Acute Anterior Spinal Cord Syndrome Following Invasive Coronary Procedures: A Rare but Devastating Complication- an editorial

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The remarkable advances in invasive cardiology over the past few decades have transformed the prognosis of patients with coronary artery disease. Coronary angiography and percutaneous coronary intervention (PCI) are now among the most frequently performed procedures worldwide, with an excellent overall safety profile. Nevertheless, as procedural volumes continue to increase, clinicians are increasingly confronted with uncommon but potentially catastrophic neurological complications beyond the well-recognised risks of cerebral stroke. Acute anterior spinal cord syndrome (ASCS) represents one such rare complication that deserves greater clinical awareness [1].

Spinal cord infarction accounts for only 1–2% of all vascular neurological disorders and approximately 5–8% of acute myelopathies [2]. Among these, ASCS is the most common clinical subtype, resulting from ischaemia within the territory supplied by the anterior spinal artery. The syndrome classically presents with sudden bilateral motor weakness, impairment of pain and temperature sensation below the lesion, autonomic dysfunction, and relative preservation of vibration and proprioception owing to sparing of the posterior columns [2,3]. Although aortic surgery remains the leading iatrogenic cause of spinal cord ischaemia, invasive coronary procedures have emerged as an increasingly recognised but under-reported precipitant [4].

Several pathophysiological mechanisms may explain the occurrence of ASCS following coronary angiography or PCI. Procedure-related hypotension, transient or prolonged cardiac arrest, systemic hypoperfusion, cholesterol or thromboembolic phenomena, contrast-induced vasospasm, and catheter-related atheroembolism have all been implicated [5,6]. The thoracic spinal cord, particularly the watershed zone extending from T4 to T8, is especially vulnerable because of its relatively limited collateral circulation and dependence on segmental radicular arteries, including the artery of Adamkiewicz [7]. Even brief periods of profound hypotension during cardiac arrest or haemodynamic instability may result in irreversible spinal cord injury.

Despite its devastating consequences, ASCS remains diagnostically challenging. The rarity of the condition, variable timing of symptom onset, and the frequent coexistence of critical cardiac illness may delay recognition. In many patients, neurological deficits become apparent only after recovery from sedation, mechanical ventilation, or post-resuscitation encephalopathy. Consequently, acute paraplegia following invasive cardiac procedures may initially be attributed to critical illness neuropathy, metabolic derangements, or cerebral hypoxic injury, thereby postponing definitive evaluation[8].

The hallmark clinical feature of ASCS is dissociated sensory loss—loss of pain and temperature sensation with preservation of dorsal column modalities. Recognition of this pattern is crucial. Magnetic resonance imaging (MRI) remains the investigation of choice, demonstrating T2 hyperintensity involving the anterior two-thirds of the spinal cord and, in some cases, the characteristic axial "owl's eyes" appearance [9]. However, MRI findings may be normal during the early hours after symptom onset, and repeat imaging may be necessary when clinical suspicion remains high [2].

Unfortunately, therapeutic options remain limited. Unlike cerebral ischaemic stroke, no evidence-based reperfusion strategy exists for spinal cord infarction. Current management is largely supportive and includes optimisation of spinal cord perfusion, maintenance of haemodynamic stability, prevention of secondary complications, and early multidisciplinary rehabilitation [10]. The role of corticosteroids is unproven, and routine administration cannot be recommended in the absence of inflammatory myelopathy [11]. Functional outcomes vary widely; while some patients experience meaningful recovery, many remain permanently disabled with persistent motor deficits, sphincter dysfunction, and reduced quality of life [3].

The expanding use of invasive coronary procedures mandates heightened vigilance for rare neurological complications. Early neurological consultation should be sought whenever patients develop acute lower limb weakness, sensory deficits, or bladder dysfunction after coronary angiography or PCI. Greater awareness among cardiologists, intensivists, neurologists, and rehabilitation specialists is essential to facilitate prompt diagnosis and optimise patient outcomes.

Future research should focus on establishing the true incidence of spinal cord ischaemia following invasive coronary procedures, identifying procedural and patient-related risk factors, and exploring potential neuroprotective strategies. Large multicentre registries and collaborative studies are urgently needed. As interventional cardiology continues to evolve, preventing and mitigating uncommon complications such as ASCS will become increasingly important in delivering truly comprehensive cardiovascular care.

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