# Paraspinal muscle claudication after fenestrated-branched endovascular aortic repair of thoracoabdominal aortic aneurysms

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### ABSTRACT

Fenestrated-branched endovascular repair of thoracoabdominal aneurysms carries a risk of spinal cord ischemia owing to extensive coverage of intercostal arteries, but other consequences of decreased flow to the paraspinal muscles have not been delineated. We describe a 54-year-old woman treated by multibranched thoracoabdominal aneurysm repair who developed severe disabling exertional thoracic and lumbar back pain after the operation. Despite physical therapy, the patient remains with disabling symptoms at 2 years of follow-up. Transcutaneous oxygen pressures confirmed exercise-induced decrease in oxygen pressure, consistent with decreased muscle perfusion. We propose the term paraspinal muscle claudication to describe these symptoms. (J Vasc Surg Cases and Innovative Techniques 2020;6:1-5.)

**Keywords:** Fenestrated-branched endovascular aortic repair (F-BEVAR); Thoracoabdominal aortic aneurysm (TAAA); Spinal cord ischemia (SCI); Paraspinal muscles; Paraspinal claudication; Transcutaneous oxygen pressure (TcPO2); DROP index; DROPmin

Fenestrated-branched endovascular aortic repair (F-BEVAR) has rapidly gained acceptance for treatment of complex aortic aneurysms since the first clinical implant by John Anderson in 1998.<sup>1</sup> The technique has been used for thoracoabdominal aortic aneurysms (TAAAs) and chronic dissections.<sup>2-4</sup> Although spinal cord ischemia (SCI) remains the most feared complication in up to 35% of patients, other effects of extensive coverage of intercostal and lumbar arteries have not been well-defined.<sup>5</sup> Specifically, the effect of segmental intercostal arterial occlusion on perfusion of paraspinal muscles has not been delineated, but experience with other arterial territories indicates that exercise-induced ischemia may occur. We present a patient with debilitating exertional thoracic and back pain following F-BEVAR for extent II TAAA and propose the term paraspinal musculature claudication.

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#### **CASE PRESENTATION**

The patient consented for publication of this case report. A 54year-old woman presented with an asymptomatic 6.3-cm extent II TAAA (Fig 1, A). Her history was notable for ischemic cardiomyopathy, prior myocardial infarction requiring placement of drugeluting stent, hypertension, and predialysis stage 5 chronic kidney disease on evaluation for possible kidney transplant. Computed tomography angiography also demonstrated 13 pairs of segmental/intercostal arteries that would be covered by endovascular repair, however the bilateral hypogastric and subclavian arteries were patent. A staged endovascular approach was recommended for treatment of the TAAA.

Endovascular repair. The first thoracic endovascular aortic repair was performed from zone 3 to zone 5 using a TX2 stentgraft (Cook Medical Inc, Bloomington, Ind). A second stage completion multibranched TAAA repair was performed 5 days later using a Cook T-branch (Cook Medical Inc) as previously described.<sup>6</sup> Completion aortography and noncontrast conebeam computed tomography revealed widely patent branches (Fig 1, B). The postoperative course was uneventful. Spinal drainage was removed on postoperative day 3 without complications. The patient endorsed some new-onset low back pain, but cerebrospinal fluid leakage was ruled out and magnetic resonance imaging did not show significant musculoskeletal abnormalities. There were no neurologic deficits and no lower extremity numbness or weakness. The patient was discharged on postoperative day 7 with a stable creatinine of 4.7 mg/dL and did not require dialysis.

**Evolution of back pain.** The thoracic and lower back pain persisted throughout weeks to months of postoperative recovery. The pain was described as worse on the left side, with onset within 2 minutes of standing. The pain extended from the low thoracic to lumbar levels and was cramping in nature. This

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**Fig 1.** Preoperative computed tomography angiography (CTA), three-dimensional reconstruction of asymptomatic extent III thoracoabdominal aortic aneurysm (TAAA) **(A)**; postoperative CTA, three-dimensional reconstruction demonstrating a widely patent four-vessel branched reconstruction **(B)**. Image reproduced by permission of Mayo Clinic Foundation for Medical Education and Research.

pain resulted in severe disability causing limitation at work and activities of daily living. Symptoms were absent in the morning and progressed throughout the day with standing and activity. The pain was alleviated by rest and lying supine. Physical therapy over the ensuing 18 months resulted in improvement to standing for 1 hour, but the patient continued to endorse significant daily limitation. Neurology and spine evaluation ruled out other etiologies. Repeat magnetic resonance imaging demonstrated modest degenerative changes in the low cervical and thoracic spine, but no mass effect on the spinal cord and no findings to indicate neuropathic or radicular etiology. Electromyography was negative and there were no significant differences in the spine when comparing preoperative and postoperative computed tomography scans. She also underwent multiple evaluations with pain medicine, and complex regional pain syndrome was ruled out as a likely etiology.

Given the exertional nature of the thoracolumbar fatigue and pain after complete coverage of the thoracoabdominal aorta, paraspinal muscle hypoperfusion was suspected. Noninvasive exercise treadmill testing was performed to evaluate the effects of exertion on the paraspinal musculature perfusion, using transcutaneous oxygen pressures ( $TcPO_2$ ).

**Transcutaneous oxygen pressure**. The treadmill test reproduced significant back pain for the patient, both during and after exercise. Although she was able to complete the

protocol, it was performed at a slower speed. The Delta from Resting Oxygen Pressure (DROP) index, which is the absolute change in  $TcPO_2$  from resting value (Appendix) was measured.<sup>7</sup> A minimal DROP value during exercise that indicates presence of buttock muscle ischemia is less than -15 mm Hg, whereas a normal study demonstrates DROP values at or close to 0 mm Hg. In this patient, the DROP value was less than 0 mm Hg but greater than -15 mm Hg during exercise, with the most significant decreases (-10 to -12 mm Hg) occurring in the lumbar areas (Fig 2). Continued aggressive physical therapy was recommended, with a focus on the paraspinal musculature, because this had resulted in some improvement of symptoms. The patient has persistent symptoms at 25 months of follow-up.

