ABSTRACT

This article reviews the case of a 65-year-old patient with unstable L1 fracture after trauma. The fracture was treated via balloon kyphoplasty, shortly after which the patient developed shortness of breath and severe headache. Subsequent computed tomography (CT) of the head revealed subarachnoid hemorrhage. CT angiography did not reveal any intracranial aneurysms or arteriovenous malformations. A massive spinal subdural hematoma, which caused the patient to develop right leg paresis and hip joint weakness with grade 2–3, was found during magnetic resonance imaging (MRI). The hematoma was removed using multi-stage laminectomy Th5-L3. A follow-up MRI showed no pathological findings. Due to the unusual findings, spinal angiography was performed, revealing the artery of Adamkiewicz (A. radicularis magna, AKA) on the L1 level on the right side. Control CT showed a suboptimal insertion of the needle into the right pedicle, which caused the injury of the artery. AKA is present in the majority of the population, and surgical attention should be paid to avoid injury. Surgeons operating on the thoracolumbar spinal cord should have a thorough understanding of the anatomical features and surgical implications of this artery.

Keywords: Artery of Adamkiewicz; Kyphoplasty; Hematoma, subdural

INTRODUCTION

Vascular injuries are difficult to diagnose but common complications in spinal surgery. Neurological deficits after spinal surgery leading to impairment of the spinal blood supply are potentially serious complications. The artery of Adamkiewicz (AKA) is the main vessel responsible for supplying blood to the spinal cord from the 8th thoracic level up to the conus medullaris. Iatrogenic injury of this artery can cause neurologic damage manifesting postoperatively, such as fecal and urinary incontinence, and impaired motor function, although sensory function is usually preserved. We encountered a rare case of spinal subdural hematoma (SSH) after kyphoplasty (KP) and report this case with a literature review.
CASE REPORT

A 65-year-old patient was admitted to the department of neurosurgery after a fall. He denied taking any regular prescriptions including anticoagulants. An L1 burst fracture was identified and treated with a balloon KP for severe therapy-resistant pain.

The balloon KP was successful, and the patient reported no subsequent sensorimotor deficits. Postoperative computed tomography (CT) revealed minimal cement leakage into the spinal canal (FIGURE 1). Shortly thereafter, the patient developed shortness of breath and severe headache. No pulmonary embolism caused by bone cement in the vein was observed on chest CT scans, and D-dimer levels were normal.

Subsequent brain CT revealed subarachnoid hemorrhage, primarily prepontine. Brain CT angiography could not identify intracranial aneurysms or arteriovenous malformations. As spinal pathology was suspected to be the cause of the bleeding, magnetic resonance imaging (MRI) of the entire spine was performed. Spine MRI revealed a massive SSH (FIGURE 2). The patient had no neurological symptoms, such as paresis or sensory disturbances, but after 2 days, he developed right leg paresis and hip joint weakness with grade 2–3. It was decided that an immediate removal of the hematoma via multi-stage laminectomy on Th5-L3 was necessary, and a lumbar drainage was placed. Subsequently, the paresis resolved, but fecal and urinary incontinence occurred. Another spine MRI was performed, which did not reveal
any pathological findings. As this was abnormal, spinal angiography was performed, which demonstrated the AKA (A. radicularis magna) on the L1 level on the right side (FIGURE 3). A control CT revealed a suboptimal insertion of the needle into the right pedicle, which was responsible for the injury to the artery.

The patient was followed up and after 1 year. The control spine MRI showed multiple intraspinal cysts (FIGURE 4). The patient was incontinent and had hip flexor palsy and gait disturbance, and walking was only possible with a walker. The patient rejected a new surgery.

**DISCUSSION**

The present case describes an extreme rarity. Complications reported in the literature after KP are often related to cement extravasation into the epidural space, causing spinal cord compression or cement migration through the epidural veins to the venous system, leading
to pulmonary embolism. To date, only 6 other cases of spinal SSH have been reported. Previous studies reported on patients who developed spinal SSH after KP. The time for symptoms to develop varied from immediately after the operation to up to 2 weeks later. In most cases, surgery was performed when neurological deficits occurred. All patients recovered fully.

The AKA, also known as A. radicularis magna, supplies arterial blood to the spinal cord from T8 to the conus medullaris. Its origin is variable, and it extends from the mid-thoracic cavity to the lumbar spine. Normally, it arises from the abdominal aorta at the level of the ninth thoracic vertebrae, slightly higher on the left than on the right side, and is 0.8-1.3 mm in diameter. After passing through the intervertebral foramen, next to the exiting spinal nerve, it merges with the ventral root to the ventral (anterior) surface of the spinal cord, ascends and forms a so-called “hairpin” arch, and directs to the anterior spinal artery and flows into it. A review of 60 studies showed that AKA is present in 84.6% of the population, and most patients have a single AKA on the left side, originating between T8 and L1. The artery plays a major role in supplying blood to the spinal cord. Injury during surgery and, consequently, the lack of flow to this vessel can cause ischemia in the spinal cord.

In order to avoid neurological complications, anatomical examination of the AKA can be performed preoperatively using CT or magnetic resonance angiography. Care must be taken to differentiate the AKA from the anterior radiculomedullary vein by tracing the artery from the aorta.

In less than 1% of all cases, subarachnoid hemorrhages, which typically occur intracranially, can also originate from the spine. The etiologies include arteriovenous malformations and fistulas as well as isolated spinal artery aneurysms. Spinal subarachnoid hemorrhage should be considered in the differential diagnosis of patients with sudden onset of back pain, myelopathy, and radiculopathy.

In this case, the intracerebral reason for the subarachnoid hemorrhage was excluded by detailed brain diagnostics. In the following angiography, performed by a professional radiologist with extensive clinical experience, no active bleeding was diagnosed.

CONCLUSION

In conclusion, the current case is considered a complication caused by improper needle insertion during KP. This could be prevented by controlling the needle when inserting it in the anterior-posterior/lateral projection using C-arm images taken during the procedure. The needle must be inserted into the lateral part of the pedicle and in depth, following mandatory control with anterior-posterior images before insertion of the needle into the body of the vertebra. When an improper needle position in the anterior-posterior projection is suspected, the needle position should be corrected.

This complication is rare and requires further investigation. Good anatomical knowledge of the AKA is necessary to ensure safety of the spinal cord during surgery. Surgeons operating on the thoracolumbar spinal cord should have a thorough understanding of the anatomical features and surgical implications of this artery.
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