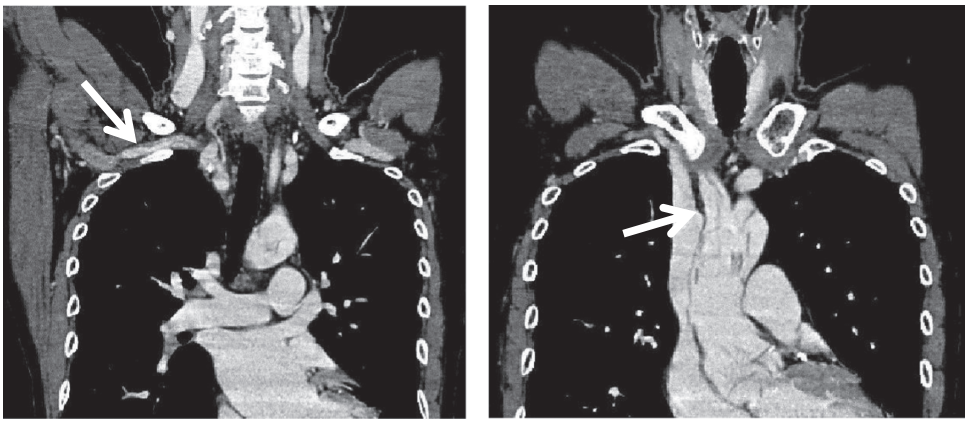

Fig. 1 Preoperative thoracoabdominal enhanced CT showed AAAD extensively involved from the brachiocephalic artery to the right axillary artery (white arrow). CT: computed tomography; AAAD: acute type A aortic dissection

successfully performed. The dissected thoracic aorta was readapted with the biologic glue, BioGlue (Cryolife International Inc., Kennesaw, GA, USA) following the establishment of cardiopulmonary bypass from femoral artery cannulation only. During surgery, near-infrared spectroscopy was measured to detect brain ischemia throughout anesthesia using the trend monitor, INVOS (Somanetics Corporation, Troy, MI, USA). Right upper ischemia was persistent with monitored low arterial pressure just after central repair of the aorta; therefore, additional concomitant upper extremity reconstruction from the proximal prosthetic graft to the right axillary artery using the side-arm of the prosthesis was immediately required to resolve persistent right upper ischemia. Since dissection of the right axillary artery was clearly revealed during anastomosis, careful anastomosis was required between the dissected right axillary artery and branch of the prosthetic graft, which prolonged the procedural time. The right upper arm was recanalized 19 hours after the onset of AAAD. Since fatal upper limb edema was unexpectedly progressive the next day with extremely high laboratory values for serum creatinine phosphokinase (CPK) of 59,659 U/L, serum myoglobin of 176,900 ng/mL, and serum aldolase 319.7 IU/L, right upper limb ischemia with early RI was diagnosed. Continuous hemodialysis (CHD) was immediately established to prevent secondary organ dysfunction such as acute kidney injury with hyperkalemia due to early RI at the intensive care unit. Compartment syndrome in his right forearm and upper arm was excluded by the measurement of intra-compartmental pressure, which was less than 30 mmHg in all areas of the right upper arm, and, thus, decompressive fasciotomy was not

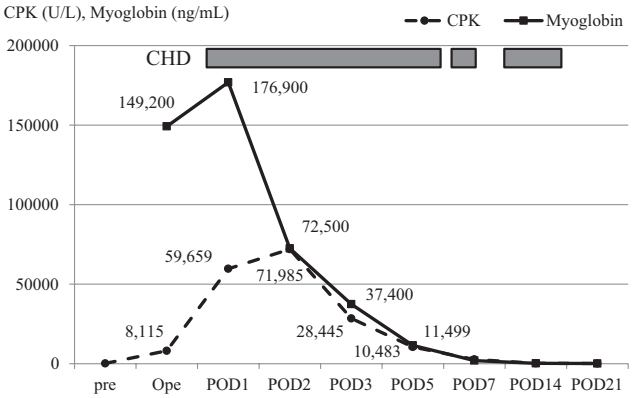

Fig. 2 Clinical course of laboratory data and CHD. CPK: creatinine phosphokinase; CHD: continuous hemodialysis; POD: postoperative day

required. The clinical course of laboratory data was shown in **Fig. 2**. His edematous right upper arm gradually improved without any hemodynamic compromise following intensive management with the immediate introduction of CHD and infusion therapy, ultimately resulting in the salvage of his right upper arm. Postoperative enhanced CT showed patient blood flow in the native right axillary artery from the brachiocephalic artery, and blood supply in the right upper limb was dominant over that in the brachiocephalic artery (**Fig. 3**). His postoperative clinical course was fair, with the residual symptoms of right upper arm neuropathy and motor paralysis gradually improving following a specific rehabilitation program and anti-hypertensive medical therapy. The patient has since returned to his regular activities and has been followed up by regular examinations at an external clinic for more than 2 years.

