Intercostal pseudoaneurysm after median sternotomy treated with percutaneous thrombin injection

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ABSTRACT

Intercostal artery pseudoaneurysm is an exceedingly rare complication seen after chest wall insult, either through trauma or operative procedures. We present a case of a 74-year-old man with mitral regurgitation and aortic stenosis who underwent aortic and mitral valve replacement via sternotomy. At the 1-month follow-up, a 1-cm pulsatile mass was noted adjacent to the left of the sternotomy in the sixth intercostal space. Concern for a pseudoaneurysm of the intercostal artery prompted evaluation with ultrasound, which demonstrated a pseudoaneurysm originating from an intercostal artery. This unusually located pseudoaneurysm was treated with ultrasound-guided thrombin injection with complete resolution. (J Vasc Surg Cases Innov Tech 2025;11:101714.)

Keywords: Intercostal pseudoaneurysm; Thrombin injection; Median sternotomy; Cardiac surgery

Intercostal artery pseudoaneurysms (ICPSAs) are exceedingly rare and have been described in the setting of traumatic injuries to the chest wall or as a complication of surgical approaches to structures within the thorax.¹⁻⁴ The prevalence of ICPSAs is unknown because only a handful of cases report this complication of sternotomy.^{2,5-8} Pseudoaneurysms are disruptions of the artery wall that result in collections of the products of the clotting cascade contiguous with the defect of the artery, contained by adjacent tissues.9 They may thrombose spontaneously or persist with to-and-fro flow through the arterial injury into the capsule containing extraarterial blood. In the case of an ICPSA, the capsule is likely contained by the intercostal muscles and ribs surrounding the intercostal artery, as well as the pleura along the interior of the chest wall. ICPSAs are at risk of free rupture, infection, pain, compression of adjacent structures, or embolism. Treatment options include ultrasound-guided compression, ultrasound-guided thrombin injection (UGTI), and open or endovascular surgical repair. The patient has provided informed consent for this case to be published.

CASE REPORT

Our patient is a 74-year-old man with known progressive calcific/rheumatic mitral regurgitation and calcific trileaflet

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aortic stenosis who presented for aortic valve replacement and mitral valve repair due to progression of his valvular disease and associated symptoms of fatigue and exertional dyspnea (New York Heart Association functional class II). He had no prior sternotomy or chest wall trauma or injuries.

His procedure was uneventful and used a typical midline sternotomy with sharp dissection and electrocautery down to the level of the sternum from the jugular notch to the xyphoid process before dividing the sternum with a sternotomy saw and placing a mediastinal retractor. Closure was performed in the standard fashion with the sternum apposed with four sternal wires, skin closed with dissolving suture, and a dry sterile dressing placed. Postoperatively, the patient recovered well apart from common arrhythmias and was discharged home 11 days after his procedure.

At the 1-month follow-up, a nontender pulsating 1-cm mass was found on the left side of his sternum, on the superficial surface between the sixth and seventh ribs. The mass would reduce with manual pressure but quickly refilled when pressure was released.

Vascular surgery was consulted for diagnosing and treating a possible ICPSA. Upon initial ultrasound examination, an active pseudoaneurysm (0.9 cm \times 1.71 cm) left of midline on the chest was identified originating from an intercostal artery. Three days after presenting with ICPSA, UGTI was performed in the outpatient vascular lab setting, with supplies shown in Fig 1. UGTI is a well-described treatment method for pseudoaneurysm in other sites, and we routinely perform them with standard technique as follows. The PSA was marked under ultrasound and draped in the usual sterile fashion. Thrombin (5000 units) was reconstituted with 5 mL of sterile saline diluent and drawn up into a 5-mL syringe. A three-way stopcock was assembled with an 18G access needle attached to the outflow (male) end, and then a 10-mL sterile saline syringe and the syringe of thrombin attached to the inflows, with the stopcock closed to the thrombin. The skin was then anesthetized over the marked pseudoaneurysm with 2 mL of 1% lidocaine. The 18G access needle was inserted at a 45° angle under ultrasound guidance, and was clearly visible within the pseudoaneurysm (see right side of Fig 2). Blood was aspirated into the sterile saline syringe to

