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Sharp Vascular Calcifications and Acute Balloon Rupture During Embolization

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Is this a dagger which I see before me . . . ?
Macbeth, Act II, Scene 1

Detachable balloon embolization has become a reliable method for treating arteriovenous fistulas [1–4]. We have been frustrated on two separate occasions by balloon failures that we have attributed to calcific vascular spikes that punctured the balloons. In a report by Hieshima and his colleagues (Hieshima et al., unpublished paper), bone fragments from an associated fracture seemed to be the cause. In our cases, there was no antecedent trauma and no fracture; nevertheless, balloon rupture occurred.

Case Reports

Case 1

A 22-year-old woman had swelling in the back of her neck since childhood. CT scans (Figs. 1A and 1B) showed a large, irregularly calcified vascular structure in the left posterior portion of the neck. A loud bruit was heard. MR imaging showed turbulent flow, and a diagnosis of vertebro-vertebral fistula was made. Angiography (Figs. 1C and 1D) confirmed this diagnosis.

We placed a 7.3-F catheter (Cook, Inc., Bloomington, IN) into the left vertebral artery and advanced a series of ITC 1.8-H balloons (Interventional Therapeutics Corp., So. San Francisco, CA) into the varix (Fig. 1E). Of eight balloons placed, five ruptured, and only three remained intact at the end of the procedure (Fig. 1F). Fortunately, those three sufficed to occlude flow through the lesion and eliminate the bruit. The patient recovered without sequela.

Case 2

A 79-year-old woman was previously diagnosed as having bilateral cavernous carotid artery aneurysms. Three years earlier, her right internal carotid artery (ICA) had been ligated in the neck to treat a right abducens nerve palsy. On the current admission, she presented with the acute onset of signs and symptoms of a left carotid artery to cavernous sinus fistula (CCF) that was confirmed by angiography (Fig. 2A).

To assess the therapeutic options (with knowledge of the previous ligation of the contralateral ICA) we performed a balloon test occlusion of the left ICA [5]. The test failed in that the patient developed a right hemiparesis and aphasia. The deficits resolved 2 min after the balloon was deflated. Since it was necessary to preserve the ICA, an ITC 1.5-H balloon was passed through the aneurysm into the fistula (Fig. 2B). The balloon was filled with contrast material (iohexol 200 mg I/ml) (Winthrop Pharmaceuticals, New York, NY).

Over the next several hours, the balloon changed position and began to decrease in size. One day after embolization, the balloon shifted position, and the patient developed an acute hemiparesis and aphasia, which resolved over the next 8 hr. By the fourth day after embolization, however, the bruit had returned, and the balloon was no longer apparent. A repeat angiogram confirmed that the balloon had deflated and the CCF had reopened.

We attempted to place three additional balloons, but each one popped immediately upon reaching the only stable position available within the CCF (Fig. 2C). Two of the balloons ruptured before they could be detached, and they were retrieved (Fig. 2D). Subsequent microscopic examination of the balloons revealed a small focal point of damage, as though they had been abraded and then punctured.

High-resolution CT on bone algorithm showed calcification in the wall of the ICA within the lumen of the aneurysm (Fig. 2E). The calcification extended only about three-fourths of the way around the artery at the exact site of the fistula. We assumed that this represented the spike upon which the balloons were impaled.

The patient returned to the angiography suite and a series of Hilal microcoils (Cook, Inc., Bloomington, IN) were inserted via a Tracker microcatheter (Target Therapeutics, Inc., Los Angeles, CA); these caused almost complete occlusion of the fistula. The bruit decreased at that time and, over the next 12 hr, disappeared completely. A follow-up angiogram 6 days later confirmed complete occlusion of the CCF.

