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Clinical note

A cervical anterior spinal artery syndrome after diagnostic blockade of the right C6-nerve root

Paul J.A.M. Brouwers[a,*], Ella J.B.L. Kottink[a], Marc A.M. Simon[b], Rik L. Prevo[c]

[a]*Department of Neurology, Medisch Spectrum Twente, P.O. Box 50.000, 7500 KA Enschede, The Netherlands*
[b]*Department of Anaesthesiology, Medisch Spectrum Twente, Enschede, The Netherlands*
[c]*Department of Neuroradiology, Medisch Spectrum Twente, Enschede, The Netherlands*

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Abstract

A 48-year-old man suffered from intractable neck pain irradiating to his right arm. Magnetic resonance imaging (MRI) of the cervical spine was unremarkable. A right-sided diagnostic C6-nerve root blockade was performed. Immediately following this seemingly uneventful procedure he developed a MRI-proven fatal cervical spinal cord infarction. We describe the blood supply of the cervical spinal cord and suggest that this infarction resulted from an impaired perfusion of the major feeding anterior radicular artery of the spinal cord, after local injection of iotrolan, bupivacaine, and triamcinolon-hexacetonide around the C6-nerve root on the right side. © 2001 International Association for the Study of Pain. Published by Elsevier Science B.V. All rights reserved.

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1. Case report

A 48-year-old man was admitted to hospital in August 1997. His medical history mentioned an infantile encephalopathy with a minor non-disabling hemiplegia on the left side. He was suffering from intractable pains in his neck irradiating to his right arm. Throughout the years he received several therapies to reduce his pains without any relief. Magnetic resonance image (MRI) of the cervical spine in 1995 was normal (Fig. 1A). Three months before admission a diagnostic C7-nerve root blockade on the right side partially reduced his pains for a few weeks. It was decided to try another right-sided diagnostic root blockade, this time of the C6-nerve root. Under fluoroscopic control, intervertebral foramina were visualized and a 22G needle was positioned in the posterior-caudal corner of the C6-foramen on the right side, using the 'tunnel vision' technique. The anterior-posterior fluoroscopic control showed that the tip of the needle was projected just over the lateral part of the facet column, well within the intervertebral foramen. Careful aspiration with a 2-ml syringe did not reveal any blood or cerebrospinal fluid. With injection of 0.2 ml iotrolan (Isovist 300®, Schering, The Netherlands) inside the root canal, fluoroscopy clearly showed spread of contrast medium along the C6-nerve root, confirming an adequate position of the needle. Then, a mixture of 0.5 ml bupivacaine 0.5% (Marcaine®, Astra Pharmaceutical, The Netherlands) and 0.5 ml triamcinolon-hexacetonide 2% (Lederspan®, AHP Pharma, The Netherlands) was slowly injected around the C6-nerve root in 1 min, again preceded by a negative attempt to draw blood or cerebrospinal fluid from the needle. Subsequently the needle was removed and within the next minute the patient was asked to resume a sitting position. At that moment, after this seemingly uneventful procedure, the patient acutely developed a flaccid paralysis and suffered from severe breathing difficulties without losing consciousness. He was immediately sedated and intubated, and artificial ventilation was started. Although his blood pressure remained within normal range, the attending anesthesiologist considered development of a total spinal block as an explanation, caused by intrathecal leakage of local anesthetics. Neurological examination after 30 min revealed a fully alert and normal orientated man, with normal function of the cranial nerves except for the accessory nerve. He was completely paralytic below the C3-level including trapezius and sternocleidomastoid muscles and diaphragm. All sensory modalities and deep tendon reflexes were lost below the C4-level. After 6 h no neurological recovery had occurred and cervical MRI revealed some increased signal intensity of the spinal cord from C2 to T1 (Fig. 1B). The patient was treated with high-

* Corresponding author. Tel.: +31-53-4872000; fax: +31-53-4872882.
E-mail address: mst.pjam.brouwers@wxs.nl (P.J.A.M. Brouwers).