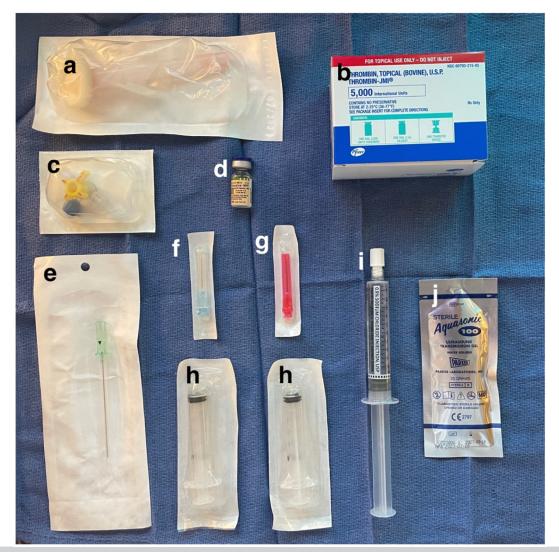


Fig 1. Supplies for thrombin injection. (A) Chlorhexidine skin prep. (B) Thrombin and diluent. (C) A three-way stopcock. (D) Lidocaine (1%). (E) AN 18G access needle. (F) A 25G infiltration needle. (G) Blunt-fill needle. (H) A 5-mL syringe. (I) Sterile saline flush. (J) Sterile ultrasound gel.

confirm placement with the pseudoaneurysm, and the access needle was flushed forward with 1 mL of sterile saline. Thrombin was then slowly injected in approximately <0.5-mL aliquots while visualizing for cessation of flow within the aneurysm. In smaller pseudoaneurysms, <1 mL of thrombin may be required—a larger pseudoaneurysm, such as in this case, requires 2 to 3 mL, but rarely is more used, because large volume thrombin injection may result in nontarget embolization. Although our setup and technique were the same as used with femoral pseudoaneurysms, we advise caution in needle placement to avoid violating the thoracic cavity given the superficial location of the ICPSA.

A completion duplex showed absence of color flow within the ICPSA. A follow-up duplex scan 3 days later confirmed total and

persistent thrombosis of the ICPSA. There was no evidence of embolism or skin necrosis on follow-up.

DISCUSSION

ICPSAs after chest wall trauma often present with hemodynamic instability secondary to hemothorax, and patients are at high risk of morbidity and mortality. ICPSA may be found as an incidental bulging and pulsatile mass adjacent to the sternum found between the ribs or even extending anteriorly to cover the bony structures of the chest wall. These entities have been treated successfully in several different ways, including embolization, stenting, and thrombin injections.^{2,4,10} Treatment of stable ICPSAs should be pursued as quickly as possible

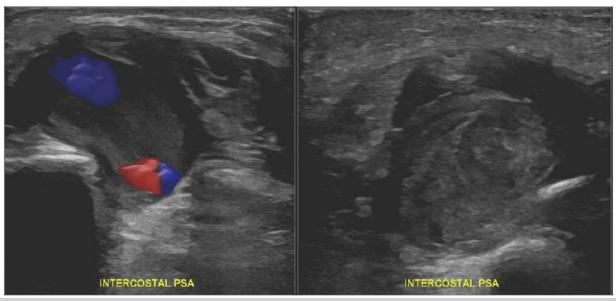


Fig 2. Before (left) and during (right) thrombin injection, with needle visible entering intercostal artery pseudoaneurysm (ICPSA) at the bottom right of image and thrombus within the sac.

owing to the risk of rupture into the thoracic cavity. Case reports of massive hemothorax owing to ICPSAs have suggested that even a stable ICPSA may rupture spontaneously and cause rapid hemodynamic collapse. 1.2 Given the lack of guidelines in treating these cases, we suggest that superficial ICPSAs be treated within the same week on an outpatient basis if possible; any signs of rupture would be obvious and patients could seek treatment. If an ICPSA is on the interior chest wall or in a very posterior position, admitting the patient and treating within 1 to 2 days would be more appropriate given the possibility that a rupture could be hidden until a hemothorax develops and the patient becomes unstable.

