

# Serious Infectious Complications Related to Extremity Cast/Splint Placement in Children

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Posted: 08/15/2011; J Emerg Med. 2011;41(1):47-50. © 2011 Elsevier Science, Inc.

## Abstract and Introduction

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### Abstract

**Background:** Extremity injuries necessitating splinting or casting are commonly seen in the emergency department (ED) setting. Subsequently, it is not uncommon for patients to present to the ED with complaints related to an extremity cast or splint.

**Objective:** To present a literature-based approach to the identification and initial management of patients with possible infectious cast/splint complications in the ED setting.

**Case Reports:** We present two cases of serious infectious complications arising from extremity cast/splint placement seen in a single pediatric ED: a case of toxic shock syndrome in an 8-year-old child, and a case of necrotizing fasciitis resulting in upper extremity amputation in a 3-year-old child.

**Conclusions/Summary:** A wide spectrum of potential extremity cast/splint infectious complications may be seen, which include limb- or life-threatening infections such as toxic shock syndrome and necrotizing fasciitis. Simply considering these diagnoses, and removing the cast or splint to carefully inspect the affected extremity, are potential keys to early identification and optimal outcome of cast/splint complications. It is also prudent to maintain particular vigilance when treating a patient with a water-exposed cast, which may lead to moist padding, skin breakdown, and potential infection. In patients with suspected serious infections, aggressive fluid management and antibiotic therapy should be initiated and appropriate surgical consultation obtained without delay.

### Introduction

Significant casting and splinting complications, although uncommon, can be more severe than many clinicians might anticipate. Even the most astute clinician may easily miss these complications. We present two cases of infectious life- or limb-threatening cast/splint complications presenting to a single pediatric emergency department (ED). We also review the pediatric and adult medical literature regarding such complications, and use these cases to highlight the red flags to look for when a patient presents to the ED with a cast- or splint-related complaint. The approach to these complaints can perhaps be best summarized by a phrase taken from the orthopedic literature: "there are no hypochondriacs in casts".<sup>[1]</sup>

### Case Reports

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#### Case 1: Toxic Shock Syndrome

An 8-year-old girl presented to the ED 2 weeks after sustaining fractures to the distal radius and ulna. A fiberglass splint was placed at an outside ED at the time of initial injury for treatment of distal shaft fractures. She was evaluated by an orthopedic surgeon for follow-up earlier in the day preceding the second ED visit, who noted friction-induced blisters and skin maceration on her right middle finger upon removal of the forearm/hand splint in his office. A radiograph confirmed healing fractures and a plaster cast was placed. Later that night, she developed a temperature of 39.4 °C (103 °F), myalgias, progressively worsening hand swelling and pain, and a diffuse, erythematous rash, all of which prompted the second ED visit.

Initial ED vital signs were as follows: temperature 38.5 °C (101.3°F), heart rate 162 beats/min, blood pressure 101/49 mm Hg, respiratory rate 24 breaths/min, and room air oxygen saturation of 100%. Removal of the cast revealed purulent blisters on two of her fingers with redness, warmth, and swelling. The extremity had good pulses, capillary refill, and sensation. She was well-appearing, and the remainder of the examination was unremarkable. Significant laboratory findings included mildly elevated liver transaminase levels (less than twice the upper limits of normal), pyuria without bacteruria (> 10 white blood cells [WBC] per high power field), and a systemic leukocytosis: WBC count 21.8 K (normal 4.8–13.0 K/mL<sup>3</sup>) with 92% granulocytes. Blood and wound cultures were obtained, and intravenous clindamycin was initiated.

During the first day of hospitalization, she was persistently tachycardic (heart rate around 150 beats/min) with borderline hypotension, which was initially unresponsive to intravenous fluid administration. Ceftriaxone and vancomycin were added empirically, and she was transferred to the pediatric intensive care unit (PICU) for closer monitoring. Her liver function tests worsened (transaminases several-fold greater than the upper limits of normal), and her plasma coagulation parameters were mildly elevated. She steadily improved with more aggressive crystalloid infusion and antibiotic therapy, and was ultimately discharged on oral clindamycin 3 days later. Wound cultures were positive for growth of methicillin-sensitive *Staphylococcus aureus*, whereas blood cultures did not demonstrate bacterial growth.

A presumptive diagnosis of toxic shock syndrome (TSS) was made. This was based on the presence of high fever, relative hypotension, diffuse macular erythroderma, with eventual desquamation. In addition, she exhibited myalgias, pyuria without bacteruria, and hepatic dysfunction. Furthermore, blood, cerebrospinal, and throat cultures were all negative.

