**Abstract:**
A 52-day-old infant presented to the emergency department with newly discovered fractures of the left arm, including fractures of the distal humerus and distal radius and an ulnar metaphyseal corner fracture. Initial presentation was concerning for nonaccidental trauma. Review of systems and physical examination findings suggested an alternative etiology. Further radiographic studies and laboratory results revealed the ultimate diagnosis.

**Keywords:**
infant; fractures; congenital syphilis; neurosyphilis

†Pediatric Emergency Medicine, Children's Hospital Colorado, Aurora, CO; †Pediatric Emergency Medicine Fellowship Program, Division of Pediatric Emergency Medicine, Assistant Professor of Pediatrics, University of Colorado School of Medicine, Children's Hospital Colorado, Aurora, CO.
Reprint requests and correspondence: Rachel Cafferty, MD, Clinical Fellow, Pediatric Emergency Medicine, Children's Hospital Colorado, 13123 E 16th Ave, Aurora, CO 80045.

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# Extremity Fractures in an Infant

**Rachel Cafferty†, Michelle Alletag†**

A former 35-week preterm twin, now 52-day-old female infant presented as a transferred patient to the emergency department (ED) for evaluation of newly discovered fractures of the left arm, including fractures of the distal humerus and distal radius and an ulnar metaphyseal corner fracture. The infant had been in foster care since birth along with her twin brother. Her foster family denied any prenatal complications or familial medical conditions. The foster mother described 5 days where the infant had progressively stopped using her left arm. For approximately 24 hours prior to presentation, the infant had no purposeful movement of the left arm and increased irritability with any repositioning or manipulation of that extremity. The family denied any known trauma or injuries but suggested that her physical therapy session the week prior might have created the newly identified fractures. The infant was otherwise at her baseline, acting well and moving all her other extremities without difficulty.

On review of systems, the foster mother noted that the infant has had a new rash for the past week, treated with hydrocortisone ointment at the recommendation of a dermatologist. The rash initially began as small erythematous papules on the buttocks and subsequently spread to the chest and extremities despite use of the topical steroid. The rash had also become more dusky and bruise-like in appearance. The infant had chronic nasal congestion and rhinorrhea since birth, for which parents were using an infant nasal aspirator daily. She had no cough, fever, difficulty breathing, apnea, or cyanosis. The infant had no irritability, lethargy, feeding intolerance, vomiting, or seizures.

The infant was born via cesarean delivery at 35 weeks' gestation. She required 4 days in the newborn intensive care unit for "respiratory concerns" and was subsequently discharged to foster care. There was a maternal history of polysubstance use and hepatitis C but no known prenatal complications. The infant

had otherwise been well without any prior medical concerns or previous surgeries. The foster parents had been applying topical hydrocortisone 0.5% ointment but no other daily medications. The infant had no known medication allergies. She had not yet received her 2-month vaccination series but received hepatitis B vaccine, vitamin K, and erythromycin after birth. She was living with her foster parents and twin brother since hospital discharge. The infant and her twin brother did not attend daycare and were exclusively cared for by their foster parents and extended foster family members. The foster mother denied any concern for infant maltreatment in the home environment.

On arrival to the ED, initial vital signs included a temperature of 36.8°C, heart rate of 158 beats per minute, respiratory rate of 60 breaths per minute, oxygen saturation of 100% on room air, and blood pressure of 99/87 mm Hg (while fussing). On examination, the infant was awake and alert. She had a strong cry and was irritable with repositioning but consoled appropriately when swaddled. Result of head, eyes, ears, nose, and throat examination was unremarkable with a soft and flat anterior fontanelle, no evidence of head injury, intact frenulum, and moist mucosa. Cardiac and pulmonary examination results were normal without murmur or adventitious respiratory findings. Abdominal examination revealed hepatomegaly with the liver tip palpated 1 to 2 finger breaths below the right costal margin as well as a small, reducible umbilical hernia. There was no splenomegaly, and the abdomen was otherwise soft, nontender, and nondistended. Genital examination result was normal. Musculoskeletal examination showed no obvious deformity, swelling, or tenderness, and the infant was moving all extremities equally and spontaneously, including the left upper extremity —with no deformity or paralysis noted. Skin findings were notable for several small erythematous macules involving the extremities and trunk (each approximately 1 cm in diameter) with a white rim and with central clearing on some lesions but no obvious plaque or scale (Figure 1). Lesions on the palms and soles were also noted with a dusky red/brown coloration that were blanching and nonpainful to the touch (Figure 2). There was no mottling, jaundice, or bruising of the skin. The neurologic examination result was normal.

