



Case report

Deep Vein Thrombosis after Cement Intravasation during Hip Hemiarthroplasty

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ARTICLE INFO

Article history:

Received 17 February 2020

Received in revised form

9 March 2020

Accepted 10 March 2020

Available online 13 April 2020

Keywords:

Cement intravasation

Hemiarthroplasty

Deep vein thrombosis

Embolism

ABSTRACT

Cement intravasation occurs in hemiarthroplasty when pressurization of cement through the medullary canal backflows through the nutrient vessels. This case report describes a 70-year-old woman who underwent hip hemiarthroplasty for a displaced left femoral neck fracture. Postoperative radiographs demonstrated radio-opacity consistent with local cement intravasation. A Doppler ultrasound study subsequently revealed a mobile thrombus at the confluence of the femoral and profunda femoris veins, as well as a nonmobile thrombus within the profunda femoris vein. The more proximal thrombus was determined to be cement that had intravasated during the index operation. The cement likely impeded venous flow, ultimately leading to the development of deep vein thrombosis just distal to the site of cement occlusion.

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Introduction

Surgical interventions for femoral neck fractures are guided by patient age, functional demands, and fracture displacement. Non-displaced femoral neck fractures have a low risk of vascular disruption and can often be treated with closed reduction and percutaneous pinning [1]. Displaced femoral neck fractures violate the vascular supply to the femoral head, leading to an increased risk for avascular necrosis [2]. Therefore, in the elderly patient, surgical replacement of the femoral neck is the preferred treatment choice for displaced fractures, either through total hip arthroplasty or hemiarthroplasty [2].

When performing arthroplasty for femoral neck fractures, attention must be given to the canal diameter. Although there can be significant variability in fixation techniques based on regional preferences, cement fixation is typically used for wider femoral canals with narrower cortices, whereas press-fit fixation is used for narrower canal diameters [2]. In addition, cement fixation may be used in patients with generalized osteopenia/osteoporosis or in cases where there is concern for fracture extension. In cemented hemiarthroplasty, polymethylmethacrylate bone cement is used to

create an interlocking fit between the prosthesis and the surrounding cancellous bone, effectively acting as a “grout” [3]. More recent generations of cement fixation use pressurization to increase the interlocking forces between the implant and trabecular bone [4,5]. However, the use of cement has been linked to rare but morbid complications such as cardiac arrhythmias and cardiorespiratory collapse, primarily from embolization of the marrow during pressurization [3,6].

Cement intravasation is another rare occurrence in cemented hip arthroplasty. This occurs when the pressurization of cement through the medullary canal backflows through the nutrient vessels. The nutrient vessels are the primary vascular supply for the femoral shaft and often originate from the profunda femoris [4,7]. Although it is often asymptomatic and harmless, cement leaving the medullary canal can cause unintended consequences. This is the first reported case in the literature of cement intravasation leading to the development of deep vein thrombosis (DVT) in the immediate postoperative period after hemiarthroplasty.

Case history

Presentation

A 70-year-old woman with a past medical history of osteoporosis and Crohn’s disease was evaluated in the emergency department after a fall while going up an escalator. Of note, she had no

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antecedent hip pain and was a community ambulator for short distances only. On physical examination, her left lower extremity was shortened and externally rotated, and she was neurovascularly intact. Initial radiographs of the pelvis and left hip showed a displaced femoral neck fracture (Fig. 1). Full-length femur radiographs demonstrated thinning femoral cortices with a moderate-sized medullary canal (Fig. 2).

Informed consent

The risks and benefits of both hemiarthroplasty and total hip arthroplasty were explained to the patient, and after extensive discussion, informed consent was obtained to proceed with hemiarthroplasty. This decision was made based on the patient's relatively low functional demands and absence of preinjury symptomatic arthritis in the hip joint.

