Spinal Cord Infarction Complicating Embolisation of Vertebral Metastasis

A Result of Masking of a Spinal Artery by a High-Flow Lesion

H.J. CLOFT*, M.E. JENSEN, H.M. DO, D.F. KALLMES

Department of Radiology, University of Virginia Health Sciences Center; Charlottesville

* Department of Radiology, Emory University; Atlanta

Key words: arteries, therapeutic blockade, spine, secondary neoplasms, spine, interventional procedures, kidney neoplasms, metastases

Summary

A 70-year-old woman presented with severe back pain secondary to metastasis of renal cell carcinoma to the second lumbar vertebral body. She had no evidence of spinal cord compression clinically or on MR imaging. Tumour embolisation was performed for pain relief. The embolisation was complicated by spinal cord infarction resulting from angiographic masking of a spinal artery by diversion of contrast material into the high-flow tumour.

Introduction

Preoperative and palliative embolisation of vertebral tumours is a well described technique¹⁻¹⁰. While serious complications from such embolisations are relatively uncommon, catastrophic injury to the spinal cord can occur.

We describe a case in which embolisation was complicated by spinal cord infarction resulting from angiographic masking of a spinal artery by diversion of contrast material into the high-flow tumour.

Case Report

A 70-year-old woman presented with severe back pain radiating to the left thigh secondary to local extension of renal cell carcinoma into

the left side of the second lumbar vertebral body. She had no evidence of spinal cord compression clinically or on MR imaging. The patient was considered not to be a candidate for surgery because her spine was not unstable and she had advanced liver and lung metastases. It was decided to perform embolisation of the tumour for pain relief.

Spinal angiography (left T11, T12, L1, and L2, and right L1 and L2 segmental arteries) showed that the tumour was fed primarily by the left first and second lumbar arteries. No spinal arteries were seen on pre-embolisation angiography.

The dorsal branch of the left first lumbar artery was superselectively catheterized with a Tracker 18 microcatheter (Target Therapeutics/Boston Scientific Corporation, Natick, MA). Superselective angiography showed dense tumour blush in the second lumbar vertebral body. A spinal branch was not angiographically demonstrated, and provocative testing was performed with intra-arterial injection of 1.25 ml of 2% lidocaine, which caused no change in her lower extremity strength or sensation.

The dorsal branch of the first lumbar artery was embolised with 150-350 micron polyvinyl acetate particles suspended in a 3 : 1 mixture of iohexol (Omnipaque 300, Nycomed, New York, NY): 3% Sotradecol (Elkins-Sinn, Cherry Lake,

NJ), which was painless for the patient and uneventful.

The dorsal branch of the left second lumbar artery was then superselectively catheterized with the Tracker 18 microcatheter. Superselective angiography with rapid hand injection showed dense tumour blush at the left lateral aspect of the second lumbar vertebra, and no spinal branch was demonstrated (figure 1A). In an attempt to achieve a more selective embolisation, the microcatheter was advanced further into the branch and a second angiogram was performed, which again demonstrated dense tumour blush and no spinal branch (figure 1B).

Because it did not appear that embolisation from the more distal microcatheter position would spare any significant normal vessels angiographically, and because we were concerned that embolising from this position might some more proximal feeders to the tumour to not be embolised, we decided to embolise from the more proximal position used in the first superselective angiogram. Provocative testing was performed with intra-arterial injection 2.5 ml of 2% lidocaine, which caused no change in her lower extremity strength or sensation. A higher dose of lidocaine was used on the second lumbar artery because the vessel fed a high-flow lesion. The dorsal branch of the second lumbar artery was then embolised with the same mixture used to embolise the first lumbar artery. Embolisation was performed under continuous fluoroscopic monitoring with road map assistance.

The embolisation was stopped after administration of 1.0 ml of the embolic material because most of the tumour blush was seen to have been eliminated on fluoroscopy, and a post-embolisation angiogram was performed. The post-embolisation angiogram demonstrated a large anterior radiculo-medullary artery (figure 1B), which had been masked by diversion of contrast through the high-flow tumour on the pre-embolisation angiogram. Most of the tumour blush was eliminated by the embolisation. Immediately following performance of the control angiogram, the patient complained of severe pain in her lower back and in both legs. Because of her pain and the fact that we were concerned that the anterior spinal artery had been embolised, we decided to terminate the procedure.

Immediately following the procedure, she could move her legs. Her strength in both legs appeared reduced, but she did not seem to be cooperating well with the neurological exam because of her pain and drowsiness caused by narcotic pain medication. Over the next twelve hours, the pain subsided and she progressively developed nearly complete paralysis and anesthesia below the waist, as well as bowel and bladder incontinence. MR imaging performed three hours following embolisation showed central high signal intensity on T2-weighted images of the lower spinal cord, consistent with infarction (figure 1C).

