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## Case Report

# Intramuscular hemorrhage and fluid extravasation into the anterior compartment secondary to intraosseous resuscitation, the “Nicked-Cortex” sign

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## ABSTRACT

Intraosseous needle access is a reliable method of vascular access used for rapid fluid resuscitation and delivery of medications in certain emergent settings. Fluid extravasation is a possible complication of intraosseous needle access that can lead to compartment syndrome. To our knowledge, imaging findings resulting from this complication have not been described. In this case report, we demonstrate conventional radiograph, computed tomography, and magnetic resonance image findings due to extravasation of resuscitation fluids following the aberrant insertion of an intraosseous needle in an unstable adult trauma patient. We also describe a new radiographic sign associated with this iatrogenic complication, the “Nicked-Cortex” sign.

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## Introduction

Intraosseous (IO) infusion provides quick and reliable temporary vascular access in emergency medical situations where intravenous access is unattainable [1]. In adult patients, this may be the method of choice in instances of cardiopulmonary arrest, shock, extensive burns, sepsis, status epilepticus, and

major traumas [2]. Proper intraosseous catheter placement should result in bone marrow upon aspiration, has easy flow into the catheter, and not result in palpable swelling near insertion site from fluid infusion [3]. Some minor complications include failure to infuse, fractured needle, and placement failure [4]. Doppler ultrasound may be used to identify improper placement with associated extravasation [3]. The complication rate of fluid extravasation from IO access is 3.7% and 0.6%

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**Fig. 1 – 3 mm lucent linear defect noted in the lateral cortex of the proximal tibial diaphysis (red arrow) consistent with the “Nicked-Cortex” sign. (Color version of figure is available online.)**

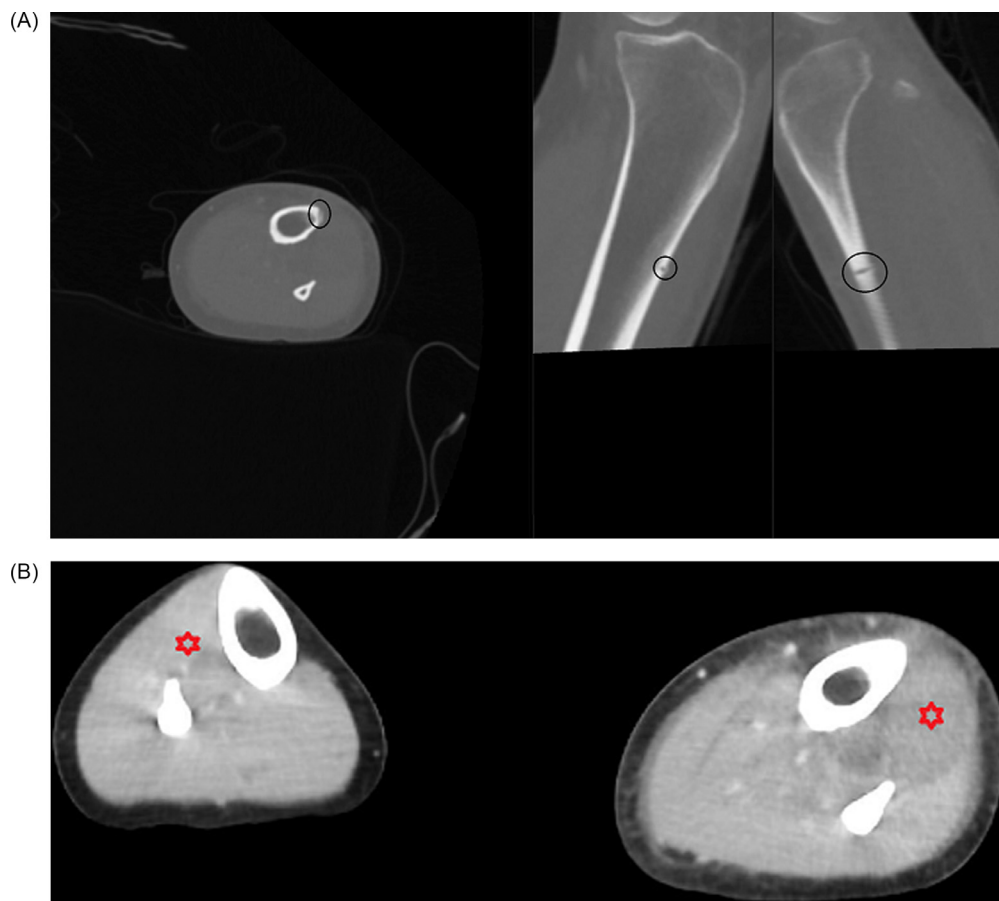
for compartment syndrome [5]. This is an important complication to recognize as there have been several case reports of compartment syndrome and severe limb ischemia due to IO placement [6,7]. Galpin et al. reported a case of bilateral compartment syndrome in a pediatric patient after placement of bilateral IO needles for emergency fluid resuscitation that necessitated 4 compartment fasciotomies [8]. Atanda et al described a case of compartment syndrome in a pediatric patient admitted for hemoperitoneum and pneumoperitoneum as a result of child abuse, requiring tibial IO access [9]. Similarly, Khan et al reported a case of compartment syndrome in an 11-year-old that needed emergency IO access due to spontaneous ventricular fibrillation [10].