Initial laboratory results were obtained including a complete blood count (CBC), comprehensive metabolic panel (CMP), lipase, type and screen, urinalysis, and urine toxicology screen. The CBC was unremarkable with a normal white blood cell count ($12.3 \times 10^3/\mu L$), normal hemoglobin (11.0 g/dL), and normal platelet

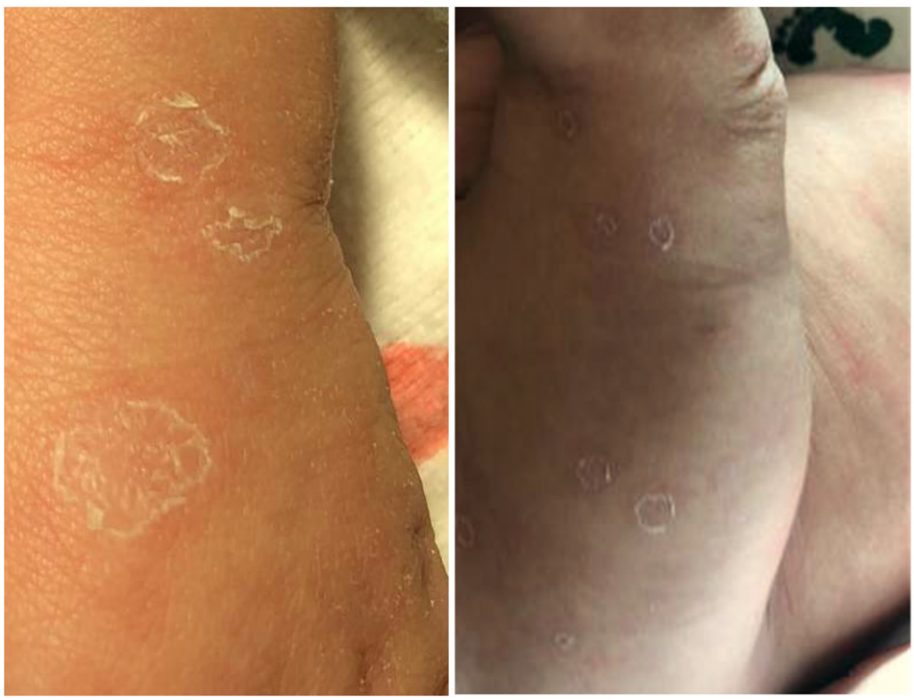

**Figure 1** Appearance of rash on trunk and extremities. Rash consists of small circumferential lesions with an erythematous base, white rim, and central clearing and no obvious plaque or scale.