Discussion

In an unpublished communication, Hieshima and his associates described the perforation of balloons by bone frag-

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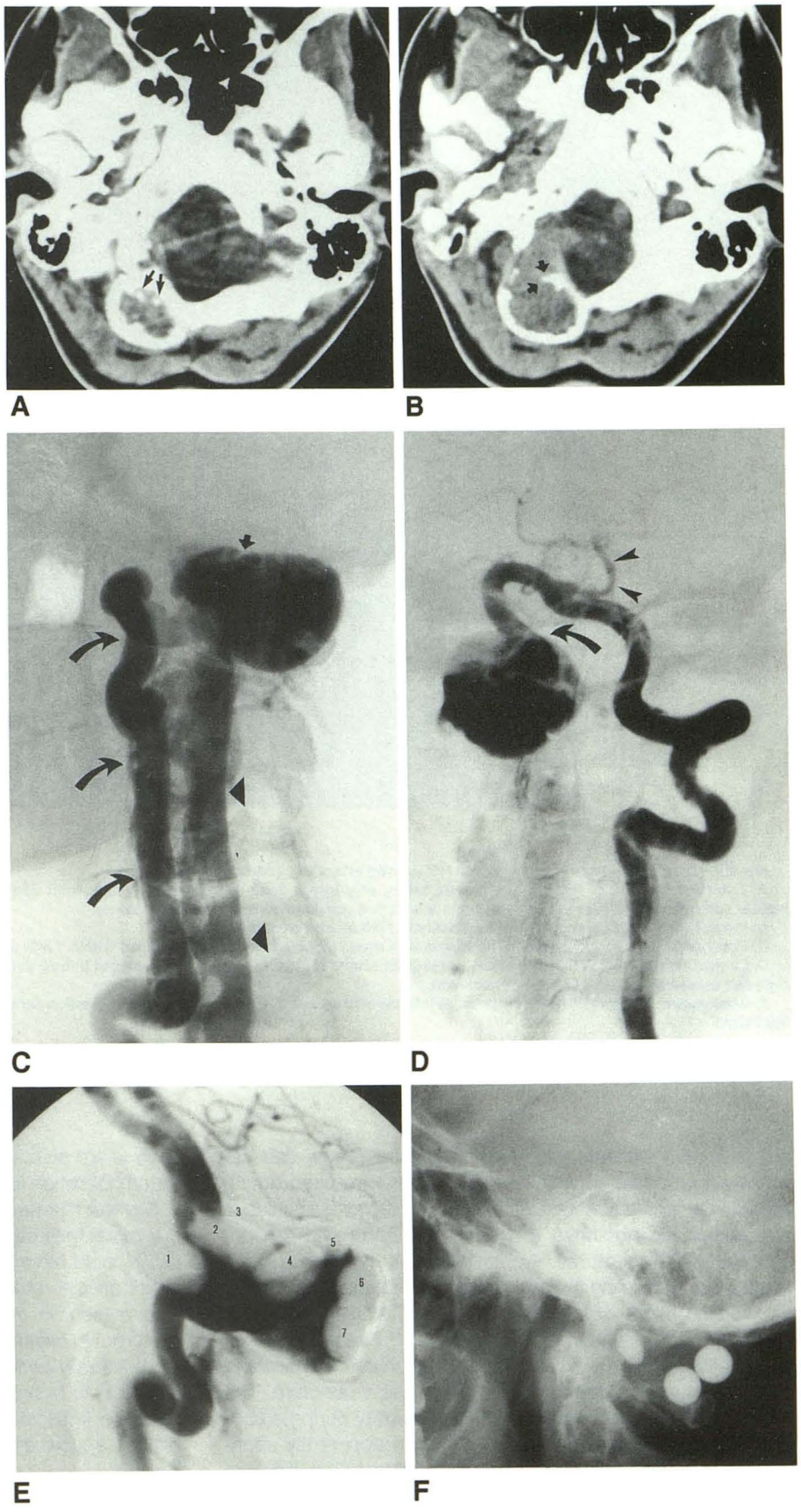

Fig. 1.—Case 1: 22-year-old woman with vertebro-vertebral fistula.