### Case report

A 33-year-old female presented for evaluation with a chief complaint of left leg pain. She was admitted to the hospital following a cardiac arrest due to attempted suicide by hanging while incarcerated. At the time of the inciting incident, advanced cardiovascular life support was initiated which included an emergent left leg IO needle placement for vascular access. She was intubated in the trauma bay in the emergency department and subsequently admitted to the neurologic intensive care unit. Postextubation, on hospital day 2, the patient complained of left leg pain. Clin-

ically, she had severe tenderness at the left shin IO site, with decreased strength on ankle dorsal and plantar flexion, and mild dorsal foot swelling. A conventional radiograph (CR) of the left leg was obtained. The radiograph of the left tibia and fibula demonstrated a small, shallow, and linear cortical defect in the proximal diaphysis of the lateral tibial cortex without an associated fracture line (Fig. 1). On physical exam, moderate localized soft tissue swelling was evident of the left knee and lower leg. The patient’s signs and symptoms were monitored until hospital day 4, when increased swelling and erythema of the left leg were noted. Differential considerations at this time included deep venous thrombosis (DVT) infection, or sequela of possible blunt soft tissue trauma during the inciting incident. Further workup was initiated.

Laboratory values demonstrated an elevated D-dimer; however, pulmonary CT angiography (CTA) was negative for pulmonary embolism (PE) and an ultrasound for DVT was also negative. The CTA to exclude PE included a delayed venous phase that extended from the pelvis through the bilateral lower extremities to further rule out DVT. The CT of the lower extremities incidentally revealed a small, shallow, and linear cortical defect in the anterolateral tibia that did not penetrate into the medullary cavity (Fig. 2a), corresponding to the findings noted in the recent left tibia and/or fibula radiographs. In addition, the CT exam revealed asymmetric soft tissue swelling of the left tibialis anterior muscle compared to the contralateral extremity (Fig. 2b). A left lower extremity



**Fig. 2 – (A) Axial, coronal, and sagittal CT planes demonstrates the lucent linear cortical defect (black circles) in 3 dimensions “CT Nicked-Cortex Sign.” No intramedullary extension of the defect noted as would be expected with successful intraosseous access. (B) Axial CT comparison of the right and left lower extremities demonstrate an asymmetrically enlarged, low density left tibialis anterior muscle compared to the contralateral lower extremity, (red asterisks) suggesting increased compartmental pressure due to excess intramuscular fluid on the left. (Color version of figure is available online.)**

