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Iatrogenic Femoral Pseudoaneurysms

From:

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Editor:

We would like to comment on the recently published article by Drs Sheiman and Brophy (1). The authors describe a 100% success rate of percutaneous thrombin injection for the treatment of simple pseudoaneurysms, whereas their success rate for complex pseudoaneurysms was only 56% (five of nine pseudoaneurysms). Complex pseudoaneurysm architecture was associated with a procedure failure. Because of realistic concern about possible native vessel thrombosis or embolization if the lobe directly connected to the native vessel was to be injected, Drs Sheiman and Brophy elected to inject the farthest removed lobe first and then perform a second injection in the directly connected lobe if persistent or recurrent flow was observed. It is noted that ". . . we cannot exclude the possibility that direct injection into this lobe (directly joined to the native vessel) could cause thrombosis, eliminate flow in the more distal lobe, and lead to total spontaneous thrombosis of the entire pseudoaneurysm."

Comparison of this report with our recent experience with 14 patients who had iatrogenic femoral pseudoaneurysms may be useful in helping to determine the most appropriate technique for this emerging new therapy. We performed percutaneous topical thrombin (Thrombin-JMI; Jones Medical Industries, St Louis, Mo) injection after obtaining local institutional review board approval. All patients failed a trial of nonguided external compression that was continued for a minimum of 2 and a maximum of 9 days. Heparin therapy was discontinued prior to thrombin injection but was recommenced immediately afterward in four cases. Four of the 14 patients had a complex pseudoaneurysm, three of whom had two interconnecting lobes, one had four interconnecting lobes, and all had a single neck. None of these patients were receiving anticoagulant therapy, but two were receiving antiplatelet drugs. The median pseudoaneurysm volume of 7.5 cm³ (range, 5–12 cm³) in the four complex pseudoaneurysms was lower than that in the cases just described (mean \pm SD, 23.3 cm³ \pm 12.8). The maximal complex pseudoaneurysm diameter in our series was 5 cm (range, 2.5–5.0 cm).

Despite many similarities between our technique and that of Drs Sheiman and Brophy (1), there are some important differences. First, from the outset of our experience, we chose to use a low concentration of bovine thrombin (ie, 200 units/mL saline). This decision was based on the results re-

cently published by Taylor et al (2), who reported an overall success rate of 93% with an average dose of 300 units of thrombin. In addition, use of a lower dose of thrombin may potentially reduce the likelihood of complications. At this time, the minimal necessary thrombin dose to achieve pseudoaneurysm thrombosis is the subject of ongoing clinical research. Using the same logic that was just hypothesized, we elected to inject the cavity adjoining the native vessel in all four cases. Immediate thrombosis of all lobes was observed with the injection of an average of 200 units (1 mL) of thrombin.

No complications occurred, and no recurrence of pseudoaneurysm was demonstrated after an average follow-up of 9 months (range, 5–12 months). It is possible that small sample numbers introduced bias into our results. The difference in the pseudoaneurysm volume in the two reports may explain why a smaller dose of thrombin was effective in our series (200 units [1 mL] vs minimum of 1,500 units). It is not clear from the report by Drs Sheiman and Brophy what dilution of thrombin was used, and, consequently, what volume was necessary to achieve thrombosis.

Thus far, we have not encountered any technical difficulty in inserting the needle into the cavity nearest the native vessel by using continuous ultrasonographic guidance. The needle tip and the injection are directed away from the neck of the pseudoaneurysm, and injection is continued until thrombosis occurs. The volume range of the simple pseudoaneurysms in the remaining 10 patients in our series varied from 5 to 20 cm³ (median, 8.5 cm³). This volume range is also smaller than that in the Sheiman and Brophy report (6.4–53.0 cm³; mean, 15.8 cm³ \pm 10.4). However, our maximal thrombin dosage was 600 units (mean, 250 units) as opposed to 1,000 units (1). We had nine of 10 procedural successes, for an overall success rate of 93% (13 of 14 successes). The single failure occurred in a patient who received warfarin sodium (Coumadin) therapy and in whom a second thrombin injection after 48 hours also failed. The patient underwent surgical repair. A simple pseudoaneurysm with a single neck was identified.

It may be that the trade-off between theoretical safety of the technique proposed by Drs Sheiman and Brophy is obviated by the theoretical additional risk of a second thrombin injection. We propose that injection of the lobe nearest to the native vessel is no more risky than injection into any simple pseudoaneurysm. Undoubtedly, as the frequency of this new therapy increases, the incidence of complications will also increase. However, controlled image-guided injection into the nearest lobe of a complex pseudoaneurysm, with the lowest possible thrombin dose, appears to be a reasonable approach.