***A*, Unenhanced CT scan at level of skull base shows the varix to have a calcific wall. Stippled calcifications within varix (*arrows*) represent intravascular spikes oriented radially inward. This was not appreciated before embolization.**

***B*, Enhanced CT scan at slightly lower level of skull base shows the point of at least one sharp, calcific, intraluminal spike (*arrows*).**

***C*, Right vertebral artery injection (*curved arrows*) shows immediate dense opacification of varix and ipsilateral vertebral vein (*arrowheads*). Note the sharp filling defect (*straight arrow*) in superior portion of varix.**

***D*, Left vertebral artery injection opacifies the basilar artery (*arrowheads*), but most of the flow refluxes down the right vertebral artery to the vertebro-vertebral fistula (*curved arrow*). A large varix is present at vestibule of venous side of fistula.**

***E*, Seven of the eight balloons introduced remain inflated at this point. Balloons filled with contrast material rest within the varix. There is almost complete cessation of early venous drainage.**

***F*, Of the eight balloons introduced initially, only three remain at the end of the procedure. The bruit was absent at this time.**

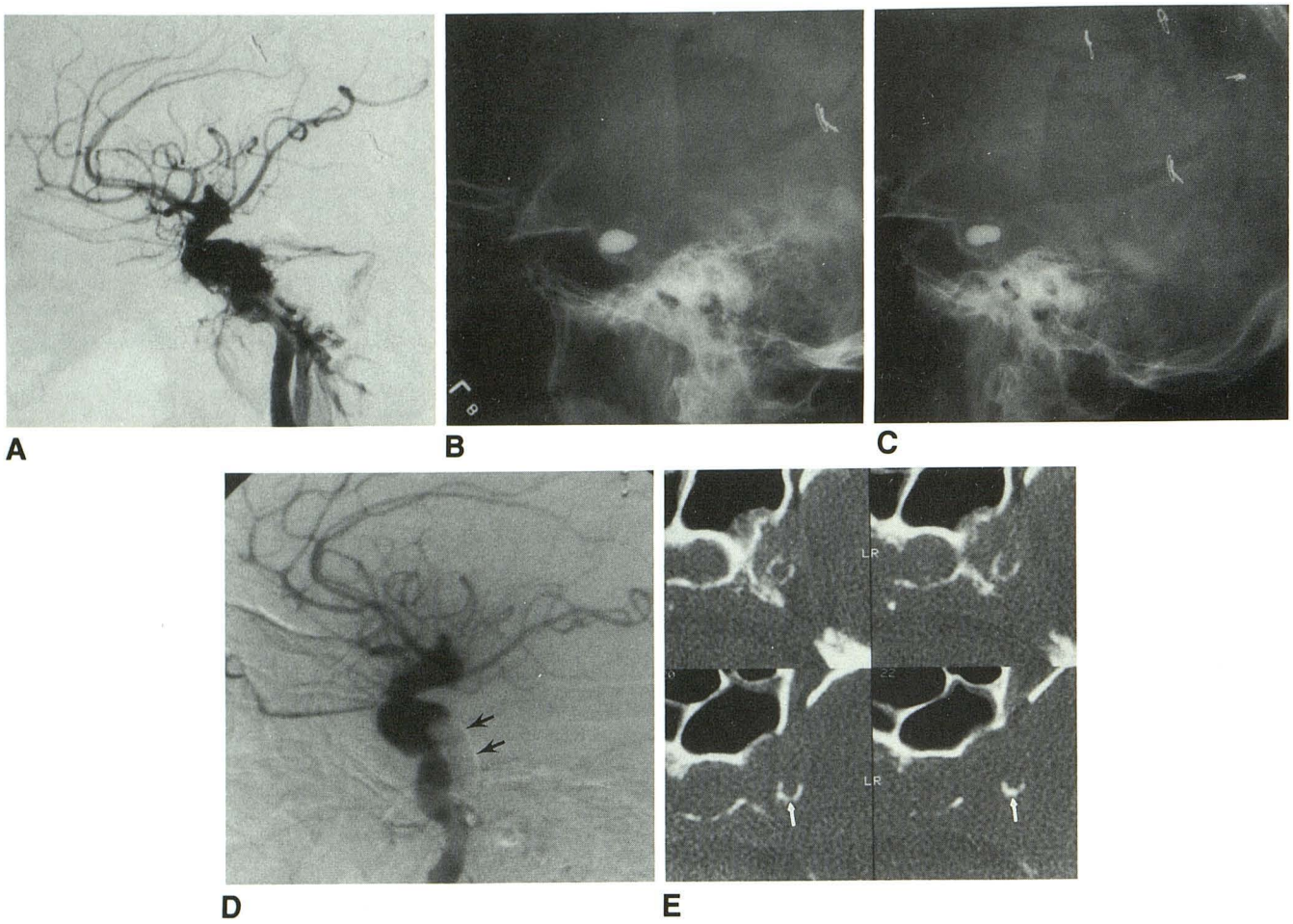

Fig. 2.—Case 2: 79-year-old woman with left carotid artery to cavernous sinus fistula.

***A*, Lateral projection of left internal carotid artery angiogram done on admission. Note rapid filling of cavernous sinus, superior petrosal sinus, and jugular vein. The wires over the parietal bone are from a previous contralateral craniotomy.**

***B*, Lateral view after detachment of first balloon. Fistula was closed at this time.**

***C*, Position of the second balloon. The fistula was again closed, but balloon ruptured within 1 min after detachment.**

***D*, Lateral view of internal carotid artery angiogram after positioning of a third balloon but before detachment. The fistula is closed. This balloon (*arrows*) ruptured before detachment and was retrieved.**

***E*, Contiguous 1.5-mm CT sections through left cavernous sinus. Both the carotid calcification (*arrow*) and the dorsum sella present sharp surfaces in the fistula.