Extraction or No Extraction? A Case of Long-Term Follow Up of a Patient with Cement Embolism Induced Right Ventricular Perforation without Pericardial Effusion

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A 78-year-old woman was referred to our hospital for further management of a polymethylmethacrylate (PMMA) embolism to the right ventricle (RV). The patient's medical history included hypertension and liver cirrhosis with hepatitis B. She developed lower back pain 6 months before admission and was diagnosed with multiple compression fractures, which were treated by percutaneous kyphoplasty of the L1, L2 and L3 vertebral bodies. A day before transfer, she was diagnosed with foreign material in her heart during a routine chest X-ray (Fig. 1A). On arrival at our center, an echocardiogram showed a hyperechogenic rod-like structure, 7 cm long, embedded in the RV apical wall without pericardial effusion (Fig. 1B). A computed tomography found the foreign material in the RV with an apical wall perforation (Fig. 1C-F). Endovascular retrieval was considered, but given the expectation that the PMMA cement would not be pliable enough to allow catheter-based retrieval and had been embedded for long time,

Fig. 1. Chest X-ray (A), echocardiography (B), and chest CT (C-F) findings from the first medical examination. A computed tomography demonstrated foreign material in the RV with an apical wall perforation (arrow head).
it was deemed impossible by a consensus decision. Since the patient had been asymptomatic for a long duration, we offered the patient close observation and follow up instead of surgical removal, which she agreed to, after a discussion of the uncertainties and possible complications. Chest CT follow ups after 1 (Fig. 2A, B) and 3 years (Fig. 2C, D) showed no further migration of any cement material or other complications. The patient has not complained of any relevant symptoms so far.

Previous studies have found the incidence of cement embolisms to range from 2.1% to 26%.\(^1,2\) Although the majority of cases were of pulmonary embolisms, some cases of intra-cardiac embolisms were also reported, which included several fatalities. RV perforation occurred when the hardened cement fragment in the ventricle was pushed through the free wall by the tricuspid valve annulus during systole. In this situation, pericardial effusion progresses to cause cardiac tamponade. To the best of our knowledge, this is the first case report of a cardiac perforation caused by migrated cement without cardiac complication, and which did not necessitate surgical or interventional treatment. The mechanism of asymptomatic RV perforation is unclear. However, because the RV is a low-pressure system, perforation may be sealed by a combination of the cement fragment itself, muscle contraction, and fibrosis, resulting in no sequela.

The extraction of a perforated embolic material in the asymptomatic patient is not mandatory when the risks of extraction outweighed the potential benefits in high-risk patients with comorbidities. The authors decided to manage the patient conservatively after discussions with the patient. Such conservative management has been described previously in cases of asymptomatic pacemaker related perforations when the risks of repositioning were expected to be high in selected patients,\(^6\) which is supported by this case report.

To prevent thrombus formation related to cement embolism, anticoagulants have been used to manage patients. However, the role of anticoagulation in patients with asymptomatic embolisms is not clear and long term follow up studies are scarce.\(^7\) Therefore, the treatment selection is often dependent on the severity of each individual case and consideration of bleeding tendency.

**CONFLICT OF INTEREST STATEMENT**

None declared.

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