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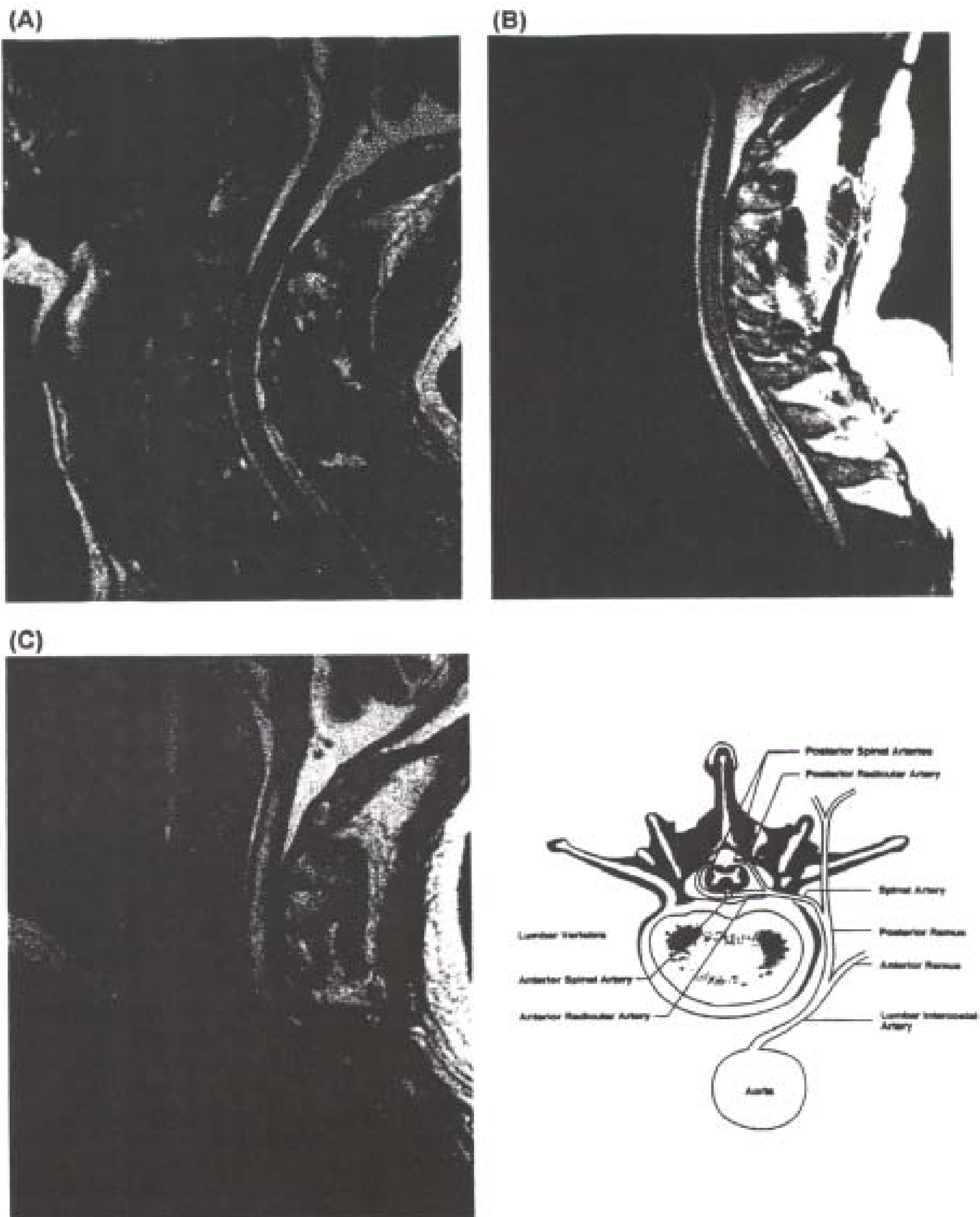

Fig. 1. Three T2-weighted MRI slices (300, 120, 3 mm). (A) (1995) shows a normal cervical myelum, (B) (6 h after the incident) shows mild swelling of the myelum with ventrally a linear shaped abnormal signal hyperintensity, and (C) (24 h after the incident) shows definite swelling and progression of the abnormal signal hyperintensity throughout the cervical myelum compatible with extensive infarction. (Diagram) Extrinsic arterial supply to the spinal cord. Radicular arteries at various levels join to form the discontinuous anterior spinal artery, which connects via the spinal arterial plexus to the pair of posterior spinal arteries. The anterior spinal artery arises rostrally from branches of the vertebral arteries and caudally from branches of the aorta. (Reproduced with kind permission from Cheshire et al., Neurology 1996, volume 47, pages 321–330).