Discussion

Preoperative embolisation for vertebral tumours is a relatively safe procedure which reduces surgical blood loss, making surgery safer and easier¹⁻⁸. Palliative embolisation to relieve pain from vertebral tumours has also been described^{9,10}. We reviewed the reports of 153 preoperative and palliative embolisation procedures for vertebral tumours in the literature¹⁻¹⁰, and found that there were only two reported complications.

These complications were a dermatomal parasthesia¹⁰, and a transient myelopathy due to tumour swelling which was relieved by surgery². One must, however, use caution when interpreting data regarding the overall complication rate from this compilation of studies, since they span a twenty year period from 1975 to 1995, and a wide variety of techniques were used. Nonetheless, these series indicate that the overall complication rate for embolisation of spinal metastases is rather low (1-2%).

The flow-controlled technique used in our case and now generally used for tumour embolisations was first described by Kerber¹¹. The basic principle of this technique is to not occlude the artery with the catheter and to allow the perfusion pressures and flow to carry the embolic material distally into the abnormality being treated.

This technique avoids ischaemic complications of embolisation secondary to wedging the catheter in the artery and pushing the embolic material into normal arteries through anastomoses that are forced open when normal perfusion pressure is exceeded by the pressure of injection. However, the flow-controlled tech-

Figure 1 A) Superselective angiography in anteroposterior projection shows dense tumour blush, and no spinal branch is demonstrated. B) Superselective angiography with the catheter located more distal in an attempt to achieve more selective tumour embolisation again shows dense tumour blush, and no spinal branch is demonstrated. C) The post-embolisation angiogram demonstrates a large anterior radiculo-medullary artery (arrow), which had been masked by diversion of contrast through the high-flow tumour. Most of the tumour blush was eliminated by the embolisation. D) MR imaging performed three hours following embolisation shows central high signal intensity on sagittal T2-weighted image of the lower spinal cord (arrow), consistent with infarction. Note metastatic tumour in the second lumbar vertebral body (curved arrow).

nique can lead to complications when, as in our case, the flow into a high-flow lesion is eliminated by embolisation, and the embolic material then flows to normal tissues which were previously angiographically occult.

This risk can be minimized by performing angiograms frequently during the course of embolisation of lesions with high flow, to monitor for diversion of blood flow to normal tissues as flow to the lesion is eliminated.

Provocative testing with barbiturate and lidocaine has been recommended when performing spinal embolisations¹¹. In our case, provocative testing with lidocaine did not prevent embolisation of the anterior spinal artery. The lidocaine which was used for provocative testing in our case simply shunted rapidly through the tumour and into the venous system without entering the spinal artery, as did the contrast on the pre-embolisation angiogram. While a barbiturate such as amobarbital or methohexital may be useful in provocative testing of anterior spinal artery branches which supply spinal cord gray matter, it is doubtful that barbiturate testing would have been helpful in this case, since it would almost certainly have been shunted rapidly through the tumour, and therefore would not have reached the anterior radiculo-medullary artery.

Also, lidocaine alone may be adequate for provocative testing of spinal arteries, as a study in monkeys demonstrated a brief period of flaccidity followed by hyperreflexia in the lower extremities and fasciculations of the thigh muscles when lidocaine was injected into the artery of Adamkiewicz¹².

We did not localize the artery of Adamkiewicz on the spinal angiogram prior to embolisation. One might argue that embolisation of the anterior spinal artery can be avoided by localizing the artery of Adamkiewicz angiographically. If we had aggressively pursued the artery of Adamkiewicz angiographically and not found it, perhaps we would have been concerned that it was masked by tumour shunting. However, the blood supply to the spinal cord is highly variable, and finding a large anterior radiculo-medullary artery does not exclude the presence of a second anterior radiculo-medullary artery.

Since the large anterior radiculo-medullary artery that we embolised at the second lumbar level caused an infarction of only the lower

spinal cord, it is extremely likely that we would have found a higher anterior radiculo-medullary artery that supplies most of the thoracic spinal cord, had we aggressively pursued it angiographically.

It is, therefore, doubtful that this complication would have been avoided by attempting to localize the artery of Adamkiewicz angiographically.

We used Sotradecol in our embolic mixture to cause sclerosis of the tumour vessels and subsequent tumour necrosis¹³. The intra-arterial use of Sotradecol rather than ethanol has the advantage of being relatively painless during the procedure. Ethanol causes severe pain when injected intra-arterially, necessitating the use of general anesthesia. The use of a liquid agent such as Sotradecol or ethanol facilitates results in tumour necrosis, but also carries a higher risk of necrosis of normal tissues, including the spinal cord.

