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SPINAL ISCHEMIA AFTER PROLONGED RESUSCITATION AND CORONARY ANGIOGRAPHY UNDER IMPELLA PROTECTION

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ABSTRACT

Spinal ischemia is a rare condition and accounts for only 1-2% of all neurovascular diseases. In this case, spinal ischemia occurred in a 46-year-old patient after prolonged resuscitation after out-of-hospital cardiac arrest and following invasive coronary angiography and percutaneous coronary intervention under mechanical circulatory support with an Impella (Abiomed, Danvers, MA, USA). Magnetic resonance imaging showed an infarct demarcation from the 5th thoracic vertebra to the conus medullaris. The supply areas of the anterior spinal artery (paraplegia, no response to pain stimulus) as well as the posterior spinal arteries (reduced sensation of touch) were affected. Magnetic resonance imaging of the brain showed both signs of hypoxic brain damage and embolic events. In terms of pathogenesis, the low-flow-phase of resuscitation with associated arterial thromboembolism as well as a thromboembolic event by the Impella need to be considered. To date, limited data on the prevalence and incidence of spinal ischemia after resuscitation and/or use of an Impella are available in the literature.

KEYWORDS

Cardiac arrest, spinal ischemia, Impella, embolism, hypoperfusion

LEARNING POINTS

- Impella (ventricular assist device) can be associated with thromboembolic events.
- The low flow phase of resuscitation can be associated with arterial thromboembolism.
- Spinal ischemia can occur after resuscitation.

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INTRODUCTION

Spinal ischemia is a rare condition and accounts for only 1-2% of all neurovascular diseases[1].

Three large blood vessels supply the spinal cord: the anterior spinal artery (supplying the anterior horns of the grey matter, the spinothalamic tracts and the corticospinal tracts) and the paired posterior spinal arteries (supplying the posterior cords and posterior horns). All three arteries originate from the vertebral arteries and are contributed by the radicular arteries (originating from the vertebral arteries, intercostal arteries and rarely directly from the aorta). The most important thoracic radicular artery is the artery of Adamkiewicz, which contributes to the anterior spinal artery between T9 and T12 in 75% of cases but can also be found cranial or caudal to this area[2].

Most cases of spinal ischemia occur in the area supplied by the anterior spinal artery and cause bilateral loss of motor function as well as loss of pain and temperature sensation caudal to the lesion. In posterior spinal artery syndrome, there is loss of proprioception and vibratory sense below the lesion as well as anaesthesia in the area of the lesion. Unilateral involvement is more common, but cases of bilateral involvement have also been described[3].

Aortic surgeries represent the most common cause of spinal ischemia. Aortic dissections can cause spinal ischemia through the same mechanism as aortic surgeries (failure of the aortic side branches). In these aetiologies the thoracic spinal cord is usually affected. Other causes of spinal ischemia are severe systemic hypotension or resuscitation, in which the lumbosacral spinal cord is predominantly affected. Spinal cord ischemia has also been described with the use of venoarterial extracorporeal membrane oxygenation (V-A ECMO) as well as in connection with vasculitis and thromboembolic disease[3].

The exact incidence of spinal ischemia after resuscitation is not known. In a case series which contained 44 cases of spinal ischemia 4 were described as resulting from resuscitation[4].

Gradual clinical recovery after spinal ischemia is possible. Older age, increased severity of impairment at nadir (maximum impairment) and peripheral vascular disease were independent risk factors for mortality and severe impairment at nadir predicted wheel-chair use and bladder catheterization 3 years later in a multivariable statistical analysis[5].

CASE DESCRIPTION

We report the case of a 46-year-old female who was resuscitated following an out-of-hospital cardiac arrest (OHCA) with refractory ventricular fibrillation (no no-flow time, paramedics present at cardiac arrest). Under ongoing

