Bone cement embolism penetrating both the aorta and the right atrium after percutaneous vertebroplasty: Thermal rather than mechanical injury?

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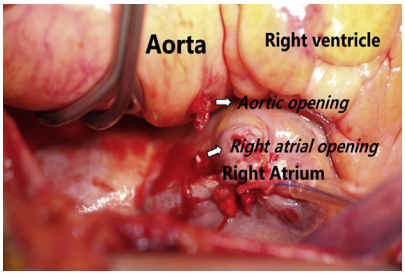

Bone cement embolism penetrated both the right atrial and aortic walls.

CENTRAL MESSAGE

Aortic perforation caused by a bone cement embolism may be thermal rather than mechanical injury.

Percutaneous vertebroplasty (PVP) has been used to treat lumbar compression fractures for more than 30 years. The polymethyl methacrylate bone cement used in this procedure can reach temperatures of 70 °C or greater.[1] Migration of the cement embolisms into the venous system is common but usually asymptomatic.[2,3] These embolisms are rod-shaped because of venous flow dynamics and settle in the right atrium, right ventricle, and pulmonary artery, with a rare risk of thromboembolism.[3] A significant embolic complication, such as cardiac perforation, requires surgical exploration with or without cardiopulmonary bypass. Cardiac perforation can occur through the mechanical force of the rod-like cement embolism during cardiac cycles, but questions arise regarding how a weak bone cement embolus can penetrate a thick aortic wall. We present a case of an 85-year-old woman who underwent PVP and experienced a bone cement embolism penetrating the aorta, which we hypothesize could have resulted from thermal rather than mechanical injury. The institutional review board approved this case report, E2022-053; date of approval: April 15, 2022.

CLINICAL SUMMARY

The patient visited our emergency department and registered a systolic pressure of 70 mm Hg. Eighteen hours earlier in a different facility, she had undergone PVP for repair of the second lumbar vertebra compression fracture. During her time at the previous hospital, she had experienced sudden onset of shock with a systolic pressure of 60 mm Hg for 15 minutes. An enhanced computed tomography scan of the chest showed hemopericardium with a dense strand in the right atrium and several thin strands in the pulmonary artery. In our emergency department, systolic blood pressure increased to 90 mm Hg with percutaneous pericardial catheter drainage and transfusion of two units of fresh-frozen plasma. The drained blood was reddish, suggesting arterial bleeding.

Emergent exploration was performed through a median sternotomy. After removal of the pericardial hematoma, systolic blood pressure increased to 120 mm Hg. The exploration surgery revealed that the tip of a white cement fragment had penetrated the roof of the right atrial appendage (Figure 1, *A*). The fragment was removed (Figure 1, *B*) and the right atrial opening closed with 3 interrupted 4-0 PROLENE pledgeted mattress sutures (Ethicon). The aortic wall facing the atrial penetrating site had a cement tip-sized aortic hole, which was temporarily closed by a loose thrombus. Removal of the thrombus led to a gushing hemorrhage from the aortic hole, which was closed with three interrupted 4-0 PROLENE pledgeted mattress sutures.

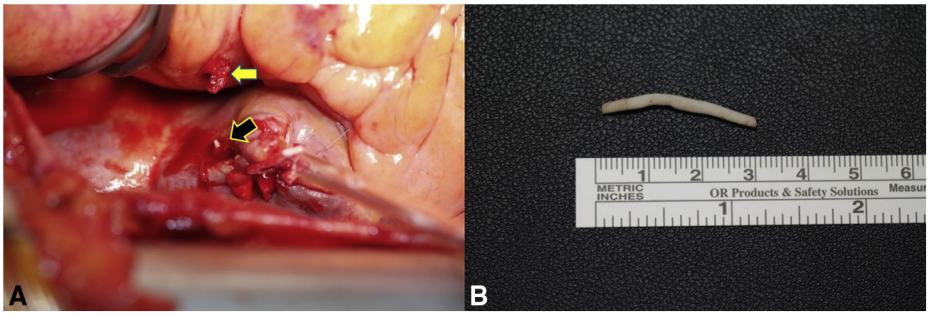

FIGURE 1. A, A cement embolism penetrated the roof of the right atrium (*black arrow*) and then the facing aortic wall (*yellow arrow*). A soft thrombus blocked the aortic hole. B, The bone cement embolism was rod-shaped and friable.

We did not use cardiopulmonary bypass because of her poor respiratory function, so that several thin emboli in the pulmonary artery were not removed, which we considered clinically insignificant. The patient was discharged home 10th day postoperatively with apixaban because of atrial fibrillation.

DISCUSSION

The incidence of cardiac perforation as a complication of PVP is very low, and the cardiac symptoms commonly arise days to months after the procedure rather than periprocedurally. Most cardiac perforations occurred in the right atrium[4] and ventricle.[5] However, in our case, perforations were found in both the right atrial wall and the aorta facing the right atrial wall. The leading cause of cardiac tamponade and hypotension in this case was aortic bleeding. The cement fragment was rod-shaped and easily fractured with a light index-finger force. The cement stick was too fragile to penetrate even the right atrial wall with mechanical force. Thus, it was incomprehensible that the weak cement stick could penetrate the thicker and more rigid aortic wall in the short period after the patient had undergone PVP. We found no literature on aortic injury due to bone cement embolism. When the bone cement is injected into the vertebra, its temperature of more than 70°C can be maintained in the bloodstream, which is 36.5°C, during migration to the right atrium or ventricle. A rod-shaped embolus could puncture the right atrium or ventricle by mechanical force if both ends extend across the limited atrial and ventricular spaces during cardiac cycles. For punctures of the aorta, however, a thermal effect should be considered as a potential mechanism of injury. Given the thickness and rigidity of the aorta, we infer that a thermal effect allowed the cement embolism to puncture the aortic wall and right atrium in this case.

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