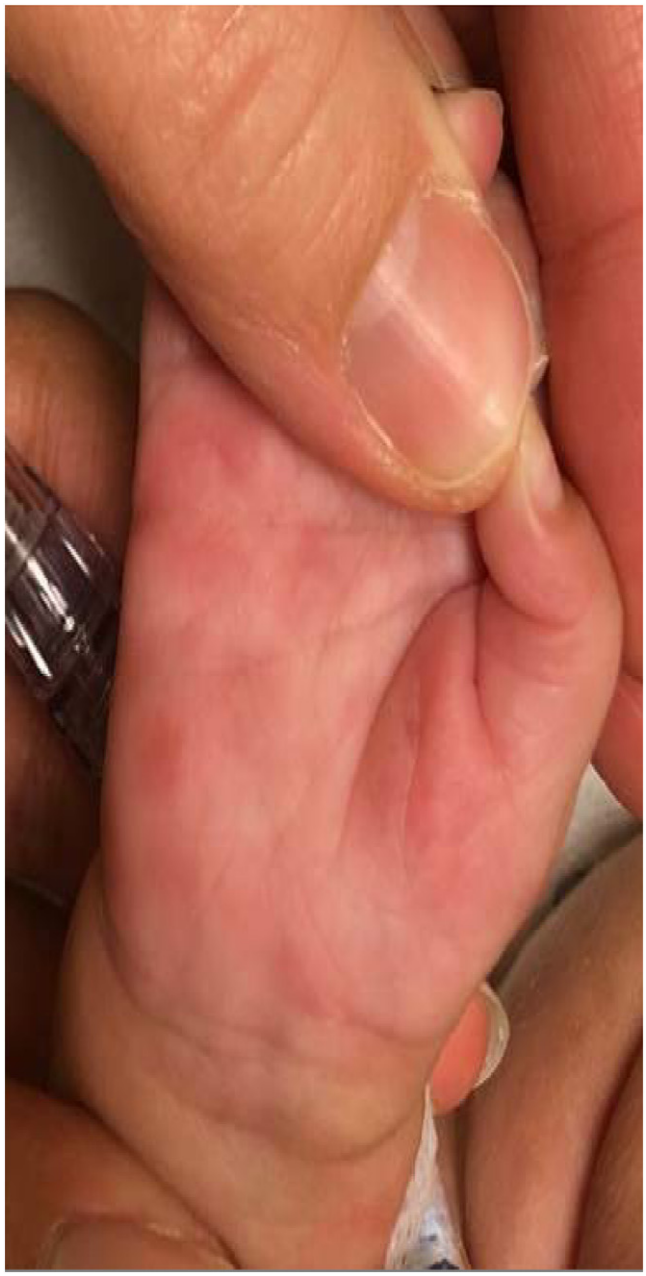

**Figure 2** Appearance of rash on palms and soles. Lesions are dusky red/brown in color, blanching, and nontender.

count ($307 \times 10^3/\mu L$). The CMP was notable for mildly elevated potassium of 5.6 mmol/L without hemolysis, mildly elevated total bilirubin at 1.3 mg/dL, elevated alkaline phosphatase at 974 U/L, and elevated alanine aminotransferase (ALT) and aspartate aminotransferase (AST) (112 U/L and 128 U/L, respectively). Lipase was normal at 45.5 U/L. Urinalysis was normal and urine toxicology screen was negative.

A head computed tomography (CT) scan without contrast and skeletal survey were completed. The head CT was negative for skull fracture or intracranial hemorrhage. Skeletal survey was notable for diffuse osteopenia with bilateral, symmetrical periosteal reaction in multiple long bones as well as radiolucency at the metaphyses, bony irregularity (most pronounced in the tibia and fibula), and cupping of the distal

fibula (Figure 3). There were no signs of healing callus seen on radiographic studies.

Given the child's history, examination, and diagnostic findings, further laboratory testing was performed, which ultimately revealed the diagnosis.

## DIFFERENTIAL DIAGNOSIS

In a nonambulatory infant with newly discovered fracture(s), nonaccidental trauma (NAT) must be considered. This infant had a metaphyseal corner fracture (bucket handle fracture) which is the fracture pattern most specific for NAT in infancy.[1] Metaphyseal corner fractures occur almost exclusively in children less than 2 years of age and are the result of force applied during to-and-fro manipulation (ie, when infants and young children are shaken and cannot protect their limbs, resulting in shearing forces to extremities). The infant in this case also had elevated transaminases (ALT 112, AST 128) which are concerning for occult intra-abdominal injury in cases of NAT. Among children less than 60 months of age referred for possible physical abuse, a threshold of >80 for both ALT and AST yields a sensitivity of 77% and a specificity of 82% for NAT.[2] In 1 study sample, 26% of cases had clinically occult abdominal trauma with elevated transaminase levels and injury on further imaging despite absence of abdominal bruising, distension, or tenderness.[2]

