Paraplegia after contrast media application: a transient or devastating rare complication? Case report

Dorothee Mielke, MD,1 Kai Kallenberg, MD,2 Marius Hartmann, MD,3 and Veit Rohde, MD1

Departments of 1Neurosurgery and 2Neuroradiology, Georg-August-University, Göttingen; and 3Department of Neuroradiology, HELIOS Hospital Berlin-Buch, Berlin, Germany

The authors report the case of a 76-year-old man with a spinal dural arteriovenous fistula. The patient suffered from sudden repeated reversible paraplegia after spinal digital subtraction angiography as well as CT angiography. Neurotoxicity of contrast media (CM) is the most probable cause for this repeated short-lasting paraplegia.

Intolerance to toxicity of CM to the vulnerable spinal cord is rare, and probably depends on the individual patient. This phenomenon is transient and can occur after both intraarterial and intravenous CM application.

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KEY WORDS contrast media application; paraplegia; complication; digital subtraction angiography; CT angiography

Case Report

History and Examination

A 76-year-old man, a general practitioner, reported increasing gait disturbances due to intermittent weakness of his legs for the 3 months prior to presentation. Lumbar or radicular pain was denied. His walking distance was reduced to 100 m. Clinical examination revealed a mild proximal paraparesis of his legs, with a muscle strength of Grade 4 according to the Medical Research Council (MRC) scale. No distal paresis, sensory deficits, signs of myelopathy, or vegetative deficits were found.

Neuroimaging Findings

An MRI study of the lumbar spine raised suspicion of a spinal dural arteriovenous fistula (dAVF). The patient underwent an uneventful spinal DSA with a total dose of 100 ml of the nonionic contrast medium iomeprol (Imeron 300 [30 g iodine/100 ml, trometamol, hydrochloric acid, and water for injection], Bracco Imaging Germany). The DSA confirmed a spinal dAVF arising from the left L-1 radiculomeningeal artery (Fig. 1). Within 1 hour after the DSA, the patient developed a paraplegia of his legs. An MRI
by the time of presentation at our clinic the patient was experiencing a mild proximal paraparesis of his legs, with a muscle strength of Grade 4 according to the MRC scale. Furthermore, he had a sensory deficit including both proximal thighs. To better delineate the relation of the dAVF to the bony structures of the spine, CTA, with intravenous injection of 80 ml of the nonionic contrast medium iomeprol (Imeron 400 [40 g iodine/100 ml, trometamol, hydrochloric acid, and water for injection], Bracco Imaging Germany), was performed. Again the patient developed a complete paraplegia of his legs. Furthermore, he had a complete hypesthesia below the L-1 level as well as an atonic sphincter muscle.

Study of the spine was performed immediately, and excluded intramedullary bleeding, ischemia, and progressive dAVF-related venous congestion (Fig. 2). The paraplegia resolved completely within 24 hours under dexamethasone medication. Surgery for the dAVF was scheduled to be performed 3 weeks after DSA. To shorten the waiting time, the patient presented at our institution 17 days after the first DSA.

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after the application of dexamethasone, which had already been started prior to the examination. Furthermore, the patient received intravenous injections of 4 mg clemastine and 250 mg prednisolone (once each).

Operation and Postoperative Course

Three days later, the fistula was ligated microsurgically via a hemilaminectomy at the L-1 level on the left side. Surgery was uneventful. The patient demonstrated no new neurological deficit postoperatively. The preoperative gait disturbance as well as sensory deficits were already in decline by the time of discharge. So far, the follow-up period is 9 months. The patient still reports unspecified sensory deficits of the left leg. Furthermore, the gait disturbance is still present, but has clearly improved during the last months. Prior to the operation the patient was able to walk only a couple of steps without assistance. At the moment he does not need any assistance for walking, but still reports episodes of stumbling.

Discussion

Possible Pathomechanisms of Neurological Symptoms After Cerebral Angiography

Short-term transient neurological symptoms as well as fully reversible but long-lasting seizures, cortical blindness, and encephalopathy have been reported after cerebral DSA with intraarterial injection of contrast media (CM). Four major pathomechanisms have been proposed to explain these neurological deficits, as follows.