Operative technique

A posterolateral approach to the hip was used with the patient in the right lateral decubitus position. After dissecting through fascia, short external rotators, and hip capsule, the femoral neck cut was made, the femoral head was removed, and an appropriately sized 45-millimeter (mm) unipolar head was selected. Attention was then turned back to the femur, for which the plan was to use the DePuy Summit (DePuy Synthes, Warsaw, IN) stem with press-fit fixation. The femoral canal was sequentially reamed to a size 5. During subsequent broaching, a size 5 stem was noted to be too proud relative to the calcar. Further broaching risked damage to the calcar, but the size 4 broach did not have sufficient rotational stability for press-fit fixation. The decision was made to use a cemented size 4 implant. A cement restrictor was placed and DePuy CMW 1 Bone Cement (DePuy Synthes, Warsaw, IN), a high-viscosity medium-setting-time polymethylmethacrylate cement, was pressurized into the canal after it had reached medium-to-high viscosity. The anesthesia team was alerted to the fact that cement pressurization was taking place, for which they closely monitored the patient's hemodynamics, avoided intravascular volume depletion, and maintained high inspired concentrations of oxygen – all of which are consistent with the standard of care [8]. Throughout this process, the patient remained hemodynamically stable. A size 4 DePuy Summit (DePuy Synthes, Warsaw, IN) cemented stem was placed along with a 45-mm + 0 unipolar head. On final testing, the hip demonstrated appropriate stability throughout intraoperative



Figure 1. Anteroposterior (AP) pelvis radiograph of injury demonstrating a left-sided, displaced femoral neck fracture.

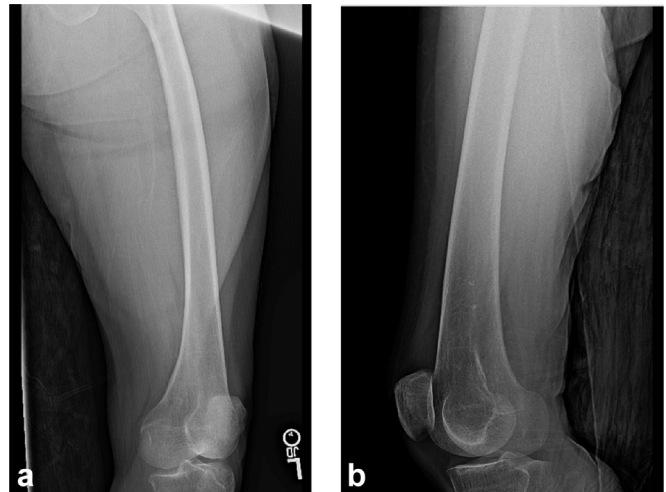


Figure 2. (a) AP femur radiograph from the time of injury demonstrating mild cortical thinning and a moderate-sized femoral canal diameter. (b) Lateral femur radiograph from the time of injury demonstrating mild cortical thinning and a moderate-sized femoral canal diameter.

examination. The soft tissue sleeve and skin were closed, and the patient was awoken uneventfully, with stable vital signs noted in the postanesthesia care unit.

Postoperative course

A postoperative radiograph taken in the operating room showed appropriate positions of the components. However, serpiginous opacity was noted in the soft tissue medial to the shaft of the femur, 10 centimeters (cm) from the lesser trochanter (Fig. 3a). After further investigation with a lateral radiograph (Fig. 3b), it was determined that the opacity was intravasation of cement into a nutrient vessel that most likely occurred during the pressurization of the canal. During this initial postoperative period, the patient remained asymptomatic and recovered in the postanesthesia care unit without complication.

On the first postoperative day, the patient started to mobilize with physical therapy but was noted to be tachycardic to 120 beats-per-minute (bpm) and febrile to 39.1 C. Her postoperative hemoglobin dropped to 7.3 grams/deciliter (g/dL) from the preoperative value of 13.2 g/dL. She received one unit of packed red blood cells, and while her hemoglobin improved to 8.9 g/dL, her tachycardia persisted. A repeat left hip radiograph redemonstrated the opacity along the medial shaft of the femur (Fig. 4).