A high index of suspicion for NAT and recognition of concerning injury patterns is paramount toward establishing this diagnosis. Failure to recognize sentinel injuries or early child abuse can result in fatal complications for the patient. It is important for providers to distinguish bruising patterns or signs of inflicted injury to help delineate NAT from other organic etiologies. In children less than the age of 4 years, bruising on the torso, ear, or neck and bruising to any region of the body in an infant under the age of 4 months are highly sensitive and specific for predicting abuse.[3] Bruising or injuries to the frenulum, angle of the jaw, auricular region, cheek, eyelid, scleral hemorrhages, and patterned injuries or petechiae are also concerning for NAT. Workup for suspected NAT should include screening laboratory tests (CBC, CMP, lipase, coagulation panel, urinalysis, and urine toxicology screen). Infants less than the age of 6 months should have CT imaging of the head in addition to a skeletal survey when evaluating for potential NAT. In patients ages 6 to 24 months, a skeletal survey is routinely performed as part of an NAT evaluation, and head imaging is recommended for altered mental status, seizure, or enlarged head size (OFC greater than the 95th percentile). Subdural hematoma, cerebral ischemia, or the combination of a skull fracture and intracranial injury is highly associated with abusive head trauma.[4] Specific fracture patterns that raise concern for NAT include posterior rib fractures, long bone fractures of the humerus and/or femur (especially in nonambulatory children), complex skull fractures, and metaphyseal corner fractures, as previously discussed. These fracture patterns are also highly correlated with abusive head trauma.[4]

When managing infants or small children with multiple fractures, providers must also consider inherited conditions or predisposing fracture risks. Osteogenesis imperfecta (OI), also known as *brittle bone disease*, is a group of genetic disorders that primarily affect the bones. Mild cases of OI are characterized by fractures during childhood or adolescence resulting from minor trauma and may be associated with blue/gray discoloration of the sclera as well as hearing loss in adulthood. More severe forms of OI (including lethal perinatal form type II and types III to IX, XV, and XVI) result in fractures in utero and frequent fractures from little or no apparent cause after birth. Individuals with impaired or deficient bone mineralization (Rickets or osteomalacia) may have skeletal deformities (bowing of long bones), radiographic findings of osteopenia, cupping at the growth plate, pathologic fractures, and/or pseudofractures (termed *Looser's zones*) which are bilateral, symmetric, and lie perpendicular to the cortical margins of bones.[5] This is in contrast to the patient in this case who had diffuse, symmetric, and bilateral long bone periosteal reactions.

The examination findings of a diffuse rash involving the palms and soles change the differential diagnosis and include both infectious and noninfectious etiologies. Numerous infectious etiologies result in rashes that may involve the palms and soles, including but not limited to the following: congenital syphilis (*Treponema pallidum*); congenital rubella; congenital cytomegalovirus; neonatal herpes simplex virus; Rocky Mountain spotted fever (*Rickettsia rickettsia*); hand, foot, and mouth disease (coxsackie virus and enterovirus); meningococcal infection (*Neisseria meningitidis*); rat-bite fever (*Streptobacillus moniliformis*), papular-purpuric gloves and socks syndrome (human parvovirus B19); and ringworm (*Tinea* species). Each rash has a classic distribution and characteristic pattern for spread on the body as well as defining appearance and clinical features. However, rashes can be nonspecific on ED presentation, so it is important to consider the many infectious etiologies, particularly those with more emergent or life-threatening implications associated with a missed or delayed diagnosis.