References

1. Sheiman RG, Brophy DP. Treatment of iatrogenic femoral pseudoaneurysms with percutaneous thrombin injection: experience in 54 patients. *Radiology* 2001; 219:123–127.
2. Taylor BS, Rhee RY, Muluk S, et al. Thrombin injection versus compression of femoral artery pseudoaneurysms. *J Vasc Surg* 1999; 30:1052–1059.

Drs Sheiman and Brophy respond:

We are in agreement with Dr Bloom that the technique for the treatment of iatrogenic femoral pseudoaneurysms with thrombin has not yet been optimized and thank him for contributing his experience. However, on the basis of the data presented, we cannot agree with the recommendation that performing image-guided injection into the cavity of a complex pseudoaneurysm directly joined to the native vessel is reasonable. First, the median volume of Dr Bloom's four complex pseudoaneurysms (7.5 cm³) was approximately one-third that of ours (20.0 cm³). Hence, the volumetric flow encountered in our complex pseudoaneurysms was nearly three times as great. The lack of complications from the injection of thrombin into the lobe directly in contact with the native vessel in his four cases does not necessarily extrapolate into a low acceptable complication rate for our cases or for this technique in general.

Additionally, Dr Bloom does not formally present his definition of a complex pseudoaneurysm. A clear distinction between a pseudoaneurysm that we formally define as complex (multiple compartments separated by a patent tract, which has a diameter smaller than the minimal dimension of the smallest lobe) and one, for example, that has a single lobe but is potentially considered complex due to multiple septations must be made. Although Dr Bloom's proposal may turn out to be correct, he cannot advocate this approach on the basis of experience with four small complex pseudoaneurysms (at least two of which, per data published at our institution [1], could potentially thrombose spontaneously). Presently, the theoretical safety offered by our technique has been successfully applied to 11 additional complex pseudoaneurysms (all with total volumes exceeding 6 cm³) without complication. Therefore, the theoretical additional risk of a second injection does not appear to be an issue.

Dr Bloom also required lower thrombin doses for the successful treatment of his four complex and 10 simple pseudoaneurysms, when compared with those in our cases. He rightfully identifies that this difference may be the result of his smaller pseudoaneurysm volumes. Indeed, Kang et al (2) found a direct relationship between pseudoaneurysm size and the dose required for obliteration. This has also been our experience, though other factors such as patient coagulation status and blood pressure likely play a role. However, there is implication in his letter that the larger doses of thrombin used in our study may not be warranted. We point out that the proposal by Dr Bloom to use the lowest possible thrombin dose for successful pseudoaneurysm treatment is a given and one that we have adhered and continue to adhere to.

Use of lower thrombin doses for pseudoaneurysm treatment in general and treatment of a complex pseudoaneurysm by means of injection into the lobe that is in direct communication with the native vessel may be shown to be optimal with future research. However, we do not believe that either technique can currently be conclusive on the basis of Dr Bloom's experience with the small number and size of the pseudoaneurysms he presents. A technique that advocates needle placement far away from the pseudoaneurysm neck while maintaining success should still be favored at this time.

References

1. Kent KC, McArdle CR, Kennedy B, Baim DS, Anninos E, Skillman JJ. A prospective study of the clinical outcome of femoral pseudoaneurysms and arteriovenous fistulas induced by arterial puncture. *J Vasc Surg* 1993; 17:125-133.
2. Kang SS, Labropoulos N, Mansour A, Baker WH. Percutaneous ultrasound guided thrombin injection: a new method for treating postcatheterization femoral pseudoaneurysms. *J Vasc Surg* 1998; 27:1032-1038.

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Schmorl Nodes: Lack of Relationship between Degenerative Changes and Osteopenia

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Editor:

We have read with interest the article by Drs Pfirrmann and Resnick (1) in which the relationship between Schmorl nodes and degenerative spinal changes was analyzed. Herniation of the nucleus pulposus of the intervertebral disk into the adjacent vertebral body leads to the formation of Schmorl nodes. Several mechanisms may underlie the formation of Schmorl nodes (2,3), including degeneration of the cartilage and alterations of the subchondral bone of the vertebral body, which in turn may be due to developmental defects or systemic processes such as osteopenia.

In the Pfirrmann and Resnick study, Schmorl nodes were associated with moderate but not advanced degenerative changes. We have conducted a similar study to test whether Schmorl nodes are related to degenerative changes of the spine or to vertebral osteopenia in vertebrae belonging to pre-Hispanic inhabitants buried in a collective cave on the island of El Hierro, in the Canary Archipelago. The sample was composed of 90 T12, 151 L1, and 91 L2 vertebrae. The number and location of Schmorl nodes were assessed at inspection. The area of these nodules was measured. The severity of degenerative changes was recorded at both the vertebral body and the interapophyseal articular surfaces and was graded as minimal or absent, slight, moderate, or severe on the basis of size and extent of osteophytes.