On the second postoperative day, the patient remained intermittently tachycardic to 110 bpm but was able to ambulate with assistance. She underwent fluid resuscitation but was still intermittently tachycardic overnight. On the third postoperative day, the patient developed shortness of breath and was taken for a computed tomography angiogram that demonstrated no evidence of pulmonary embolism (PE). The patient's fever and tachycardia resolved shortly afterward, and the cause of the patient's symptoms was deemed to be a combination of atelectasis and acute blood loss anemia. On the fifth postoperative day, the patient's vitals remained stable, but she noted new left thigh pain. A lower extremity Doppler study showed evidence of a mobile thrombus at the confluence of the femoral and profunda femoris veins, along with a nonmobile thrombus in the profunda femoris vein (Fig. 5).

After further investigation, the thrombi were noted to be of different density. The proximal mass was more opaque and mobile, and the radiology team reported that the density of the proximal

thrombus was significantly different from that of a typical biological thrombus. Given this great disparity in opacity on ultrasonography, it was ultimately concluded that the proximal thrombus was a foreign body, presumably cement that had intravasated during the operation. Because the location of this cement thrombus was different from the site of cement intravasation visualized on postoperative radiographs, it was thought that a cement piece either broke off from the larger, more distal intravascular cement collection, or it entered the vascular system before this larger cement collection. Regardless, the presence of this cement thrombus was thought to have impeded venous flow and led to the development of a deep vein thrombus distally. The internal medicine team was consulted, and the patient was started on a course of therapeutic apixaban. She continued to progress with physical therapy and was discharged home on postoperative day 7. Owing to the fact that the patient lived across the country, a postoperative clinic visit was arranged with a different surgeon and no further follow-up was obtained locally. At the time of discharge, she was hemodynamically stable with no symptoms suggestive of cardiopulmonary morbidity.

Discussion

This case report describes the intravasation of cement into the vasculature during hemiarthroplasty, a rare event that can occur during pressurization of cement into the medullary canal. To the authors' knowledge, this is the first reported case of cement

intravasation that resulted in DVT after hemiarthroplasty, highlighting the importance of postoperative monitoring when this phenomenon is identified.

While cement intravasation has been reported in arthroplasty, it has been most frequently observed after vertebral kyphoplasty or vertebroplasty [9,10]. In such scenarios, cement leaks into the vertebral venous plexus and often embolizes to the pulmonary circulation [9,10]. Although not as frequent as in these spine procedures, cement intravasation has been found to occur in rare instances after hip arthroplasty [4,5,11–14]. Skyrme et al. [11] described a case of intravenous cement extravasation after hip hemiarthroplasty, where cement was found to penetrate into the accompanying vein of the second perforating branch of the profunda femoris artery. The patient remained completely asymptomatic despite this complication, and subsequent duplex ultrasound showed no evidence of any associated venous thrombus. Similarly, Wang et al. [4] published a case series of retrograde, cement arteriovenograms of femoral nutrient vessels, all of which had no acute or chronic medical consequences. In almost all published case reports, these episodes of cement leakage into the nutrient vessels were asymptomatic and detected only via radiographs, resolving without complication [4,5,11–14]. Unlike kyphoplasty or vertebroplasty, cement intravasation after hip arthroplasty is not typically associated with PE [4,5,11–14].

