

## Clinical Notes

### Ischemic monomelic neuropathy obscured by diabetes and stroke after thoracic endovascular aortic repair

*Ahmad J. Abdulsalam, MD, Biju Gopinath, MD, Buthaina M. Alkandari, MD, FFRRCSI, Daa Shehab, MD, FRCP, Salem A. Alkandari, MD, FBPRM.*

A 58-year-old male was transferred to the emergency department with back pain after a traffic accident. On arrival, his heart rate was 80 beats per minute, blood pressure was 115/65 mmHg, and oxygen saturation was 97%. His level of consciousness, based on the Glasgow Coma Scale, was E 2, V 4, M 6. He showed slight confusion but could communicate and interact. Past medical history revealed that he was a smoker and hypertensive, with poorly controlled diabetes. He had no known history of vascular disease. Chest computed tomography (CT) showed a large saccular aneurysmal dilatation in the thoracic aorta, just distal to the left subclavian artery (LSA) (type B).

Minutes after the CT scan, the patient went into frank shock (arterial pressure: 50/30 mmHg). Therefore, he was placed under general anesthesia in the angiography room and underwent an emergency thoracic endovascular aortic repair (TEVAR). During the repair, due to a non-sufficient length of the proximal

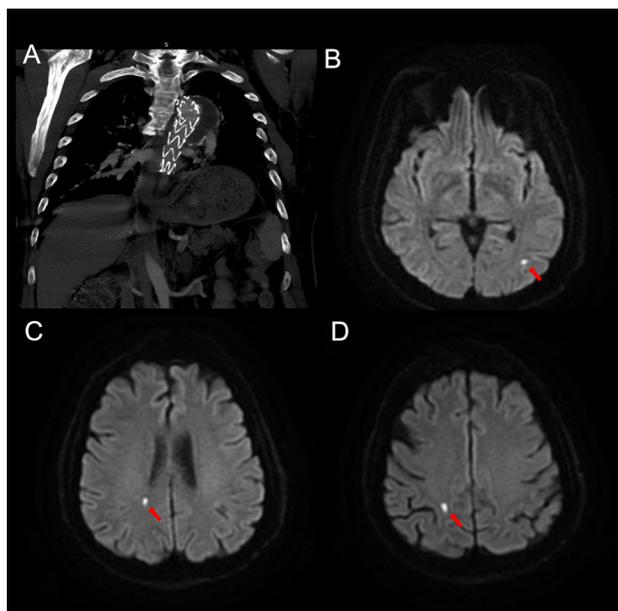
landing zone, the LSA was covered. The proximal landing zone was set to be between the left common carotid artery and the LSA. A stent graft was subsequently fully deployed (Valiant Captivia Medtronic Proximal Freeflo, straight, 22 mm×152 mm). An angiogram showed persistent slow filling of the LSA from the arch, with no evidence of endoleak or antegrade flow (Figure 1A). Late flow was noted into the LSA from the left vertebral artery.

Subsequent to the procedure, the patient was shifted to the ICU due to hypotension (arterial pressure: 80/50 mmHg). One day later, the patient complained of left-sided weakness, numbness, and pain in the upper limb, along with reduced grip strength and arm and wrist movement. Examination revealed a 3/5 reduction in motor power in the left upper limb including shoulder abductors, elbow flexors, elbow extensors, wrist flexors, wrist extensors, with finger flexors, finger extensors. In the left lower limb, including the hip flexors/abductors, knee flexors and extensors and ankle plantar and dorsiflexors, the motor power was 4/5. Deep tendon reflexes were brisk on the left side and the plantar reflexes were upgoing. Sensation was also impaired in the whole left upper limb, accompanied by a stocking-glove pattern in all four limbs. A brain CT showed multiple ill-defined hypodensities. A follow-up MRI revealed three acute lacunar infarctions in the right parietal and left occipital lobes (Figure 1B, C, D). The patient was therefore diagnosed with a stroke after the TEVAR.

The stocking-glove pattern and decreased sensation in the left upper limb prompted electromyography and nerve conduction studies (NCSs) 1 month later (Table 1). The findings were severe sensory motor axonal neuropathy of the left upper limb, with a distal to proximal gradient. The right upper limb had evidence of sensory motor axonal polyneuropathy. These findings were correlated as ischemic neuromuscular injury to the left upper limb, with superimposed axonal distal polyneuropathy (diabetes). Therefore, he was diagnosed with both diabetic neuropathy and ischemic monomelic neuropathy (IMN).

After a one-year follow-up, his motor weakness improved mildly. Despite being on a high dose of gabapentin, he still complained of neuropathic pain. His left arm also had a slightly lower blood pressure (95/65mmHg) when compared to the right arm (130/80mmHg).

We present a patient with diabetic neuropathy who developed a stroke and IMN after TEVAR. TEVAR is known to cause complications like stroke, which is thought to be mainly associated with covering of the LSA during the procedure.<sup>1,2</sup> By contrast, IMN due



**Figure 1** - Post-TEVAR CT MIP images demonstrating a) proper apposition of the graft, b,c,d) MRI brain (diffuse weighted images) revealing small high signal intensity lesions of both cerebral hemispheres.

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**Table 1 -** Motor conduction study showed absent CMAP for left median nerve. Left ulnar nerve showed markedly reduced CMAP amplitude with mild reduction in conduction velocity.