dose methylprednisolone. The following day MRI showed extensive infarction of the spinal cord (Fig. 1C). During the next fourteen days, sense for light touch, position, and vibration returned completely without any improvement in motor performance, compatible with the clinical picture of a cervical anterior spinal artery syndrome. His hospital stay was complicated by numerous pneumonia's and in September he finally died after he developed a stomach perforation. Unfortunately, the family refused permission for a postmortem.

2. 2. Discussion

The anterior spinal artery syndrome is defined as complete motor paralysis with loss of pain and temperature sense, but sparing of position, vibration, and motion sense in the posterior columns. It occurs when the territory of the anterior spinal artery, supplying the ventral two-thirds of the spinal cord, is involved (Cheshire et al., 1996; Suh et al., 1996).

Spinal arteries follow nerve roots to the cord and then bifurcate into anterior and posterior radicular arteries. The anterior radicular arteries supply the anterior spinal artery, and the posterior radicular arteries feed the posterior spinal arteries (diagram). From the end of the fourth month of embryonic life, in general, one or two anterior radicular arteries remain effective in carrying blood to the anterior spinal artery, usually at the C6-level. The exact number of anterior radicular arteries and the level at which they enter the spinal canal varies greatly (Schoenen, 1991). Infarction due to interruption of a major anterior radicular artery is clinically indistinguishable from the anterior spinal artery syndrome (Cheshire et al., 1996; Suh et al., 1996).

An acute perfusion problem of the cervical spinal cord occurs infrequently and may have several rare causes ranging from frank atlanto-occipital dislocation to mere sneezing. Iatrogenic etiologies to interrupt perfusion to the anterior spinal artery or its supplying vessels are quite rare and have included isolated reports related to vertebral arteriography, thoracolumbar sympathectomy, celiac plexus block, abdominal arteriography, subclavian vein catheterization, lumbar epidural anesthesia during labour, therapeutic renal artery embolization, single radicular artery ligation, thoracoplasty, pneumonectomy, intra-aortic balloon pump counterpulsation, porto-caval shunt placement, intrathecal lidocaine or phenol, hypotension and cardiac surgery (Cheshire et al., 1996; Humphrey, 1992).

The clinical and radiological picture in our patient was compatible with an anterior spinal artery syndrome caused by a large spinal cord infarction (Suh et al., 1996; Cheshire et al., 1996). We suggest that this infarction was the result of an impaired perfusion of the major feeding anterior radicular artery, after local injection of iotrolan, bupivacaine, and triamcinolon-hexacetonide around the C6-nerve root on the right side. The cause of this perfusion problem remains unclear. We encountered no difficulties during the procedure. A negative attempt to draw blood or cerebrospinal fluid from the needle, together with spread of contrast medium along the C6-nerve root, excluded at that moment, an intravascular injection with iotrolan. The needle was not moved any further, and a second attempt to draw blood or cerebrospinal fluid was again negative, which makes a subsequent intravascular injection with bupivacaine and triamcinolon-hexacetonide very unlikely. On the other hand we cannot totally exclude any movement of the needle between the two injections. May be the anterior radicular artery was manipulated, causing a dissection or spastic reaction leading to occlusion of the vessel. Bupivacaine was not mixed with adrenaline, therefore adrenaline as a cause of vasoconstriction could be excluded. Hypotension did not occur.

With this case report we would like to draw attention to this very serious complication of a diagnostic cervical nerve root blockade, and probably also of therapeutic cervical nerve root blockades, especially at the C6-level on the right side. In most patients collateral supply of the cervical spinal cord is obviously sufficient but as our case shows, sometimes there is one major feeding cervical anterior radicular artery only. If this specific artery becomes iatrogenically occluded by a presumably spastic reaction or dissection on local injection, a devastating complication may be the result.

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