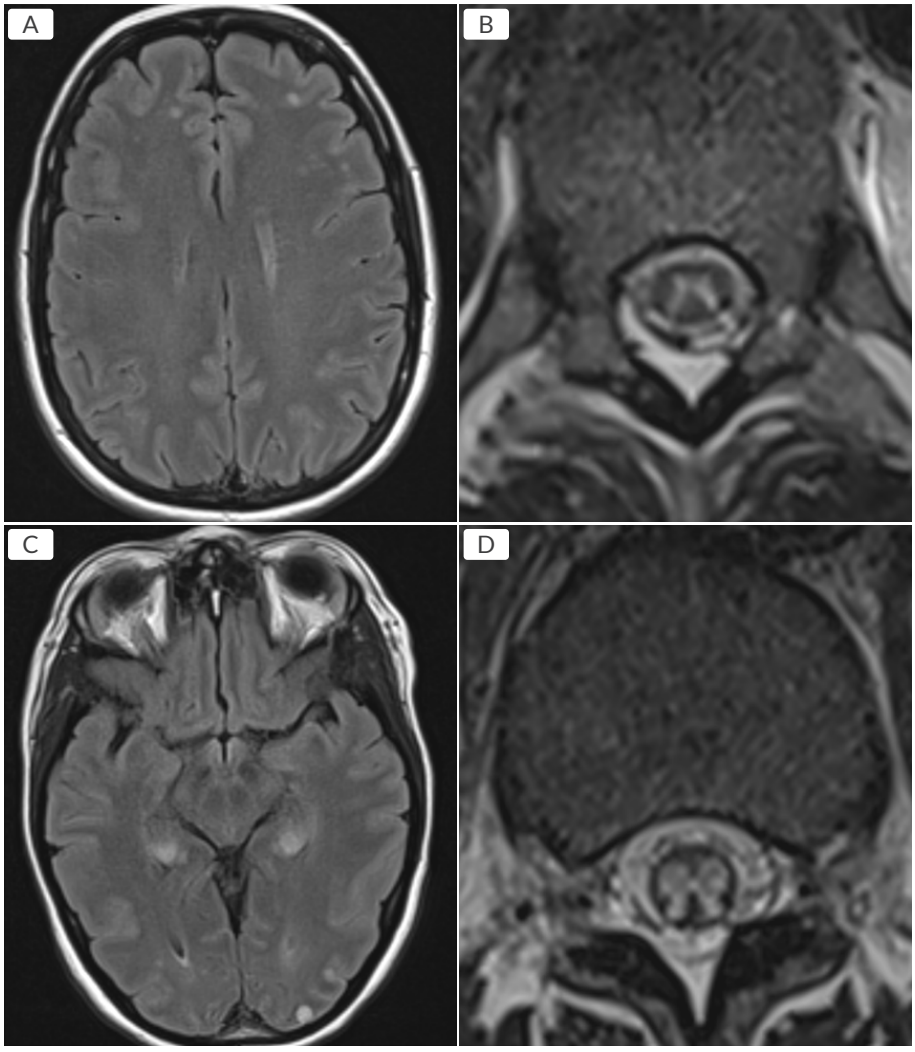


Figure 1. *Magnetic resonance imaging (MRI) of the brain (A, C) and spine (B, D); A, C) T2 fluid-attenuated inversion recovery (FLAIR) transverse. A) Multifocal restricted diffusion with hyperintensity in FLAIR and a patchy pattern, predominantly in the peripheral cortex and some subcortical. C) Demarcation in FLAIR and minor swelling along the hippocampus. B; D) T2 turbo spin echo transverse hyperintensity in the anterior and posterior horns of the grey matter (B) T9/10, (D) T10/11.*

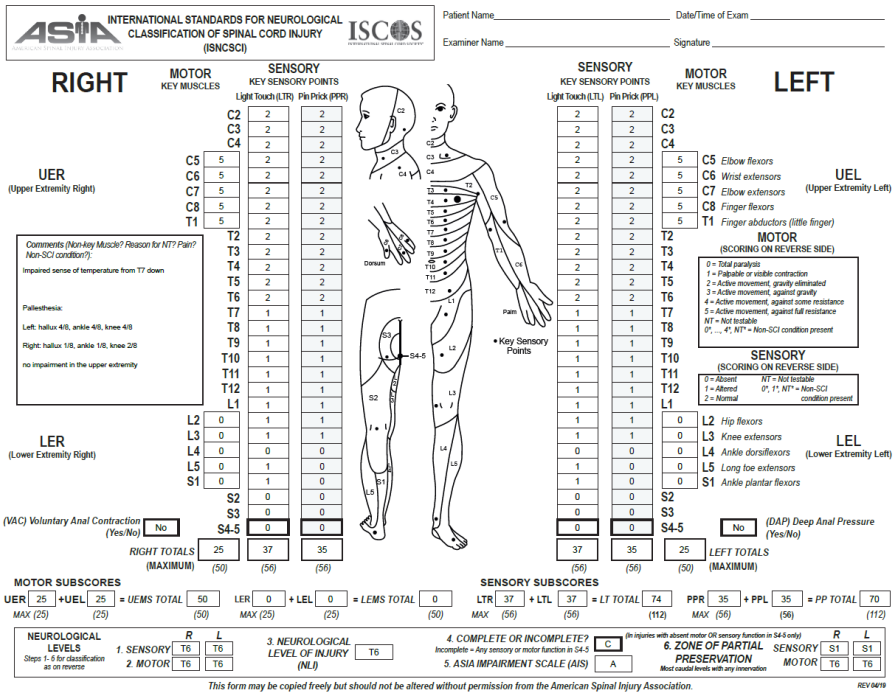


Figure 2. International standards for neurological classification of spinal cord injury carried out in the rehabilitation clinic 2.5 months after diagnosis: paraplegia, impaired sense of temperature from T7 down, impaired sense of vibration of the lower extremity, altered or absent sense of touch from T7 down and loss of voluntary contraction of the anal sphincter.