#### DISCUSSION

Exercise-induced pain is a common symptom with occlusive disease of the pelvis and lower extremities, but has not been described with extensive occlusion of intercostal and lumbar segmental arteries. Most of the concern with endovascular TAAA repair involves the risk of spinal cord injury, which ranges from 0% to 40% in contemporary F-BEVAR series.<sup>5</sup> Although focus has centered on strategies to prevent SCI such as permissive hypertension, cerebrospinal fluid drainage, and



**Fig 2.** Graphical representation of paraspinal transcutaneous oxygen pressures (*TcPO*<sub>2</sub>), with the greatest drops being observed in the left low back (–10 mm Hg) and right low back (–12 mm Hg) during exercise and resolving with rest. Image reproduced by permission of Mayo Foundation for Medical Education and Research.

temporary sac perfusion, no attention has been given to prevention of other symptoms such as paraspinal muscle claudication.<sup>2,5</sup> We propose the term paraspinal muscle claudication to describe new-onset exercise induced back pain that occurs after extensive sacrifice of segmental intercostal and lumbar arteries.

The theory that a single end-artery such as the notorious artery of Adamkiewicz is the primary arterial supply to the spinal cord has been challenged by Griepp et al.<sup>8</sup> The importance of extrasegmental arteries was demonstrated in porcine models by Strauch et al,<sup>9</sup> who noted that when the subclavian or median arteries were occluded, fewer intercostal or lumbar arteries could be sacrificed without jeopardizing cord perfusion.<sup>10</sup> Etz et al<sup>10</sup> later performed a series of anatomic studies, where methyl methacrylate was injected into juvenile pigs' circulation in order to make a cast model of the collateral spinal cord network. This work revealed a vast matrix of small arteries and arterioles that connect the relatively modest intraspinal arterial network, to a dense collateral pathway that interconnects within the paraspinal muscles.<sup>10</sup> Recently, this new understanding of the collateral network through paraspinal muscles has been harnessed for indirect spinal cord monitoring during TAAA repair, with near infrared spectroscopy being used intraoperatively in conjunction or in lieu of neuromonitoring.<sup>11-13</sup>

Although the focus of extensive aortic coverage is on cord perfusion, the potential deleterious effects on paraspinal muscles have been overlooked. Microarray-based investigations have demonstrated differential gene expression in paraspinal muscles during staged aortic coverage.<sup>14</sup> However, the focus of these studies was to

aid in SCI prevention and not to identify arterial insufficiency of the paraspinal muscles. This patient developed exertional muscular pain after extensive aortic coverage. Although the  $TcPO_2$  minimal DROP did not reach the threshold of -15 mm Hg used for a positive buttock claudication study.<sup>7</sup> the drop was highly suggestive of decreased perfusion. The hypothesis of paraspinal claudication owing to diminished perfusion after aortic coverage is consistent with the exertional nature of her pain. Furthermore, improvements in standing time and activity after long-term physical therapy could potentially be equated to a supervised walking program in the treatment of traditional lower extremity claudication.

There are several limitations of using TcPO<sub>2</sub> to assess paraspinal muscle perfusion. This application of exercise TcPO<sub>2</sub> has not been previously reported or validated against other methods of noninvasive testing. However, the use of exercise TcPO<sub>2</sub> to assess buttock perfusion in lower extremity ischemia has been assessed and validated.<sup>7,15,16</sup> Similarly, near infrared spectroscopy intraoperative monitoring uses similar principles with a greater depth of penetration to assess paraspinal perfusion as a surrogate for neuromonitoring, as several studies have shown correlation with traditional motorpotentials.<sup>11-13</sup> evoked and somatosensory-evoked Another limitation of this study is the inability to explain the absence of any SCI, given that neural tissue is typically more sensitive to changes in perfusion than skeletal muscle. A prospective protocol, including imaging of the collateral network at preoperative and postoperative intervals, is needed to both validate this specific application of exercise TcPO2 to the paraspinal musculature, Journal of Vascular Surgery Cases and Innovative Techniques Volume 6, Number 3

and assess for any anatomic changes that occur in the paraspinal collateral network after extensive stent graft aortic coverage.

## CONCLUSIONS

Exertional back pain after F-BEVAR for TAAAs may occur owing to decreased perfusion and insufficient compensation by the paraspinal collateral network. Physical therapy focused on the paraspinal muscles may improve symptoms. Further studies are needed to confirm and further delineate the effects of extensive aortic coverage on this muscular bed.

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## APPENDIX.

Formula to calculate the decrease from rest of oxygen pressure (DROP) index at site of interest

$$\begin{split} \mathsf{DROP}_{(site)} &= [\mathsf{PO}_{2(site)} \ t_t - \mathsf{PO}_{2(site)} \ t_0] \\ &- [\mathsf{PO}_{2(chest)} \ t_t - \mathsf{PO}_{2(chest)} \ t_0] \end{split}$$

 $PO_{2(site)} t_{t}$  oxygen pressure at site of interest at time t $PO_{2(site)} t_{0}$ : oxygen pressure at site of interest at time 0 or rest

 $PO_{2(chest)} t_t$ : oxygen pressure at chest probe at time t  $PO_{2(chest)} t_0$ : oxygen pressure at chest probe at time 0