**

ments in four patients with acute fractures. They also described a fifth case in which sharp copper wire used during an earlier embolization was responsible for rupture. The cases we present do not have a history of trauma and are not associated with fractures. We usually think of vascular calcifications as paralleling the artery wall. The idea that a vascular calcification might extend into the lumen, more or less *perpendicular* to the wall, is a novel concept. Even if it has an endothelial covering, a vascular calcification can be hard and sharp. As such, it appears that it can cause a puncture. The interventionalist should consider this possibility during embolizations. If multiple balloons rupture in an apparently spontaneous manner, then it seems reasonable to try different placements for the balloon to avoid perforating spicules. If such sharp points cannot be avoided, an alternative method for embolization (coils, for example) must be used.

Several alternative hypotheses for the balloon ruptures in our cases may be considered. For instance, a defective miter valve could have caused deflation. However, the valve was intact on the retrieved balloons in case 2 and a perforation was present in the dome. Overinflation could also cause a rupture. However, in both our cases several of the balloons were actually *under*inflated.

Hyper- or hypoosmolality of the material within the balloon could be implicated in balloon failure. Hypoosmolality might lead to a slow deflation of a balloon; hyperosmolality might lead to a slow overinflation and eventual rupture. We used our standard iohexol mixture (200 mg I/ml, made by diluting

the 300 mg I/ml solution 2:1 with sterile water, not saline). This solution has not resulted in slow balloon deflation in any of our other cases. In case 1, balloons that ruptured did so in less than 10 sec. This does not occur as a result of minimal hypo- or hyperosmolality.

It is interesting to speculate about the cause of the CCF in the second case. The patient had a cavernous aneurysm when the contralateral ICA was ligated, and then presented 3 years later with a CCF. The increased flow in the remaining ICA may have been a causal factor. However, it is conceivable that the pulsating aneurysm was perforated by the same calcification that lanced the balloons. Variously, rupture of the aneurysm may have reoriented a calcific spike to make it perpendicular to a lumenal vascular surface.

We believe that vascular calcifications can cause balloon failure during embolization. While detachable balloons should remain a mainstay of treatment, alternative embolic agents may be required and should be considered when repeated balloon ruptures occur.

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