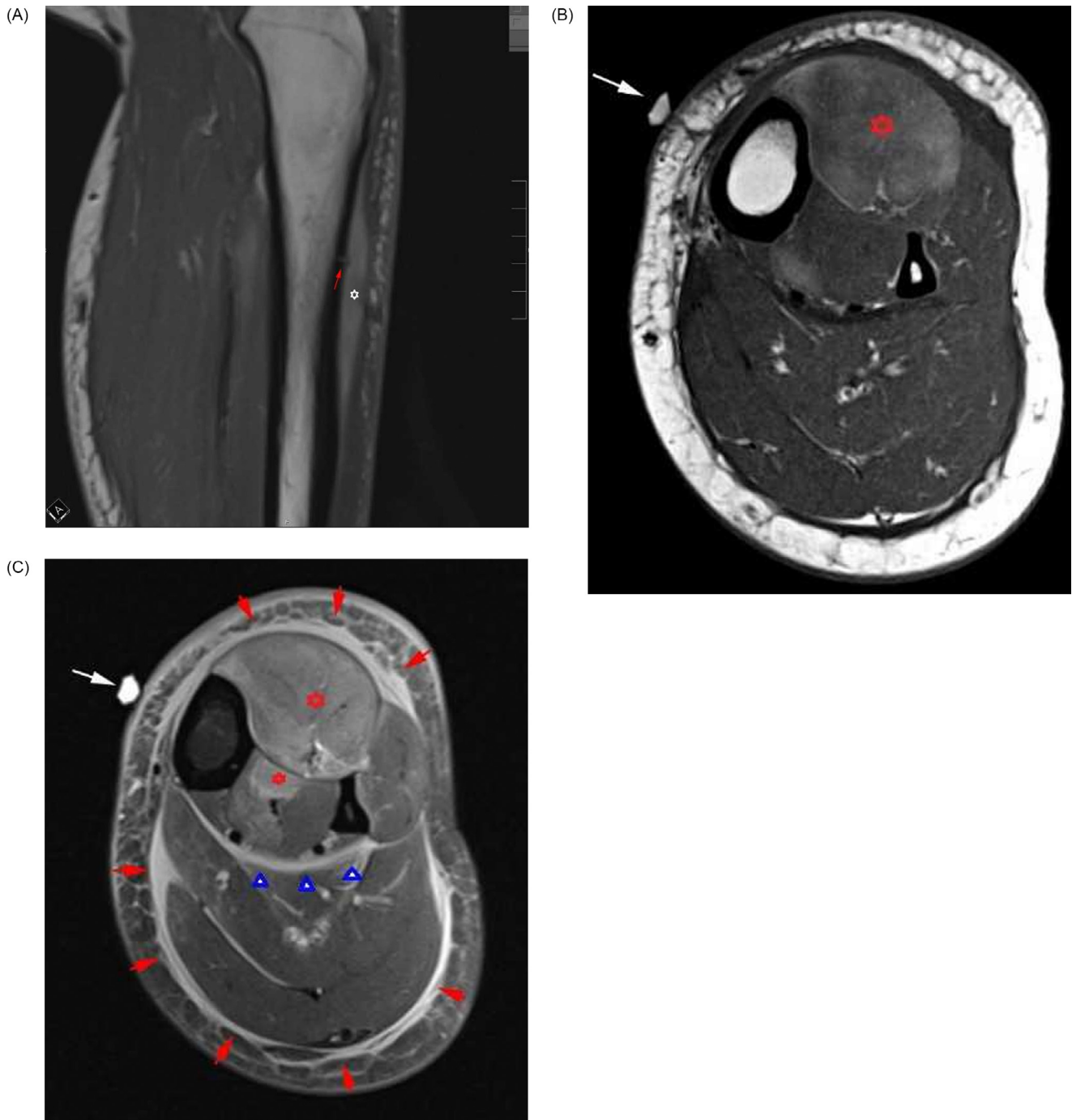
magnetic resonance imaging (MR) study was ordered to evaluate for soft tissue swelling and compartment syndrome. MR images revealed a small, partial-thickness cortical defect along the anterior lateral aspect of the proximal tibia consistent with the attempted IO placement (Fig. 3A-C). There was marked distention of the anterior muscle compartment with less conspicuous distention of the deep posterior and proximal aspect of the lateral compartments. Within the anterior compartment, there was abnormally increased T1 and T2 signal consistent with early subacute intramuscular hemorrhage [11]. The marrow signal of the tibia and/or fibula was normal. Of note, the patient elected to prematurely terminate the MR exam due to discomfort and the contrast portion of the study was not performed. Surgical consultation for a potential fasciotomy was obtained. Despite the aforementioned imaging findings, the surgical evaluation deemed that the clinical signs and symptoms of compartment syndrome were equivocal and watchful waiting approach was adopted. Intracompartmental pressure measurements were not obtained. Over the next few days, the patient’s pain and swelling gradually decreased and she was subsequently discharged from the hospital.