When cement intravasation does occur, the site of intravasation is similar across most reported cases, owing to the relatively consistent anatomic location of the proximal femoral nutrient vessels [4,5,11–14]. A histologic examination of the femoral shaft found that the most proximal vascular channel was, on average, 10.1 cm distal to the lesser trochanter and had an average diameter of 1 millimeter [15]. Another cadaver study found that the nutrient artery consistently entered the femoral shaft about 16.6 cm distal to the greater trochanter [16]. This location is of importance when assessing postoperative radiographs, as cement noted outside the femoral canal may be misinterpreted as intraoperative fracture. The treating clinician should be aware of this phenomenon and where it

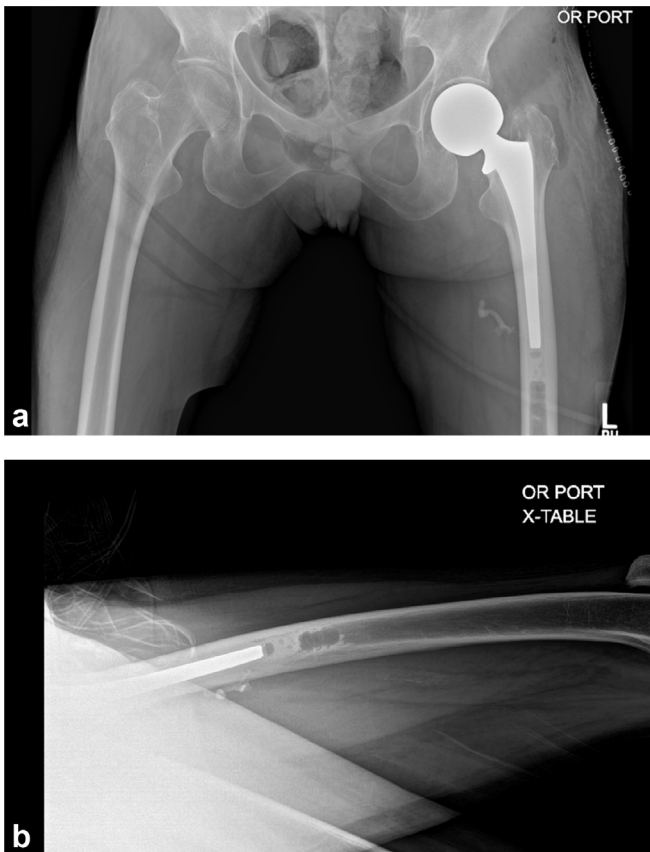


Figure 3. (a) Postoperative AP pelvis radiograph taken immediately in the operating room demonstrating serpiginous opacity in the soft tissue medial to the shaft of the femur. (b) Postoperative lateral femur radiograph taken immediately in the operating room demonstrating serpiginous opacity in the soft tissue posterior to the shaft of the femur.

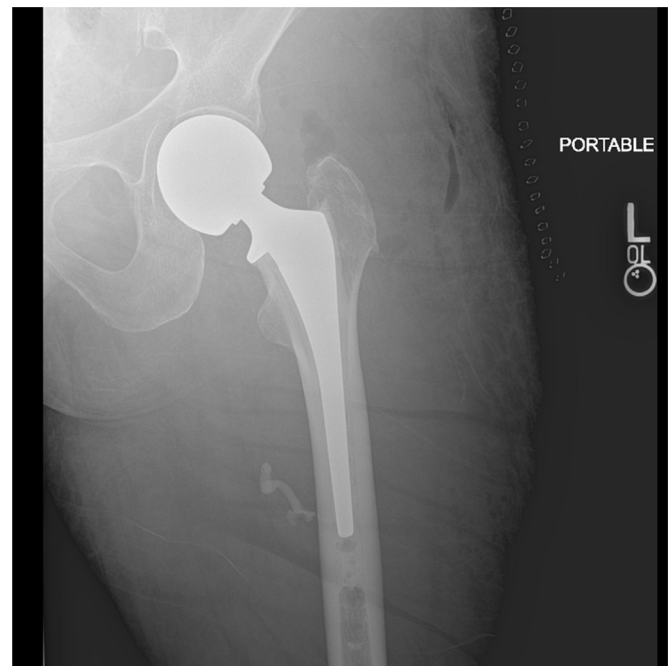


Figure 4. Repeat AP left hip radiograph taken on the first postoperative day demonstrating the continued presence of serpiginous opacity medial to the femoral shaft.



