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Ultrasound-Assisted Compression of the Femoral Artery in a Hypotensive Patient with Expanding Hematoma Post Cardiac Catheterization

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Abstract

Haemorrhagic complications are infrequent, but serious potential events post cardiac catheterization. We present a case where we were able to stabilize a hypotensive patient with a large, expanding femoral hematoma utilizing ultrasound-assisted compression. We demonstrate a unique adjunct to manual compression of arterial bleeding for more rapid stabilization of patients with serious haemorrhagic complications.

Keywords: Ultrasound compression; Post cardiac catheterization; Hypotensive patient

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Introduction

Approximately 1 million cardiac catheterizations are performed annually in the United States [1,2]. Considered the gold standard for detecting coronary artery disease, this invasive procedure has known associated risks. The complication rate is approximately 2% for femoral accessed cardiac catheterization, ranging from minimal adverse reactions, to life threatening sequelae [3]. Haemorrhagic complications, though rare overall, remain one of the most common. Compared to pseudoaneurysm or arteriovenous fistulae formation, hematomas overlying the site of catheter insertions are seen more frequently following femoral accessed cardiac catheterizations. Access site hematomas are usually controlled with manual compression of the site, with only severe haemorrhage requiring reversal of anticoagulation or antiplatelet therapy, or even surgical intervention [4]. Other methods, such as the use of femoral compression devices, with evidence of superiority over manual compression, are not frequently used in catheterization labs due to high cost [5]. Sandbag application and cold-pack mediated vasoconstriction is supported by little clinical evidence and are not always routinely available, making both, far less common in controlling femoral hematomas after coronary angiography [6].

In the emergency department, ultrasound provides a vital tool for visualization of compressible vascular structures. In a prospective study done by Blaivis et al. compression of the abdominal aorta demonstrated decreased flow to the common femoral arteries, monitored using pulse wave Doppler on beside ultrasound. The results of this study demonstrated, with certain pressure, control of life-threatening haemorrhage in critical femoral or inguinal penetrating wounds might be possible [7].

Case Report

A 62-year-old male, with a past medical history significant for hypertension presented to the Emergency Department (ED) complaining of acute onset right groin pain and swelling. He had just been released from a nearby hospital a few hours prior after having a right femoral accessed cardiac catheterization, which did not reveal significant disease. The patient reported the procedure was performed for an evaluation of a three-week history of chest pain. The patient decided to walk down several flights of stairs rather than being taken to his vehicle via wheelchair on hospital discharge. On the drive home, he developed right groin pain and it was during this time that he noticed significant swelling at the puncture site. The patient denied abdominal or back pain. His medications included Aspirin and Plavix.

On examination, the patient was a well-appearing man. His vital signs were initially stable with a blood pressure of 104/68, pulse 74 bpm, oral temperature 97.8 F, respiratory rate of 20% and 98% oxygenation on room air. On examination there was a large, right femoral ecchymotic hematoma that was non-pulsatile and tender to palpation (Figure 1). The patient had full sensation of his lower extremity with normal, palpable peripheral pulses. The patient had 5 out of 5 muscle strength of his lower extremities as well. The remaining portions of the physical exam were unremarkable. Duplex arterial ultrasound of the right lower extremity revealed a large hematoma measuring $5 \times 10 \times 9.1$ cm (Figure 2). There was no sonographic evidence of a pseudoaneurysm. The patient's laboratory data demonstrated a hemoglobin 13.6, hematocrit 41.5 and platelet count of 277. Peripheral venous access was obtained and the patient was given Dilaudid for his pain. Vascular surgery consultation was placed.

Approximately one hour after initial evaluation, the patient became hypotensive with a blood pressure of 89/56. The patient now complained of feeling lightheaded. He became pale and diaphoretic. No active bleeding was seen externally at the right groin. The emergency physician reviewed the ultrasound and noted the hematoma contained mixed echogenicities and large anechoic areas thought to be active bleeding. Intravenous fluids were started immediately, emergent blood for transfusion was ordered, and point-of-care bedside chemistry indicated a two unit drop of hemoglobin from 13.6 to 11.2 and a drop in hematocrit from 41.5 to 33.0. The patients blood pressure dropped further into the 60's. Bedside ultrasound was used to locate the right femoral artery and direct compression of the artery was held for approximately 20 minutes. After this maneuver, vital signs improved with blood pressure rising to 110/70. As per the patient, he felt less pain and thought there was a decrease in the size of hematoma. At this point, the vascular surgeon arrived at the bedside, ultrasound compression of the femoral artery



Figure 1 Large, right femoral hematoma post cardiac catheterization.



Figure 2 Sagittal sonogram of the right groin showing a large hematoma measuring $5 \times 10 \times 9.1$ cm.



Figure 3 A 24-hours follow up of right groin hematoma, with decrease in size.

was replaced with tight compression bandage with ace wrap and Kerlix Gauze. Blood pressure continued to improve with the systolic rising into the 140s. The patient had been stabilized and after discussion with vascular surgery blood products were placed on stand-by. The patient was subsequently admitted to the Surgical Intensive Care Unit for continued observation and monitoring. The patient's Plavix and Aspirin were placed on hold. A computerized tomography scan of the abdomen and pelvis with intravenous contrast revealed an ill–defined right groin hematoma without retroperitoneal haemorrhage. The hematoma gradually decreased in size, the patient's hemoglobin

and hematocrit remained stable, and the patient continued to clinically improve and was safely discharged home after two days (Figure 3).

Discussion

Femoral artery hematoma following cardiac catheterization is a potential life threatening emergency. Typically with appropriate pressure and compression bleeding is controlled. However, more advanced techniques may be necessary in more critical cases. Despite most emergency departments having access to ultrasound machines, use of ultrasound for arterial compression is uncommon. Largely, the treatment for an expanding hematoma has been manual direct pressure. Utilizing the ultrasound transducer for direct localization offers a unique alternative, allowing for precise application of direct pressure.

The use of bedside ultrasound to locate and apply direct

compression of the femoral artery is relatively easy. In our case the Emergency Medicine physician located the vessel and directed a technician to "keep the black circle in the middle of the screen and push". Given the large size of our patient's hematoma manual compression and artery localization was extremely difficult without the use of ultrasound.

In our case ultrasound-assisted compression was able to rapidly stabilize our patient and stop the bleeding. The patient was spared blood product transfusion and ultimately further intervention.

Conclusion

The use of ultrasound-assisted compression of the femoral artery can help control an expanding hematoma in hypotensive patients post cardiac catheterization. We report this novel technique and suggest it be used more frequently to help manage these potential serious procedural complications.

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