Reports of PSAs along the chest wall may also originate from the internal mammary artery with a similar presentation to ICPSAs. These have been reported as complications of sternotomy and closure with sternal wires more frequently than ICPSAs. Similarly, these cases were treated with either embolization, stenting, and thrombin injections to good effect.

Accurate imaging is critical to allow for percutaneous thrombin injection treatments for ICPSA. In our case a combination of ultrasound was the primary imaging methodologies used to define and treat the ICPSA, although computed tomography angiography may identify less superficial cases. If covered stenting or embolization are pursued, then invasive arteriography is a sufficient imaging medium for both identifying and treating the ICPSA.

Owing to the success of our case and review of the small body of literature for this pathology, we advocate for UGTI as the preferred treatment for ICPSAs that are suitable for injection. The procedure is well-described in

literature in other arteries, but the novelty of this case is in the unusual location. UGTI is minimally invasive, well-tolerated, and highly effective for ICPSAs. Thrombin injections are less invasive than open or endovascular surgical repair and are feasible in locations where ultrasound-guided compression is not possible owing to lack of bony structures against which to compress or when compression has already failed.

However, to perform UCTI, the ICPSA must be safely accessible with a needle; in this case, the ICPSA was superior to the ribs, but in cases where the ICPSA is posterior to the ribs, there is a risk of pneumothorax and alternate treatment should be considered. UGTI is also best suited to pseudoaneurysms with a long and narrow neck, because nontarget embolization is more likely in a short or wide neck anatomy. Prior reports demonstrate that diagnosis and successful treatment with thrombin injection may be as prompt as within the first week after surgery, or as late as 3 months.^{1,2} There was substantial variance in follow-up imaging, ranging from serial studies at 2, 7, and 14 days after treatment, to a single follow-up scan at 3 months. Our case demonstrates diagnosis at 1 months, with UCTI 3 days later. There have been at least three reports of ICPSA embolization failure requiring further intervention. 3,13,14

Open and endovascular surgical treatment of ICPSAs has been performed with success in the past; however, in some of these cases the ICPSA was discovered intraoperatively or after less invasive measures had failed.^{15,15} Open treatment of the ICPSA typically involves aneurysmectomy, whereas endovascular treatment may include coil embolization or stenting. In contrast, UGTI is much less time and resource intensive, with the added

advantage of not leaving permanent hardware in the body. Serious complication rates from thrombin injections to treat PSAs are also remarkably low, especially in the case of intercostal arteries that do not have substantial downstream embolization risk. In this case, we safely performed this procedure in an outpatient setting, although in higher risk UGTI areas such as the femoral artery, patients may be observed for several hours or overnight to identify complications such as limb ischemia from nontarget embolization. Conservative treatment has also been reported in literature, but in a report by Bluebond-Langer et al, the authors noted that the diagnosis of ICPSA was initially missed and had they pursued treatment the patient may have avoided multiple subsequent hospitalizations.

CONCLUSIONS

This case reports the successful treatment of a poststernotomy ICPSA with UGTI. This complication is exceedingly rare after sternotomy and care during sternotomy closure to not injure the intercostal arteries may prevent it. However, cardiac surgeons should be aware of this potential complication and the ability to treat it successfully with a minimally invasive approach in the outpatient setting with the help of vascular surgery colleagues. Based on our experience described in this case report, we recommend close interval follow-up for repeat ultrasonography 3- to 5 days after injection to ensure repeat intervention is not required. This intervention is effective, safe, and cost effective and should be considered strongly for the treatment of any chest wall pseudoaneurysms.

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