Figure 5. Lower extremity Doppler ultrasound demonstrating a mobile, cement thrombus (A) at the confluence of the femoral and profunda femoris veins, along with a nonmobile thrombus (B) in the profunda femoris vein.

tends to occur, as erroneously returning to the operating room for treatment of suspected periprosthetic fracture can add time, expense, and morbidity to the hospital stay of such patients.

Although most reported cases of cement intravasation during hip arthroplasty have had no associated patient morbidity, there are a few noteworthy exceptions. Deylgat et al. [17] described the case of a 55-year-old man who underwent hip resurfacing and was subsequently found to have intravascular cement that had migrated to the inferior vena cava. Like our case, the patient ultimately

developed DVT owing to alterations in the venous flow distal to the intravascular cement. Another report by Skjølsvik et al. [18] detailed the case of a 77-year-old woman who presented with cardiac tamponade 2 months after undergoing total hip arthroplasty. Further studies found that intravascular cement had perforated her right atrium, an otherwise lethal complication had it not been diagnosed and treated promptly [18]. These cases highlight the importance of serially monitoring patients with known cement intravasation, as late embolism of cement can occur with potentially devastating consequences.

Although further research is needed to determine the risk factors for cement intravasation, it is theorized that some patients may have innate anatomic risk factors that can predispose them to these events. These risk factors may include a less oblique and wide nutrient foramen, as well as small endosteal canal diameters [15,19]. In addition to a high degree of cement pressurization [19], low cement viscosity has also been described as a risk factor for cement intravasation. In one study, it was found that low-viscosity cement favors the path of least resistance, resulting in preferential leakage into nearby vessels rather than deeper bone penetration [20]. This was supported by the case report of Deylgat et al. [17], who noted that in their case of cement intravasation, the cement was pressurized while it was still in a very low viscosity state. The phenomenon of cement intravasation is likely multifactorial and depends on a combination of patient-specific factors, cement properties, and high degrees of cement pressurization.

Although most patients with cement intravasation remain asymptomatic in the immediate postoperative period, the few prior case reports of cardiovascular and pulmonary complications underscore the significance of continued postoperative monitoring of this condition. Routine postoperative radiographs will help determine whether the cement is still lodged in the nutrient vessels. The clinician should have a low threshold to order studies for any signs or symptoms suggestive of cement embolism, such as chest pain, shortness of breath, tachycardia, tachypnea, or lower extremity pain. Because cement has been found to embolize not just to the lungs but also to the great vessels and heart, a diagnostic workup should be conducted based on broader symptomatology, not just on those symptoms specific to PE. If cement embolism is detected, the appropriate services should be promptly consulted, as monitoring