## Discussion

IO vascular access was first introduced by Drinker in 1922 as a method for accessing the systemic circulation via the intramedullary venous plexus of the bone marrow. With the advent of more effective intravenous equipment and techniques, IO access lost popularity until the 1980s when IO access was reintroduced, particularly for rapid fluid infusion during resuscitation in pediatric patients [12].

A study performed in Heidelberg Germany in 2007 compared the results of inserting IO needles using the manual approach vs the semiautomatic system into adult human cadavers [13]. The semiautomatic system was proven to be more effective. The following 3 criteria were accepted as signs of a successful IO puncture into cadaver bone:

1. Sudden loss of resistance;
2. Stable, “springy” hold after releasing the stylet from the needle;
3. Free flow of saline without surrounding soft tissue swelling. This finding was documented as the safest, most reliable sign of proper insertion.



**Fig. 3 – (A)** Sagittal T1 weighted MRI of the left lower extremity shows a small partial thickness cortical defect “MR Nicked-Cortex Sign” in the anterior tibia (red arrow). Note the adjacent high T1 signal fluid within the anterior soft tissues (white Asterisk). **(B)** Axial T1 weighted MRI of the left lower extremity demonstrates an enlarged tibialis anterior muscle belly (red asterisk) with high intrinsic T1 weighted signal. There are anterior and posterior bowing of the deep fascial planes of the anterior compartment concerning for acute anterior compartment syndrome. The MRI marker demonstrates the patient’s reported area of concern (white arrow). **(C)** Axial PD fat saturated MRI of the left lower extremity demonstrates high intrinsic signal predominately within the tibialis anterior, and to a lesser degree within the tibialis posterior muscles (red asterisks). There are thickening and edema associated with the intermuscular fascia (blue arrow heads) and circumferential edema in the deep fascial planes (red arrows). (Color version of figure is available online.)

The semiautomated system proved to have slightly better results and was associated with fewer technical complications. Dasgupta et al. found that manual insertion of IO needles resulted in an irregular entry site into the bone resulting in a higher chance of extravasation. In contrast, they found that powered or assisted IO access systems provided more rapid access and a very predictable entry site, minimizing the risks of extravasation [6].

IO associated extravasation of fluid into the soft tissues may result from “incomplete penetration of the needle through the cortical bone, extension of the cannula through the proximal tibia into the posterior compartment of the leg, extravasation through previous intraosseous puncture sites, and extravasation through the nutrient vessel foraminae [8].” IO access should be avoided in fractured extremities because of the risk of fluid extravasation [9]. In addition, secondary extravasation of fluid into the surrounding soft tissues can occur as a result of increased intraosseous pressure from a high rate of infusion or due to a large total volume infused [10].

Although the risk of iatrogenic compartment syndrome is not well documented, radiographic documentation of the delayed complications of IO placement is even more rare. In this case, the patient presented with specific signs and symptoms approximately 4 days after admission, 2 days after extubation, which necessitated the need for advanced imaging for further evaluation. The MR images corroborated the CT findings, revealing that the IO needle did not enter the medullary cavity of the tibia. In addition, the anterior compartment was distended, and it exhibited abnormally bright T1 and T2 signal intensity. The T1 shortening is a finding most consistent with an early subacute intramuscular hemorrhage.

Anterior compartment syndrome is a serious complication that can be associated with acute intracompartmental hemorrhage. Compartment syndromes are a clinical diagnosis that can be quantified by measurement of intracompartmental pressures; however, imaging findings on MR are suggestive of compartment syndrome when there is a diffuse intramuscular edema pattern combined with increased muscle girth and bowing of deep fascial membranes [14]. In addition, the usage of intravenous contrast can be useful to assess for perfused vs devitalized muscle and fascia.

In this case, the attending radiologist immediately conveyed the concern for compartment syndrome to the medical team and as a result, general surgery was consulted for a potential fasciotomy. Despite the aforementioned imaging findings, the patient’s clinical condition was deemed to be improving and therefore a fasciotomy was not performed. After discharge, the patient became lost to follow up and it is still unknown whether or not this patient remains at risk for long-term complications.