## **Case 2: Necrotizing Fasciitis**

A 3-year-old otherwise healthy boy was brought to the ED after falling from playground equipment, resulting in a Gartland type I (non-displaced) supracondylar fracture. He was neurovascularly intact. There were no skin or soft tissue wounds, lesions, or abrasions identified. He was splinted without manipulation in a posterior long arm fiberglass splint, and discharged after arranging orthopedic follow up within the week.

The child returned to the ED 3 days after the initial evaluation, complaining of increased redness and swelling above the splint, particularly over the right shoulder. He appeared comfortable, in no significant distress, with normal vital signs. An area of ecchymosis and swelling was noted over his right shoulder, without any crepitus or bony tenderness to palpation. In addition, a 1-cm superficial abrasion was noted in his right axillary region. Normal capillary refill was noted distally.

The discomfort was presumed to be secondary to a tight splint, causing constriction, edema, and discomfort. The splint was therefore replaced. Due to the new shoulder pain, a radiograph of the right shoulder was obtained to exclude additional injuries that might have been missed on the first evaluation. The radiograph was negative for fracture or visible soft tissue abnormalities. He was discharged home with the diagnosis of shoulder contusion.

The following day the child arrived in the office for orthopedic follow-up, where he was noted to be toxic-appearing. He was taken by ambulance to the nearest ED, where his vital signs were: temperature of 40.8°C (105.5°F) per rectum, heart rate 220 beats/min, respiratory rate 60 breaths/min, an undocumented blood pressure, and a capillary refill time of 10 s. The extremity was dusky, without a radial pulse. He was intubated for respiratory distress, and intraosseous access was obtained. He was aggressively fluid resuscitated. Intravenous ceftriaxone, vancomycin, and metronidazole were administered. He was transferred back to the initial institution for pediatric orthopedic evaluation and PICU admission.

On arrival to the PICU, central venous access was established, gentamicin and ceftazidime were administered, and a norepinephrine infusion was begun. The WBC count was 5800/mL<sup>3</sup> with 52% band forms. A presumptive diagnosis of necrotizing fasciitis was made, and the child was immediately taken to the operating room. A gangrenous right arm was amputated and debridement of the anterior chest wall was necessary due to extensive soft tissue necrosis. After surgery, his vital signs improved and signs of sepsis rapidly resolved. Cultures obtained in the operating room demonstrated heavy growth of *Streptococcus pyogenes*. He required several additional operations for debridement, but was ultimately discharged home without further complications.

## Discussion

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To place these cases into context and provide an up-to-date discussion, a literature search was performed for English language articles, case reports, reviews, and textbook chapters published in the last three decades. The orthopedic and emergency medicine literature was the focus of the search. Although there were relatively few publications specifically related to cast and splint complications, those that were found often cited each other and demonstrated several consistent recommendations.

Clinicians should be mindful of important historical features placing patients at increased risk for cast- or splint-related infectious complications. Based on a review published in the orthopedic literature, those at highest risk for cast/splint-related complications include: patients that cannot effectively communicate (extremes of age, altered mental status, multi-trauma, or developmental delay); patients with decreased sensation (nerve blocks, neuropathies) or immune compromise (diabetes, other), and patients with spasticity or cerebral palsy (Table 1).<sup>[1]</sup> Stasikelis reported that as many as 5 out of 79 patients with cerebral palsy had infected decubitus ulcers beneath their cast when removed.<sup>[2]</sup> In addition to friction or pressure from direct cast/splint contact, "scratching" under the cast with a foreign object, such as a coat hanger, can breach skin integrity and increase the risk of subsequent infection.<sup>[3]</sup> Friction-induced skin breakdown from the fiberglass splint was postulated as the initial portal for infection in the case of necrotizing fasciitis presented.

**Table 1. Risk Factors for Casting/Splinting Complications**

Extremes of age
Altered mental status
Multi-trauma and distracting injuries
Developmental delay
Altered sensation (nerve blocks, neuropathies)
Diabetes
Immune compromise
Cerebral palsy/spasticity

Historically, plaster casts were routinely applied to patients with fractures. However, over the past 20 years, fiberglass has taken the place of plaster as it dries faster, creates a lighter, stronger cast, and is more resilient to water immersion. Whereas wet plaster casts often fall apart and require recasting, a wet *fiberglass* cast will not decompose and can lead to moist padding, skin breakdown, and potential infection.<sup>[1]</sup> The primary disadvantage of fiberglass is that it may not allow as much room for swelling when compared to plaster.<sup>[4,5]</sup> However, regardless of the casting material utilized, a foul-smelling cast can be a clue to damp padding, with associated skin breakdown or underlying infection.

