

## SHORT REPORT

## Challenging Current Conservative Management of Uncomplicated Acute Type B Aortic Dissections

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**Introduction:** Despite weak evidence, current treatment guidelines for uncomplicated acute type B aortic dissection (uATBAD) consistently recommend intensive and rapid lowering of systolic blood pressure and heart rate.

**Report:** The case of a 62 year old man with uATBAD, who was treated according to guidelines, is presented. Owing to an unknown chronic occlusion of the left carotid artery combined with intensive hypotensive treatment, the patient developed a cerebral infarct.

**Discussion:** The case illustrates a severe complication of the widely accepted management of uATBAD. This case, along with scrutiny of guidelines and the evidence behind these guidelines, provoke questions regarding the rationale of current conservative management, and whether it should be challenged with alternative strategies employing a more cautious blood pressure regimen. It also highlights the importance of evaluating the vessels of the supra-aortic trunk when determining the extent of the dissection.

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### CASE REPORT

The patient was a 62 year old man. He was a current smoker with excessive alcohol consumption, but had no previous medical history. He presented with sudden onset of severe chest and back pain. On arrival, the patient was in pain but conscious; his blood pressure (BP) was 180/100 mmHg and heart rate was 100 beats per minute (bpm). Assessment of the arterial circulation to all extremities was normal, as was neurological examination. Electrocardiography and the biomarker troponin were normal.

A computed tomography angiogram (CTA) revealed an aortic dissection, starting just distal to the left subclavian artery extending down to the renal arteries (Fig. 1). Both true and false lumens were patent, and all visceral arteries were perfused by the true lumen with no signs of end organ ischaemia. The maximum aortic diameter was 42 mm.

The patient was transferred to the intensive care unit for invasive and aggressive BP management, with a target systolic pressure  $\leq 110$  mmHg and heart rate of  $\leq 60$  bpm. Intravenous labetalol was administered, and an additional angiotensin converting enzyme inhibitor was required. The

patient's pain soon subsided, but his BP was fluctuating between 80 and 160 mmHg.

On day 3 after admission the BP was increasing, and he developed neurological signs of suspected withdrawal, which was eventually perceived as delirium. The patient was sedated and required intubation.

To rule out a retrograde dissection involving the left carotid artery causing cerebral hypoxia, a new CTA was performed, but this time it included the neck and brain. This revealed a stationary image of not only the aortic dissection, but also a chronic occlusion, previously unknown, of the left internal carotid artery, along with signs of subacute cerebral infarction, located in the watershed area of the left parieto-occipital part the brain (Figs. 2 and 3).

A higher BP strategy was then allowed, keeping the systolic pressure around 140 mmHg. The rest of the hospital stay was uneventful, the patient demonstrated no focal neurological deficit, and was referred for ongoing antihypertensive care. The patient signed a consent form approving this publication.

### DISCUSSION

In The International Registry for Acute Aortic Dissections (IRAD), 2.3% of all patients with acute type B aortic dissection (ATBADs) present with stroke, but stroke as a complication of intensive hypotensive treatment for uncomplicated ATBAD (uATBAD) is rare.<sup>1</sup> This case illustrates the risk of such a treatment strategy and motivates scrutiny of the evidence behind it. Although, the patient's aortic

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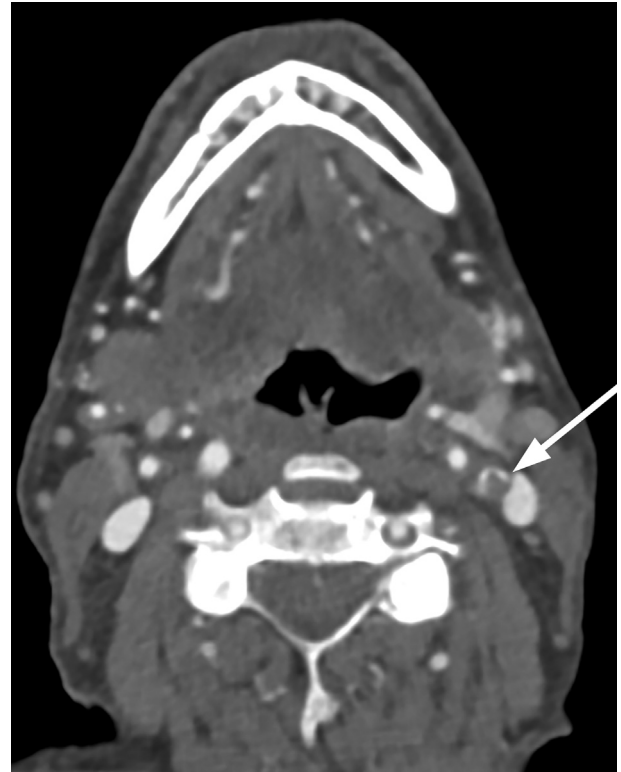


**Figure 1.** Computed tomography angiogram (sagittal view) of the aorta in the acute phase revealing an aortic intimal dissection membrane distal of the left subclavian artery.