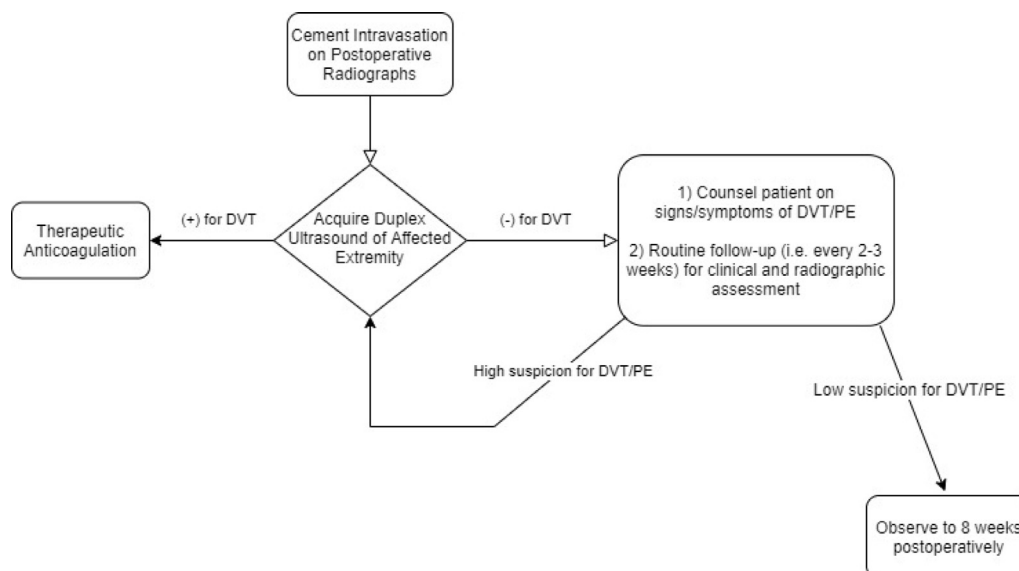


Figure 6. Suggested algorithm for management of a patient with radiographic cement intravasation after hip arthroplasty. Given the rarity of cardiopulmonary complications after cement intravasation, duplex ultrasonography and clinical/radiographic monitoring should suffice in the majority of cases. DVT, deep vein thrombosis; PE, pulmonary embolism.

and treatment for the sequelae of cement embolism will often require a multidisciplinary approach. Our case also demonstrates that even if embolism does not occur, the patient should be monitored for signs and symptoms of local DVT. Because cement can impede venous flow, vessels in the vicinity of the cement intravasation site will be prone to greater degrees of stasis, which is a known risk factor for the development of DVT. For patients with cement intravasation, clinicians should educate them on the symptoms associated with DVT, as this complication may develop after the time of discharge and should be recognized and treated promptly to minimize the risk of cardiopulmonary morbidity.

If cement intravasation is noted on postoperative radiographs after hip arthroplasty, the authors suggest acquiring a lower extremity duplex ultrasound that extends to the more proximal veins of the thigh (Fig. 6). This noninvasive, low-cost study provides a relatively quick means to characterize the extent of intravascular cement and determine if there is an associated thrombus that has developed because of local venous stasis. If a thrombus is visualized, the patient should be placed on an appropriate therapeutic anticoagulation regimen. If there is no associated thrombus, the patient should be counseled on the signs and symptoms of DVT, PE, and cardiopulmonary compromise, with emphasis placed on seeking out care in an urgent fashion if symptoms of these conditions develop. In addition to regularly scheduled postoperative clinic visits, the clinician may consider having the patient follow-up at more frequent intervals (ie, every 2 to 3 weeks) during the initial 8-week postoperative period. At these clinic visits, the clinician can assess for signs and symptoms of DVT/PE and radiographically monitor the intravasated cement. If concern arises at these visits, repeat duplex ultrasonography can be performed. Beyond 8 weeks postoperatively, there are no reports in the literature of complications arising from intravasated cement, and routine monitoring can likely be aborted unless the patient experiences changes in their cardiopulmonary status.

Summary

Cement intravasation after hip arthroplasty is a rare and usually asymptomatic finding. This phenomenon is likely multifactorial and depends on a combination of patient-specific anatomic factors, high degrees of cement pressurization, and cement properties, such as low cement viscosity at the time of pressurization. Clinicians should be aware of the typical anatomy and location of the proximal femoral nutrient artery. This will not only aid in the diagnosis of cement intravasation but will also help prevent the misdiagnosis of suspected periprosthetic fracture from cement seen outside of the femoral canal on postoperative radiographs. In very rare cases, cement intravasation can cause significant cardiopulmonary morbidity or induce the development of DVT owing to local stasis in venous flow. For this reason, patients with cement intravasation should be closely monitored for signs or symptoms of DVT and cement embolism. Given the potential for rare but catastrophic complications, further research is warranted on this topic, as the existing evidence is limited to case reports. This is the first reported

case of DVT after cement intravasation during hip hemiarthroplasty and highlights the importance of ongoing postoperative monitoring for DVT in patients who experience this phenomenon.

Conflict of interest

The authors declare there are no conflicts of interest.

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