Infectious complications of casting and splinting range from mild, local skin infections, such as cellulitis or abscess, to life-threatening conditions such as necrotizing fasciitis, gangrene, or TSS (Table 2).<sup>[1-3,6-8]</sup> Almost all of these diagnoses may be suggested by simple inspection of the skin under the cast or splint, as well as a thorough neurovascular examination of the affected extremity. It may seem self-evident, but it bears mentioning that complete removal of the cast or splint is necessary to fully expose the affected extremity for a complete examination. In the potentially rushed, high acuity environment of a busy ED, it may be tempting to shortcut this critical step.

**Table 2. Infectious Complications of Casting/Splinting**

Potential Infectious Complications
Cellulitis
Abscess
Necrotizing fasciitis
Clostridium gangrene
Toxic shock syndrome

For patients in whom a necrotizing soft tissue infection is suspected, further laboratory testing or diagnostic imaging should be considered. Laboratory studies should include a complete blood count with differential, C-reactive protein, and a basic metabolic panel, specifically for creatinine, sodium, and glucose.<sup>[9]</sup> Abnormalities in these laboratory parameters have been shown to have a high predictive value in discriminating necrotizing fasciitis from severe cellulitis. Additional tests of value include wound and blood cultures, bicarbonate, lactate, and creatinine phosphokinase.<sup>[10]</sup>

Imaging is also an important modality in diagnosing necrotizing infections. Although subcutaneous air on plain radiograph is specific for necrotizing skin infection, the sensitivity may be as low as 17%.<sup>[11]</sup> Computed tomography (CT) and magnetic resonance imaging (MRI) are more effective in the evaluation of abnormal soft tissue gas dissecting along fascial planes, as well as fascial thickening and stranding.<sup>[12]</sup> MRI is very sensitive for the identification of fascia necrosis, but is less specific and may overestimate the extent and severity of infection. Specific data regarding the sensitivity and specificity of CT compared to MRI are unavailable, but relying on either of these studies may delay definitive diagnosis and treatment in a critically ill patient. In practice, clinical judgment is the most important element in the diagnosis of necrotizing soft tissue infections, and radiographic studies are more useful for equivocal presentations.<sup>[9,13]</sup>

The diagnosis of TSS is based on several clinical and laboratory criteria, which include the presence of fever, hypotension, diffuse rash, and desquamation.<sup>[14]</sup> In addition, three or more of the following systemic symptoms must be present: gastrointestinal symptoms, muscular symptoms, mucous membrane involvement, renal findings, hepatic abnormalities, thrombocytopenia, or signs of central nervous system involvement. Furthermore, cultures of urine, cerebrospinal fluid, and blood (excluding staphylococcal bacteremia) must be negative. Finally, if obtained, diagnostic studies for alternative diagnoses (such as Rocky Mountain spotted fever, leptospirosis, and measles) must be negative. Imaging does not play a role in diagnosing TSS per se, unless the diagnosis is unclear and other diagnoses need to be excluded.

The most important consideration in treatment of either necrotizing soft tissue infections or TSS (which may coexist) is arriving at the correct diagnosis. One cannot treat what one fails to diagnose. Early treatment of TSS includes aggressive fluid therapy to maintain hemodynamic status, as well as appropriate antibiotic therapy. Antibiotic therapy should include coverage for both *Staphylococcus aureus* and *Streptococcus pyogenes*.

Clindamycin is the mainstay of antibiotic treatment for TSS, as it decreases toxin synthesis.<sup>[15-17]</sup> The primary treatment of necrotizing fasciitis and gangrene is rapid surgical debridement. However, like TSS, antibiotics and aggressive fluid resuscitation should also be initiated immediately. The inhibitory effect of clindamycin on protein synthesis, termed the "Eagle effect," provides a synergistic effect to its bacteriostatic activity. For this reason, it is a first-line agent for both TSS and necrotizing fasciitis.<sup>[16]</sup> Second-line agents such as vancomycin, linezolid, or daptomycin should also be considered to improve coverage of Gram-positive species, including methicillin-sensitive and methicillin-resistant *Staphylococcus aureus*. In addition, there is at least some literature to suggest a potential synergistic role of clindamycin with beta-lactam antimicrobials in combating necrotizing soft tissue infections, particularly when streptococcal species are involved.<sup>[18,19]</sup> For these reasons, combination therapy with piperacillin/tazobactam, clindamycin, and vancomycin is preferable in patients without known penicillin hypersensitivity.