**Figure 2.** Axial view of computed tomography of the brain demonstrating signs of subacute cerebral infarction, located in the watershed area of the left parieto-occipital part the brain.

dissection was classified as uncomplicated the situation was not, and substantiates the need for early visualisation of the supra-aortic vessels. In retrospect, it is also important to question whether it would have been more advantageous



**Figure 3.** Axial view of computed tomography of the neck, displaying a chronic occlusion of the left internal carotid artery, arrow.

to have performed an acute thoracic endovascular aortic repair once the occlusion of the ICA was revealed, which could then have been followed by carotid revascularisation, in the setting of recurrence of cerebral symptoms. This alternative approach could also have affected the possibility of managing the blood pressure positively.

Aortic dissections confined to the descending aorta—type B dissections—are treated by endovascular surgery if complicated by end organ ischaemia, aortic dilatation, or persisting severe pain. However, about 75% of all ATBADs are uncomplicated and do not require surgery and are instead managed conservatively.<sup>2</sup> This conservative management comprises continuous, invasive BP measurement—primarily administration of intravenous beta blockers with the goal of reducing systolic BP  $\leq$  100–120 mmHg, and heart rate to  $\leq$  60 bpm and impulse force (dP/dt). First line therapy is beta blockers, in order to use the anti-inotropic effect to reduce stress on the aortic wall, and thus prevent further aortic dilatation. When beta blockers are not sufficient, adding calcium channel antagonists and/or renin angiotensin inhibitors can be used as compliments. This management is widely accepted and is recommended by the European Society for Vascular Surgery's 2017 guidelines.<sup>2</sup> In the long-term, in order to meet these BP requirements, patients might need up to five different antihypertensive drugs.

The evidence for this medical strategy is weak. Many patients with ATBAD present with severe hypertension and are probably in need of their pressure being reduced, but to what level, at what pace, and with which drug?

As pointed out by Lederle et al., the medical management strategy of patients with ATBAD has somehow escaped the scrutiny of randomised controlled trials (RCTs).<sup>3</sup> The data on which the medical management strategy are based rely on dated animal studies from the 1960s and small, clinical observational studies from the 1970s, with great risk of confounding.<sup>4,5</sup> As the results showed that medical therapy for aortic dissection was superior to that of previous efforts of surgery at the time, the medical approach was rapidly embraced, and has not been questioned or properly evaluated since.

RCTs for optimal blood pressure, such as the SPRINT study, have shown that BP targets of <120 versus <140 mmHg resulted in fewer fatal and non-fatal major cardiovascular events, although with higher rates of adverse events.<sup>5</sup> However, in patients with peripheral artery disease (PAD) a J-shaped association between level of BP and all cause mortality has been shown, which has altered changes in antihypertensive guidelines for the subgroup of patients with PAD to a recommended level of 140/90 mmHg.<sup>6</sup>

Current management with prompt lowering of the BP to very low levels lacks comparison with other acute hypertensive states. In a recent study, including 84 patients treated conservatively according to guidelines for uATBAD, 43% developed in hospital renal failure, of which about half required surgical treatment. This implicates potential harm due to hypotensive treatment in a substantial number of patients.<sup>7</sup>

A Cochrane review published in 2014 did not find any evidence of any antihypertensive drug being superior to any other for aortic dissection.<sup>8</sup> Interestingly, one observational study, comprising 171 patients, indicated an improved outcome in patients with ATBAD with tight heart rate control of <60 bpm, with equal BP management <120 mmHg in both groups.<sup>9</sup>

The importance of BP for organ perfusion is undisputed, and there probably exists a threshold value, maybe specific for each organ, under which hypoperfusion results. Regarding ATBAD, there is not enough evidence to support the BP goals of current conservative medical management. Hence, it is possible that by intensive BP lowering, physicians cause more harm than good.

It is concluded that the need for better evidence for BP treatment in patients with ATBAD is warranted. While awaiting such evidence, perhaps a more moderate regimen with safer BP management should be adopted? The question that follows is what BP level should then be advocated and at what pace? Most likely, the regimen should be individualised. For hypertensive emergencies, the European Society of Cardiology recommends a 25% reduction in BP during the first hours and then proceeding cautiously<sup>10</sup>: perhaps an interval between pain relief and before oliguria occurs, which could probably allow for pressures between 120 and 140 mmHg, as suggested by Lederle et al.<sup>3</sup> This could potentially enable a quicker transit to oral

antihypertensive medication, shorten hospital stays, and, ultimately, also reduce the costs of treating these patients. In summary, it is time for a RCT of intensive medical treatment of ATBAD.

This case also raises the question of the need to screen patients with ATBAD for carotid artery pathologies. Unfortunately, existing guidelines do not offer assistance with this.<sup>2</sup> The incidence of stroke in the acute setting was 2.3% in IRAD, and was not further detailed, but the total incidence for type B and type A dissections together was 5%.<sup>1</sup> In view of the results of IRAD and this rare case, it is difficult to strongly propagate an argument for screening of the carotid arteries in patients with ATBAD. However, owing to the accessibility and ease of performing CTA or duplex of the carotid arteries in this group of patients, it is the authors' conclusion that it is worthwhile to avoid and prevent catastrophic cerebral insults.

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