Nerve	Stimulus	Recording	Dist (mm)	Motor side to side comparison								
				LatOn (ms)		B-Pamp (mV)		B-PArea (mVms)		CV (m/s)		
				L	R	L	R	L	R	L	R	
Median	Wrist	APB	NR	80	NR	4.50	NR	6.67	NR	22.8	NR	n/a
	Elbow		NR	225	NR	9.00	NR	5.71	NR	21.2	NR	50.0
Ulnar	Wrist	ADM	80	80	3.58	3.33	0.37	4.27	1.3	11.4	n/a	n/a
	B. Elbow		255	230	9.42	7.67	0.21	3.93	0.7	11.1	43.7	53.1
	A. Elbow		100	100	11.92	9.58	0.34	4.18	1.3	11.8	40.0	52.2

APB - abductor pollicis brevis, ADM -abductor digiti minimi, mVms - millivoltmillisecond, R - right, L - left, CV - conduction velocity, CMAP - compound muscle action potentia

**Table 2 -** Sensory conduction study showed absent SNAP for left median, radial and ulnar nerves. Right side showed low amplitude SNAP.

Nerve	Stimulus	Recording	Dist (mm)	Sensory Side-To-Side Comparison								
				LatOn (ms)		LatNPK (ms)		B-PAMP (µV)		CV (m/s)		
				L	R	L	R	L	R	L	R	
Med/Uln/Rad	Wrist	R Thumb	NR	100	NR	1.92	NR	2.47	NR	4.74	NR	52.2
		M Thumb	NR	100	NR	2.85	NR	3.45	NR	7.18	NR	35.1
		5 <sup>th</sup> dig.	NR	140	NR	2.93	NR	3.68	NR	4.28	NR	47.7

N - number, R- right, L- left, SNAP- Sensory nerve action potential, CV- conduction velocity,

**Table 3 -** Needle EMG of upper limb showed denervation potentials in the left biceps and bilateral first dorsal interosseous. Left upper limb showed neurogenic polyphasic MUAPs in all sampled muscles. Recruitment pattern was reduced with more reduction in distal muscles. Right side showed few polyphasic in extensor digitorum and first dorsal interosseous. Right deltoid was mostly unremarkable.

Needle EMG Summary										
Muscle	Root	Ins. Act	Fibs.	PSW	Fascics.	Polyp.	MU amp.	MU Dur.	Pattern	Recruit
<i>Left</i>										
Deltoid	Axillary C5-C6	Nor	0	0	0	++	+	++	Reduced	Mod. Redu
Biceps Brachi	Musculocut C5-C6	Nor	1+	0	0	++	+	++	Reduced	Mod. Redu
Triceps	Radial C6-C7	Nor	0	0	0	++	+	+	Reduced	Mod. Redu
Ext Dig. Com	Post Inter C7-C8	Nor	0	0	0	++	+	++	Reduced	Severe Redu
First Dors. Int	Ulnar C7-T1	Nor	1+	1+	0	+	+	+	Reduced	Poor Activation
<i>Right</i>										
Deltoid	Axillary C5-C6	Nor	0	0	0	+	+	+	Full	Normal
Ext. Dig. Com	Post Inter C7-C8	Nor	0	0	0	++	++	+	Full	Normal
First Dors. Int	Ulnar C7-T1	Nor	1+	0	0	++	++	+	Full	Normal

PSW - Positive sharp waves, MU -Motor unit, EMG - Electromyography, MUAPs - Motor unit action potentials

to TEVAR has been described only once before in the literature.<sup>3</sup> The IMN is a type of peripheral neuropathy that develops as a rare complication after shunting of blood flow or due to acute non-compressive occlusion of the major proximal limb artery after vascular surgery; both of these blood-flow disruptions cause ischemia.<sup>4,5</sup> Notably, IMN tends to occur in patients with pre-existing microvascular disease, like our patient who had uncontrolled diabetes.<sup>4,5</sup>

Many clinicians have difficulty diagnosing IMN. In general, neurologic motor and sensory deficits appear

immediately after the vascular procedures.<sup>3</sup> However, many patients present with other comorbidities, such as severe neuropathic pain and allodynia, which can overshadow the motor function losses. Since our patient had diabetic neuropathy, along with hemiparesis from the stroke, clinically it was very difficult to detect and suspect this rare neuropathy.

As in our case, electrodiagnostic studies are helpful for differentiating IMN from other conditions.<sup>3-5</sup> The NCSs usually show axonal loss and reduced motor and sensory nerve conduction velocities in the median,

radial, and ulnar nerves.<sup>3-5</sup> Needle electromyography primarily reveals acute denervation and reduced motor unit recruitment of the upper limbs.<sup>3-5</sup>

In conclusion, we note to physicians that IMN occurs not only in dialysis patients but also in those with other vascular accesses, like TEVAR. Hence, improved awareness of this disease entity and clinical suspicion of neurologic deficit in the immediate post-operative period can provide early diagnosis and treatment. Last but not least, nerve conduction examinations are important for clarifying the obscurity of IMN in the presence of comorbidities like stroke and diabetes.

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*From the Department of Physical Medicine and Rehabilitation (Abdulsalam, Alkandari), Physical Medicine and Rehabilitation Hospital, Andalous, Department of Physical Medicine and Rehabilitation (Gopinath, Shehab), Mubarak Al-Kabeer Hospital, Jabriya, and from the Department of Medical Imaging (Alkandari), Jaber Al-Ahmad Hospital, South Surra, Kuwait. Address correspondence and reprint requests to: Dr. Ahmad J. Abdulsalam, Department of Physical Medicine and Rehabilitation, Physical Medicine and Rehabilitation*

*Hospital, Andalous, Kuwait. E-mail: dr.ahmad.j.abdulsalam@gmail.com  
Orcid ID: <https://orcid.org/0000-0002-3592-6933>*

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