The amount of spinal cord infarction might have been less if we had used the 150-250 micron polyvinyl alcohol particles alone, but embolisation with these particles alone probably would not cause significant tumour necrosis, and therefore might have resulted in both a lesser degree and shorter duration of pain relief.

Unfortunately, not enough data is available in the literature to definitively discern the relative risks and benefits of the various agents available for tumour embolisation. In pre-operative embolisation where tumour necrosis is not a goal, liquid agents and small particles should be avoided to avoid ischaemia of normal tissues, including the spinal cord.

Embolisation of tumours with N-butyl cyanoacrylate and ethanol via direct percutaneous puncture has been proposed as an alternative to trans-arterial embolisation for pain relief^{14,15}. Percutaneous vertebroplasty with methyl methacrylate has also been used successfully to relieve pain from vertebral metastases¹⁵. These direct puncture techniques may result in less risk of embolisation of normal arteries, including spinal arteries. However, vertebroplasty would not have been a good option in our case, because the patient had symptoms from radicular involvement, which would not have been relieved by and could have been made worse by injection of methyl methacrylate.

In summary, embolisation of spinal lesions

with high flow can be complicated by spinal cord infarction resulting from angiographic masking of a spinal artery by the diversion of contrast into the high-flow lesion. While it may be impossible to completely eliminate the risk of spinal cord infarction in these lesions, awareness of this potential complication allows one to be vigilant in looking for unmasked spinal arteries as the embolisation proceeds, and thereby minimize injury to the spinal cord. It

might also prompt one to embolise lesions with high flow with larger particles. Liquid agents and small particles should especially be avoided in pre-operative embolisations where it is not necessary to cause tumour necrosis.

Embolisation via direct puncture technique may also be a safer option in these lesions. It is also important to be aware that provocative testing does not screen for this potential complication.

References

- 1 Breslau J, Eskridge JM: Preoperative embolisation of spinal tumours. *JVIR* 6: 871-875, 1995.
- 2 Smith TP, Gray L et Al: Preoperative transarterial embolisation of spinal column neoplasms. *JVIR* 6: 863-869, 1995.
- 3 Hilal SK, Michelsen JW: Therapeutic percutaneous embolisation for extra-axial vascular lesions of the head, neck, and spine. *J Neurosurg* 43: 275-287, 1975.
- 4 Broaddus WC, Grady MS et Al: Preoperative superselective arteriolar embolisation: a new approach to enhance resectability of spinal tumours. *Neurosurgery* 27: 755-759, 1980.
- 5 Bhojraj SY, Dandawate AV, Ramakantan R: Preoperative embolisation, transpedicular decompression and posterior stabilisation for metastatic disease of the thoracic spine causing paraplegia. *Paraplegia* 30: 292-299, 1992.
- 6 Olerud C, Jónsson H et Al: Embolisation of spinal metastases reduces preoperative blood loss: 21 patients operated on for renal cell carcinoma. *Acta Orthop Scand* 64: 9-12, 1993.
- 7 Sundaresan N, Choi IS et Al: Treatment of spinal metastases from kidney cancer by presurgical embolisation and resection. *J Neurosurg* 73: 548-554, 1990.
- 8 Gellad FE, Sadato N et Al: Vascular metastatic lesions of the spine: preoperative embolisation. *Radiology* 176: 683-686, 1990.
- 9 O'Reilly GV, Kleefield J et Al: Embolisation of solitary spinal metastases from renal cell carcinoma: alternative therapy for spinal cord or nerve root compression. *Surg Neurol* 31: 268-271, 1989.
- 10 Soo CS, Wallace S et Al: Lumbar artery embolisation in cancer patients. *Radiology* 45: 655-659, 1982.
- 11 Kerber CW: Flow-controlled therapeutic embolisation: a physiologic and safe technique. *Am J Neurorad* 1: 77-81, 1980.
- 12 Doppman JL, Girton M, Oldfield EH: Spinal Wada test. *Radiology* 161: 319-321, 1986.
- 13 Cho KJ, Williams DM et Al: Transcatheter embolisation with sodium tetradecyl sulfate. Experimental and clinical results. *Radiology* 153: 95-99, 1984.
- 14 Casasco A, Herbeteau D et Al: Devascularization of craniofacial tumours by percutaneous puncture. *Am J Neurorad* 15: 1233-1239, 1994.
- 15 Gangi A, Dietemann JL et Al: Interventional radiologic procedures with CT guidance in cancer pain management. *Radiographics* 16: 1289-1304, 1996.

Harry J. Cloft, M.D., Ph.D.
Department of Radiology
Emory University
1364 Clifton Rd. N.E.
Atlanta, GA 30322
E-mail: harry.cloft@emory.org