While the reported frequency of extravasation from IO placement is relatively low at 3.7%, and the frequency of compartment syndrome is only 0.6%, recognizing this complication is vital to avoid limb loss. Other serious complications include contractions (Volkmann ischemic contractions), long standing neuropathies, myonecrosis, and systemic complications such as renal failure from rhabdomyolysis [15]. In addition to compartment syndrome, IO access has been associated with iatrogenic fractures, injury to growth plates, cellulitis, and one rare case of a popliteal artery thrombosis [9].

Fat embolism has been raised as a theoretical risk although not reported clinically [9]. Chalopin et al reported osteomyelitis associated with IO placement in 6 patients, suggesting direct inoculation as the route of infection [16]. Interestingly, previous researchers found that direct inoculation of bacteria into the medullary cavity of animal models did not reliably produce osteomyelitis. It was only when Scheman et al pretreated the intraosseous injection site with the sclerosing agent sodium morrhuate, that osteomyelitis could be ensured after the bacterial inoculation [17]. In effect, Scheman found it exceedingly difficult to induce osteomyelitis without first creating aseptic necrosis. Two case reports of IO induced osteomyelitis specifically describe the emergent use of adrenaline through the IO route, with one case using high doses (1:1000; 0.1 mg/kg) after the standard concentration of (1:10,000; 0.01 mg/kg) had little effect. Both of these cases reported a localized inflammatory response around the IO site that resulted in cutaneous necrosis as well as subsequent osteomyelitis [10,18]. It could be theorized that multiple or high doses of IO adrenaline could elicit a local marrow infarct, thus predisposing one to osteomyelitis similar to the model described by Scheman. Presumably, the mechanism of an adrenaline induced marrow infarct would be due to its known vasoconstrictive properties.

Given that most patients that require IO access are in some stage of extremis, often requiring prolonged intubation, it is vital for clinicians and radiologists to recognize IO complications in the obtunded patient to avoid long term complications. In patients with limb swelling and pain status post IO placement, it is important to confirm the history of IO placement in the clinical record or on admitting radiographs. If an IO is not apparent from the initial imaging, searching for subtle radiographic signs of a small osseous cortical defect on CR should raise the suspicion for errant IO needle placement. The “Nicked-Cortex” sign is postulated to occur as the result of an IO glancing off of the cortex at entry. Ideally, the IO entry site should be as perpendicular to the cortical bone surface as possible to limit the possibility of slipping off of the cortex and into the adjacent muscle compartment. Admittedly, given the inherent urgency necessitating the placement of IO access, often in the field, conditions can be less than ideal. Further studies are necessary to determine the incidence of the “Nicked-Cortex sign”; however, given its small size and innocuous appearance it would be easy for the uninitiated to overlook this finding. In subacute to chronic cases one could surmise the formation of a small amount of periosteal new bone formation at the site of the insult. Ostensibly, the CR version of this sign would have a low sensitivity but high specificity for errant IO placement. The CT correlate to this sign is of similar appearance to CR but the ready access to multiplanar reconstruction tools, available on most late-modeled picture archive and communicating systems, may help to perceive this sign and thus increase the sensitivity. On MR, the small cortical defect was challenging to identify but not imperceptible. The true value of MR in cases of IO extravasation are findings of increased compartment tension and/or pressure as depicted by bowing of the deep compartment fascia, fascial thickening, and intramuscular infiltration of fluid. Increased intercompartmental fluid, whether attributed to edema, pus, or hemorrhage can ultimately lead to an acute compartment

syndrome. In this case, on MR, the signal intensity exhibited characteristics most indicative of hemorrhage, presumably from damage to a vascular vessel within the anterior compartment. Additionally, infiltration of fluid through the misplaced IO, especially if aided by increased bag pressure, could conceivably have a similar effect on the anterior compartment. While the MR findings of errant IO placement and extravasation are nonspecific and could be similar in appearance to necrotizing fasciitis, careful assessment of the patient's event timeline, history of recent IO placement in the area of concern, and the presence of the "Nicked-Cortex Sign" should lead one to the correct diagnosis.

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