1) Direct displacement of blood by the CM lasts only seconds, but might produce enough tissue hypoxia to alter brain function transiently in some cases. 2) High concentrations of CM cause aggregation and clumping of red blood cells, leading to local stasis and occlusion of small arterial branches. 3) Conventional iodinated CM have high affinities for plasma water, low affinities for plasma proteins, and extremely low partition coefficients. Normally, CM do not penetrate an intact blood-brain barrier (BBB). However, it has been shown in animal models that the hyperosmolarity of intraarterially injected ionic CM can cause BBB breakdown, allowing CM to leak into brain tissue and exert their chemotoxic property. Possibly,
the chemotoxic effects are enhanced by red blood cell aggregation, by induction of ischemia (contributing to BBB breakdown), and by prolongation of the CM transit time. It has been shown that the effect of local ischemia, induced either by mere CM injection or by red blood cell aggregation, might be intensified if underlying vascular disease of the brain has already caused a situation of hyperperfusion or ischemia.

Possible Pathomechanisms of Neurological Symptoms After Cerebral CTA

In CTA, intravenous instead of intraarterial CM are being applied. Complete recovery of neurological deficits within hours to days after CTA of the cerebral vessels is much more rare than after DSA, but has been reported. Junck and Marshall report that the peak osmolality of blood plus CM perfusing the CNS is not nearly as high as with arteriography, but that the osmolality remains elevated for a longer time, which might lead to an opening of the BBB and leakage of the chemotoxic CM into the brain parenchyma. They further suggest that the barrier defect is being repaired while the concentration of CM remains high; thus the CM may be unable to diffuse freely back into the blood and their clearance from the CNS may be prolonged, with a subsequent risk for prolonged chemotoxic effects.

Neurological Deficits After Spinal DSA and CTA?

A permanent neurological deficit after diagnostic spinal DSA with CM injection into the radiculomeningeal artery has been described only once: Oumerzouk et al. reported on the case of a 73-year-old man with sudden worsening of a preexisting paraparesis after spinal DSA. This patient harbored a dAVF as well. The patient underwent surgery, but unfortunately the paraplegia remained, with only slight improvement. The authors suggested a sudden increase of venous pressure as the cause for this devastating complication.

Transient neurological complications after spinal DSA have not been described yet; in a retrospective study reviewing 302 spinal angiographies, no neurological complications were encountered and no case reports were retrievable. The same holds true with spinal CTA; a neurological deficit after spinal CTA has never been mentioned in the international literature. Thus, our case report appears to be unique. Because this complication is fully reversible and also occurs after intravenous CM application, not after the sudden increase of the spinal venous pressure, the causative pathomechanisms are probably similar to those assumed in cerebral DSA with transient neurological symptoms. Because of this assertion, the occurrence of this complication after spinal DSA and CTA should not be regarded as being completely unexpected. The dAVF led to focal intramedullary edema as seen on MRI (Fig. 2), which can be taken as a sign for an already disturbed BBB. Thus, CM could have easily entered the spinal cord, with a subsequent local chemotoxic effect that did not resolve until the CM were being washed out. Contrast media can cause a combination of excitatory effects associated with their chemical nature and inhibitory effects associated with their hyperosmolality. In our case, the patient demonstrated only inhibitory effects.

Conclusions

Transient paraplegia after intraarterial as well as intravenous application of CM for spinal DSA and CTA has never been reported before. Because CM neurotoxicity together with a disturbed BBB is the most probable cause, the already reported complications after cerebral DSA and CTA suggest that the uniqueness of the present case is related to the considerably less-frequent need for spinal DSA and spinal CTA. Thus, despite being unique up to now, we strongly believe that this complication will repeatedly occur with the increasing use of spinal DSA and CTA in the future. Therefore, indications for CM application in spinal diseases should be carefully selected, especially if a neurological deficit related to intravascular CM application has already occurred.

References


Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Mielke. Acquisition of data: Mielke. Analysis and interpretation of data: Mielke, Kallenberg, Rohde. Drafting the article: Mielke. Critically revising the article: Mielke, Hartmann, Rohde. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Mielke. Administrative/technical/material support: Kallenberg, Hartmann.

Correspondence

Dorothee Mielke, Department of Neurosurgery, Georg-August-University Göttingen, Robert-Koch-Strasse 40, 37075 Göttingen, Germany. Email: dorothee.mielke@med.uni-goettingen.de.