## Conclusions

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It is important for emergency clinicians to adopt the same mantra quoted in the orthopedic literature: "there are no hypochondriacs in casts".<sup>[1]</sup> Although infectious complications after cast and splint application are rare, they may be easily overlooked, and may result in significant morbidity and mortality. Many of these complications can be identified by simply removing the cast or splint, carefully inspecting the affected area, performing a thorough examination, and simply considering the diagnosis. In addition, it is prudent to maintain particular vigilance when treating a patient with a water-exposed cast, which may lead to moist padding, skin breakdown, and potential infection. In patients with suspected serious infections, aggressive fluid and antibiotic therapy should be initiated immediately with agents such as clindamycin, vancomycin, and piperacillin/tazobactam to broadly cover Gram-positive, Gram-negative, and anaerobic species.

## References

1. Halanski M, Noonan KJ. Cast and splint immobilization complications. *J Am Acad Orthop Surg* 2008;16(1):30-40.
2. Stasikelis PJ, Lee DD, Sullivan CM. Complications of osteotomies in severe cerebral palsy. *J Pediatr Orthop* 1999;19:207-10.
3. Carmichael KD, Goucher NR. Cast abscess: a case report. *Orthop Nurs* 2006;25:137-9.
4. Davids J, Greenville S. Skin surface pressure beneath an above-the-knee cast: plaster casts compared with fiberglass casts. *J Bone Joint Surg* 1997;79:565-9.
5. Berman A, Parks B. A comparison of the mechanical properties of fiberglass cast materials and their clinical relevance. *J Orthop Trauma* 1990;4:85-92.
6. Netzer G, Fuchs B. Necrotizing fasciitis in a plaster-casted limb: case report. *Am J Crit Care* 2009;18:286-7.
7. Hawkins B, Bays P. Catastrophic complication of simple cast treatment: case report. *J Trauma* 1993;34:760-2.
8. Aldape MJ, Bryant AE, Stevens DL. *Clostridium sordellii* infection: epidemiology, clinical findings, and current perspectives on diagnosis and treatment. *Clin Infect Dis* 2006;43:1436-46.
9. Wong C, Khin L, Heng K, Tan KC, Low CO. The LRINEC (Laboratory Risk Indicator for Necrotizing Fasciitis) score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections. *Crit Care Med* 2004;32:1535-41.
10. Stevens D, Bisno A, Chambers H, et al. Practice guidelines for the diagnosis and management of skin and soft-tissue infections. *Clin Infect Dis* 2005;41:1373-406.
11. Cunningham J, Silver L, Rudikoff D. Necrotizing fasciitis: a plea for early diagnosis and treatment. *Mount Sinai J Med* 2001;68(4&5):253-61.

12. Majeski J, John J. Necrotizing soft tissue infections: a guide to early diagnosis and initial therapy. *South Med J* 2003;96:900–5.
13. Blankenship R, Baker T. Imaging modalities in wounds and superficial skin infections. *Emerg Med Clin North Am* 2007;25(1):223–34.
14. Centers for Disease Control and Prevention. Case definitions for infectious conditions under public health surveillance. *MMWR Recomm Rep* 1997;46(RR-10):39–40.
15. Schlievert PM, Kelly JA. Clindamycin-induced suppression of toxic-shock syndrome-associated exotoxin production. *J Infect Dis* 1984;149:471.
16. Eagle H. Experimental approach to the problem of treatment failure with penicillin. I. Group A streptococcal infection in mice. *Am J Med* 1952;13:389–99.
17. Stevens D, Bryant A, Yan S. Invasive Group A Streptococcal infection: new concepts in antibiotic treatment. *Int J Antimicrob Agents* 1994;4:297–301.
18. Sriskandan S, McKee A, Hall L, et al. Comparative effects of clindamycin and ampicillin on superantigenic activity of *Streptococcus pyogenes*. *J Antimicrob Chemother* 1997;40:275–7.
19. Zimbelman J, Palmer A, Todd J. Improved outcome of clindamycin compared with beta-lactam antibiotic treatment for invasive *Streptococcus pyogenes* infection. *Pediatr Infect Dis J* 1999;18:1096–100.