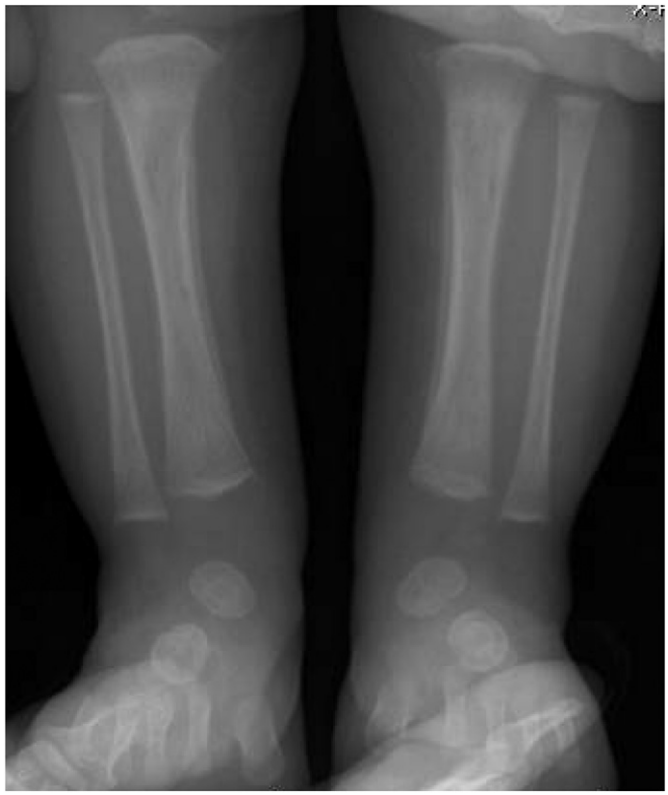

**Figure 3** Radiographic findings on skeletal survey, demonstrating diffuse osteopenia, symmetrical periosteal reaction of bilateral tibia, radiolucency at the metaphyses, and cupping of the distal fibula.

Noninfectious conditions that may result in a rash on the palms and soles include Kawasaki disease, serum sickness, drug reaction, dyshidrotic dermatitis (palmoplantar eczema or pompholyx), psoriasis, and palmoplantar keratoderma. The rash of Kawasaki disease can be nonspecific and transient, but other features of the disease help in making this diagnosis. A medication/drug trigger can often be identified as correlating with a more characteristic rash pattern when considering serum sickness or acute drug reaction as well as more serious skin manifestations such as Stevens-Johnson syndrome and toxic epidermal necrolysis. See Table 1 for a description of both infectious and noninfectious rashes with involvement of the palms and soles.

## CASE PROGRESSION AND DIAGNOSIS

After the skeletal survey was completed, the ED physician and attending radiologist discussed the diffuse symmetrical periosteal reaction and metaphyseal irregularities, which can be findings seen in both NAT and congenital syphilis. The trauma surgery team, child abuse specialists, and social work service were involved in the case given concern for potential NAT. However, in the setting of rash and chronic rhinitis, congenital syphilis became the leading diagnosis. Further diagnostic testing was performed, including serum rapid plasma reagin (RPR), serum fluorescent treponemal antibody absorption (FTA-ABS), and a lumbar puncture to collect cerebral spinal fluid (CSF) for venereal disease research laboratory (VDRL) evaluation. The infant was also tested for human immunodeficiency virus (HIV) and hepatitis.

The serum FTA-ABS and RPR were reactive (RPR titer of 1:512), confirming the diagnosis of congenital syphilis. The patient was admitted to the general pediatrics service and started on intravenous (IV) penicillin G. After the first dose of antibiotics, the infant developed the Jarisch-Herxheimer reaction, characterized by fever and tachycardia, which was treated supportively. A peripherally inserted central catheter was placed for a presumed 10-day treatment course. On day 3 of hospitalization, the CSF VDRL resulted as reactive with a titer of 1:4, consistent with neurosyphilis. As a result, IV penicillin G treatment was extended to a 14-day treatment course. During hospitalization, the infant had an ophthalmology and audiology evaluation, results of which were both normal. HIV and hepatitis test results were negative. The remainder of her hospital course was uneventful, and the infant was discharged on hospital day 16. She

**TABLE 1 Infectious and noninfectious etiologies for rashes which may have palm and sole distribution, displaying the characteristic rash presentation and pattern of spread**