resuscitation using a mechanical cardiac compression device (Autopulse) the intubated patient was taken to the cardiac catheterization laboratory of a university hospital. There, return of spontaneous circulation occurred after approximately an hour and 45 minutes (maximum lactate 13.3 mmol/l, end-tidal carbon dioxide between 2.1 and 4.6 kPa). An Impella (Abiomed, Danvers, MA, USA) was inserted via the right femoral artery (left ventricular assist devices, such as the Impella micro-axial flow device, pump blood from the left ventricle or left atrium to the aorta. It works on the principle of an Archimedes' screw, which revolves at high speeds draws blood out of the left ventricle and ejects it proximally into the ascending aorta). Recanalization of an ostial occlusion of the left anterior descending artery was performed in underlying single-vessel coronary artery disease. The systolic blood pressure values during the intervention were between 60 and 110 mmHg with an upward trend. Critical ischemia of the right leg being suspected, the Impella was removed immediately after the intervention. The patient was transferred to the intensive care unit (blood pressure on arrival 115/92 mmHg). An echocardiography revealed severely impaired left ventricular systolic function (visually estimated 25-30%) without any evidence of a left ventricular thrombus.

The patient was extubated without any problems on the first day after the OHCA. She was disoriented reaching a score of 14 on the Glasgow coma scale (GCS) and presented with paraplegia. Sensibility could not be adequately assessed initially due to a language barrier.

Magnetic resonance imaging (MRI) of the brain (with contrast agent) and of the entire spine (without contrast agent) revealed multiple patchy embolic acute/subacute infarcts bilaterally supra- and infratentorial, bilateral symmetric acute/subacute infarcts of the hippocampus (based on the pattern and the context possibly of hypoxic origin) and an acute/subacute extended spinal infarct demarcation from the 5th thoracic vertebra (T5) to the conus medullaris *(Fig. 1)*. A lumbar drainage was not inserted due to dual platelet inhibition after coronary intervention and the risk of spinal bleeding.

The clinical course was complicated by, presumably hypoxic, encephalopathy and aspiration pneumonia with difficult weaning and ultimately tracheotomy. In the further course, there was an improvement in vigilance with a maximum score of 11 on the GCS in a patient with tracheostoma (eye opening response 4, motor response 6, verbal response 1). However, the paraplegia persisted. The patient was transferred to a rehabilitation clinic with focus on neurorehabilitation after 19 days in the intensive care unit. A follow-up 10 weeks after the event revealed persisting complete flaccid paraplegia with bowel and bladder dysfunction. From T7 downwards there was limited sensation of touch as well as limited sharp/dull discrimination and limited temperature sensation *(Fig. 2)*.

DISCUSSION

Our report presents the rare clinical picture of spinal ischemia after prolonged resuscitation and use of an Impella during percutaneous coronary intervention. Regarding the aetiology of the spinal ischemia, hypoperfusion seems more likely than emboli in both the anterior spinal artery and the posterior spinal arteries at the same time. Further differentiation of the aetiology based on the imaging was not possible.

The fact that both the supply areas of the anterior spinal artery and those of the posterior spinal arteries were affected speaks in favour of systemic hypoperfusion. Due to the watershed zone between T4 and T8 (with relatively few radicular arteries) the thoracic spinal cord is considered to be particularly vulnerable to ischemia[6]. In this case the

ischemia extended from the 5th thoracic vertebra to the conus medullaris. Similar cases of spinal ischemia after cardiac arrest with the highest level of impairment in the thoracic spinal cord can be found in the literature[6-8].

However, autopsy reports of patients with spinal ischemia following cardiac arrest or severe hypotension show that the lumbar spinal cord is predominantly affected (the lumbar spinal cord exclusively in 69.7% of cases, n=66). The thoracic spinal cord was affected in only 7.6% of cases and never alone[9].

In addition to systemic hypoperfusion, an embolic origin is also theoretically possible. The patient's MRI of the brain showed multiple embolic infarcts. A study on the characteristics of acute cerebral ischemia after cardiac arrest revealed that almost 62% of patients with MRI-detected ischemia showed the pattern of small multifocal emboli (possibly due to a hypercoagulable state and cardio-embolism)[10]. There are no case reports of spinal ischemia after implantation of an Impella, but cases of ischemic stroke with an embolic pattern with an Impella in place have been described. However, these events occurred on average 5 days after the insertion of the Impella[11]. In this patient the Impella was removed immediately after percutaneous coronary intervention.

In summary, both systemic hypoperfusion and an embolic event are possible causes of spinal ischemia in this case, whereby the latter could have been caused by either arterial thromboembolism associated with the low-flow phase of resuscitation or thromboembolism associated with the Impella.

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