In 74 cases, osteopenia was assessed with histomorphometry in undecalcified vertebral bone specimens by measuring trabecular bone mass (TBM); and in 115 cases, by measuring bone mineral density (BMD) with dual-energy x-ray absorptiometry

(QDR 2000 software w 5.54; Hologic, Boston, Mass). Conventional radiographs were also obtained in all the vertebrae.

We found Schmorl nodes in 16.67% of T12 vertebrae, in 47.68% of L1, and in 40.66% of L2; the total incidence was 37.35% (124 cases). In 61 cases, the nodes were multiple. In 107 cases, the nodes were in the superior vertebral plate, whereas in 58 cases, they were in the inferior plate. In 41 cases, the nodes appeared in both the superior and inferior plates. There was no association between degenerative changes in the vertebral body and the Schmorl nodes (although there was a trend, $\chi^2 = 2.68$; $P = .105$), between Schmorl nodes and degenerative changes at the interapophyseal articular surfaces, or between the area of the Schmorl nodes and degenerative changes at both the vertebral body and the articular surfaces. Vertebrae with Schmorl nodes showed a significantly higher mean BMD ($0.53 \text{ g/cm}^2 \pm 0.11$) than vertebrae without Schmorl nodes (0.44 ± 0.11 , $t = 4.0$, $P < .001$). There was also a trend for higher TBM in the vertebrae with Schmorl nodes ($18.25\% \pm 5.19$) than in those without Schmorl nodes ($16.11\% \pm 5.01$, $t = 1.72$, $P = .089$). A significant correlation was observed between BMD and TBM ($r = 0.56$, $P < .001$).

Thus, we failed to find any relationship between vertebral degenerative changes and Schmorl nodes or between osteopenia and Schmorl nodes. The higher BMD and the nonsignificantly higher TBM values of the vertebrae with Schmorl nodes are probably explained by the distorting effect of the sclerotic rim that surrounds long-standing chronic Schmorl nodes.

References

1. Pfirrmann CWA, Resnick D. Schmorl nodes of the thoracic and lumbar spine: radiographic-pathologic study of prevalence, characterization, and correlation with degenerative changes of 1650 spinal levels in 100 cadavers. *Radiology* 2001; 219:368–374.
2. Fahey V, Opeskin K, Silberstein M, Anderson R, Briggs C. The pathogenesis of Schmorl's nodes in relation to acute trauma: an autopsy study. *Spine* 1998; 23:2272–2275.
3. Resnick D, Niwayama G. Intravertebral disk herniations: cartilaginous (Schmorl's) nodes. *Radiology* 1978; 126:57–65.

Drs Pfirrmann and Resnick respond:

We appreciate the comments by Dr González-Reimers and colleagues and the interest in our study (1). In their letter, they mention two interesting points that deserve comment.

Dr González-Reimers and colleagues analyzed the presence of Schmorl nodes and the degenerative changes of the spine. Because they used whole paleontologic vertebrae, they were also able to analyze the posterior elements of the spine, which was not done in our work. The presented results are in line with the results of our investigation. In the statistical analysis, Dr González-Reimers and co-workers found a trend for the association of Schmorl nodes with degenerative changes of the spine. In a larger sample and with the analysis of the intervertebral disk height, this trend would probably be significant.

Osteoporosis has been emphasized as a cause of Schmorl nodes, but this correlation is not yet certain (2,3). Analysis of this association is inherently difficult. Most investigations have been performed after the formation of Schmorl nodes, that is, after the healing of the osseous structures. With the healing of bone, sclerosis and callus formation increase the BMD, and preexisting osteoporosis may be masked. Weakness of the end plate and of the underlying trabecular bone that is caused by reduced bone mineral density at the time of the formation of the Schmorl node seems intuitive but remains unproved.

References

1. Pfirrmann CW, Resnick D. Schmorl nodes of the thoracic and lumbar spine: radiographic-pathologic study of prevalence, characterization, and correlation with degenerative changes of 1,650 spinal levels in 100 cadavers. *Radiology* 2001; 219:368–374.
2. Boukhris R, Becker KL. Schmorl's nodes and osteoporosis. *Clin Orthop* 1974; 0:275–280.
3. Hansson T, Roos B. The amount of bone mineral and Schmorl's nodes in lumbar vertebrae. *Spine* 1983; 8:266–271.

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