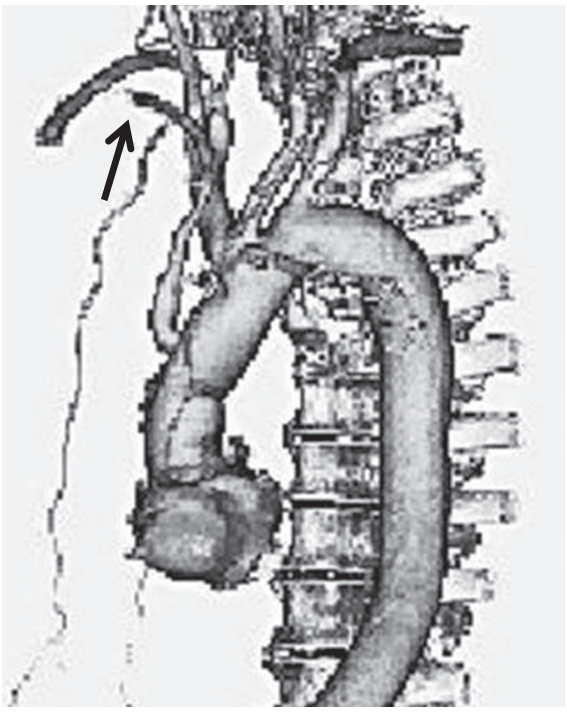

Fig. 3 Postoperative thoracoabdominal enhanced CT revealed that blood supply to the right upper limb was dependent on native blood flow from the brachiocephalic artery and the side-arm branch of the aortic Dacron graft conduit was almost occluded and blood flow might be competitive (black arrow). CT: computed tomography

Discussion

Approximately one-quarter of patients with AAAD will have evidence of peripheral vascular MP at the time of their initial presentation.[3] Furthermore, upper extremity ischemia due to MP of AAAD has been reported as a rare phenomenon, at a prevalence of a few percent.[1,2] This rare unexpected comorbidity may be used to resolve ischemic issues following emergent initial central repair of AAAD.[2–4] Although most lower extremity MP due to AAAD resolves after central aortic replacement, peripheral revascularization during or after aortic repair is sometimes required, as reported previously in only 11 out of 51 patients (21.6%) with lower limb ischemia.[1] Early postoperative interventions for perfusion deficits lead to a high rate of end-organ salvage if extremity ischemia is progressive or sustained.[4] Percutaneous interventional procedures and delayed surgery may be considered for patients with clinically apparent MP because of the very poor prognosis of immediate surgical therapy.[5] However, the strategy of immediate reperfusion, stabilization, and planned operative repair for AAAD with MP is still debated and carries a significant risk for early and late mortality.[6]

One of the adverse and unexpected clinical outcomes following extremity RI is myonephropathic metabolic syndrome (MNMS), which necessitates immediate intensive management for end-organ salvage.[7] MNMS is diagnosed by the elevated CPK levels associated with rhabdomyolysis, acute renal injury, and significant metabolic acidosis. The release of potassium, myoglobin, aldolase, histamine, and bradykinin from necrotic muscle into the bloodstream is harmful and results in life-threatening multiple organ failure. The probability of RI may be dependent on the duration of ischemia in the extremities, and the management of these complex syndromes generally consists of early revascularization with concurrent fasciotomy if needed, the re-establishment of metabolic acidosis by hemodialysis (HD) to prevent acute renal ischemia, or the early amputation of ischemic extremities for life rescue. These critical complications have also been recognized in surgical cases of thoracoabdominal aortic repair, and resolve and salvage by HD.[8] Previous studies reported that free radicals are responsible for MNMS; therefore, the therapeutic strategy for MNMS involves the correction of hyperkalemia, acidosis, the removal of myoglobin, and the protection of renal function and other organs by HD.[9,10]

It currently remains unclear why early RI following additional upper extremity revascularization occurred as an unexpected adverse outcome in our case. In general, severity of RI presented by the deteriorated laboratory data depends on the patient's physical status such as muscle volume and structure which was exposed by ischemia. We proposed the following reasons: 1) MP during CPB, 2) ischemia involving not only the upper limb, but also muscles in the right side of the chest and back, 3) persistent perioperative hypotension, 4) prolonged CPB, the aortic cross clamp time, and selective cerebral perfusion time during surgery, 5) inappropriate cerebral perfusion or cannula dislodgement of SCP at the brachiocephalic artery, and 6) the unexpected adverse effects of additional peripheral bypass to the right upper extremity. Inappropriate perfusion to the right upper extremity during hypothermia and unexpected extensive ischemic muscle volume including right upper extremity and entire right-sided thoracic area were the likely cause of early RI in this case; therefore, concomitant reconstruction of the brachiocephalic artery with central repair needs to be considered the next time.

Consequently, this is an extremely rare adverse phenomenon after central repair of AAAD, and more clinical experience is needed to resolve this rare complication.

Conclusion

We reported the successful intensive managed case with upper extremity RI following central repair of AAAD.

Disclosure Statement

There is no conflict of interest to declare.

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