Spondylitic Spinal Stenosis

To the Editor: The article by Weinstein and colleagues (Weinstein PR, Karpman RR, Gall EP, et al: Spinal cord injury, spinal fracture, and spinal stenosis in ankylosing spondylitis. J Neurosurg 57:609-616, November, 1982) regarding lesions causing spinal cord injury in ankylosing spondylitis (AS) was extremely comprehensive and informative. However, their contention that “spinal stenosis” has not been previously described as a contributing cause of spinal cord compression in AS is inaccurate. Good, et al., published a case report of a man with progressive paraparesis and extradural myelographic block at the T-8 level who recovered excellent function following decompressive laminectomy and removal of hypertrophied adipose tissue from the posterior extradural space. On pathological examination, this adipose tissue revealed foci of chronic inflammatory cells. This patient also had destructive changes of the disc space adjacent to the block, but had no evidence of spinal fracture. The cases of spinal stenosis reported by Weinstein, et al., were diagnosed by computerized tomography following acute spinal fractures, and no comment was made on operative findings in the three patients who underwent decompressive laminectomies. Although the authors were reticent about concluding that the spinal stenosis in their patients was a “dysplastic narrowing of the neural canal as a manifestation of inflammatory and proliferative processes in AS,” the case report of Good, et al., would lend some credence to that very hypothesis. Even though it may represent a rare clinical manifestation, the case of Good, et al., does demonstrate the potential harmful chronic effects of spondylitic spinal stenosis, which can be reversed by appropriate diagnosis and surgical treatment.

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Reference

Response: Dr. Keller kindly draws our attention to the article by Good, et al., that was published subsequent to submission of our paper to the Journal of Neurosurgery. These authors reported a case of a paraparesis associated with myelographic block which was due to hypertrophy and inflammation in the epidural soft tissues adjacent to the site of thoracic spine pseudarthrosis. Their patient was only later found to have ankylosing spondylitis (AS) as determined by radiographic and clinical evidence of sacroiliac joint arthritis. They have presented an interesting example of another complication of this disease that can occur in association with pseudarthrosis, even when ankylosis of the spinal column has not yet developed. It is perhaps to be associated with the development of caudal radiculopathy in those patients with AS who also develop cystic dilatation of the lumbosacral thecal sac. Unfortunately, these patients do not improve following surgical decompression as did the patient described by Good, et al.,

I disagree with Dr. Keller's view that his case is, in fact, an example of spinal stenosis due to or associated with AS. Spinal stenosis is defined as narrowing of the spinal canal due to developmental dysplasia of the neural arch, often with soft-tissue hypertrophy present as a contributory factor. The cases we reported had typical short pedicles and laminae. Spinal cord compression by epidural tumor, abscess, or (as described in the case at issue) inflammation associated with ankylosing spondylodiscitis would not ordinarily be classified as spinal stenosis. Operative findings in our two fracture cases included typical ligamentous ossification. In the one patient operated on for decompression of thoracic myelopathy, spinal stenosis with hypertrophy of bone and ligaments was found, with no evidence of inflammation or enlargement of the epidural fat layer. Although pseudarthrosis was present, there was no evidence of intraspinal callus formation.

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Balloon Embolus

To the Editor: I was extremely interested in the complication of the detachable balloon technique as related by Chalif, et al. (Chalif DJ, Flamm ES, Berenstein A, et al: Microsurgical removal of a balloon embolus to the internal carotid artery. Case report. J Neurosurg 58:112-116, January, 1983). It is my belief that this complication could happen to anyone who is treating a traumatic carotid cavernous fistula with detachable balloons, although I have not personally encountered it. I do not agree, however, with the explanation given by the authors. I think that is is nearly impossible for a balloon which is already detached in the cavernous sinus to dislodge and embolize the bifurcation of the internal carotid artery (ICA). In all likelihood, the balloon was detached in the carotid siphon at the level of the fistula and lost part of its contrast