| Diagnosis | Characteristic Rash Presentation |
|---|---|
| Congenital syphilis (*T pallidum*) | Maculopapular rash consisting of small dark red-copper lesions most pronounced on the palms and soles but also prominent on the back, buttocks, and posterior thighs. Desquamation and crusting may be a late finding.[15] |
| Congenital rubella | Petechiae and purpura "blueberry muffin" lesions, which indicate extramedullary hematopoiesis.[15] |
| Congenital cytomegalovirus | Diffuse petechiae and purpura, often associated with jaundice.[15] |
| Neonatal herpes simplex virus | Coalescing or clustering vesicular lesions with an erythematous base which may be localized or disseminated.[15] |
| Rocky Mountain spotted fever (*R rickettsia*) | Blanching, erythematous macules that evolve into petechiae with time. Rash initially presents on the ankles and wrists and spreads toward the trunk. Involvement on the palms and soles is hallmark but typically occurs with late-stage disease.[15] |
| Hand foot and mouth disease (coxsackie virus and enterovirus) | Vesicular or papulovesicular rash on the hands, feet, and oral mucosa. Lesions are nonpruritic but occasionally painful. Lesions on the hands and feet often involved the dorsal aspect of the digits, interdigital space, palms, and soles. Lesions on the buttocks and upper thighs are also common. An oral enanthem (vesicles on the tongue and buccal mucosa) often accompanies the exanthem.[15] |
| Meningococcal infection (*N meningitidis*) | A maculopapular eruption (resembling a viral exanthem, particularly rubella) may transiently appear as an early finding in meningococcemia. Rash rapidly progresses to petechial lesions, which may coalesce into larger purpura.[15] |
| Papular-purpuric gloves and socks syndrome (human parvovirus B19) | Rapidly progressive edema and erythema of the hands and feet with subsequent papules, petechiae, and/or purpuric lesions. Rash has characteristic acral distribution with sharp demarcation at wrists and ankles.[15] |
| Ringworm (*Tinea* sp.) | Pruritic, circular, erythematous lesions with scaling patch or plaque that spreads centrifugally. Progression of rash results in central clearing of lesions with raised erythematous border remaining, resulting in ring-shaped plaque. Plaques may coalesce. Tinea corporis may involve palms and soles. Tinea pedis ("athlete's foot") may have interdigital lesions, moccasin-type hyperkeratotic erythematous lesions with diffuse eruption involving soles, or infrequently a vesiculobullous presentation on the medial aspect of the feet.[15] |
| Rat-bite fever (*S moniliformis*) | Maculopapular, purpuric, or petechial rash predominantly involving the peripheral extremities, including the palms and soles. Lesions may become confluent and may desquamate.[15] |
| Kawasaki disease | Polymorphous, generalized rash that may be morbilliform, maculopapular, scarlatiniform, or erythema multiforme–like. Associated changes in the peripheral extremities include erythema of the palms and soles, induration of hands and feet, and periungual desquamation. Kawasaki disease may trigger a psoriasiform eruption in children not previously diagnosed with psoriasis.[15] |
| Dyshidrotic dermatitis (palmoplantar eczema, pompholyx) | Deep-seated vesicles or bullae localized to the palms and soles, often involving the lateral aspect of the fingers. Rash is intensely pruritic.[16] |
| Pustular psoriasis | May present with pustules limited to the palms and soles (pustulosis palmaris et plantaris) or intertriginous distribution beginning in infancy. Pustular psoriasis of the palms and soles is differentiated from pompholyx by the presence of cloudy, purulent fluid within vesicles and more chronic presentation.[16] |
| Palmoplantar keratoderma | Acral hyperkeratosis which can be diffuse or limited to regions of hands and feet but often present on the dorsal aspect. Lesions are translucent hard papules that may have a central depression and resemble flat warts.[16] |
| Serum sickness | Pruritic lesions which may be urticarial in nature but longer-lasting than hives. Skin changes may be generalized to involve the feet and hands or limited to the trunk. The rash has a variable presentation and may include palpable purpura, morbilliform eruptions, papules, maculopapular lesions, or palmar erythema. Mucus membranes are not involved.[16] |
| Drug reaction | Exanthematous drug eruption is the most common type of drug hypersensitivity reaction and typically presents as a morbilliform or maculopapular eruption. Rash consists of diffuse, symmetric erythematous macules or small papules after initiation of a drug treatment.[16] |

Table 1 (continued)

| Diagnosis | Characteristic Rash Presentation |
|---|---|
| Stevens-Johnson syndrome, toxic epidermal necrolysis | Severe mucocutaneous eruptions triggered by medications. Skin lesions are initially ill-defined and then coalesce, forming erythematous macules with purpuric centers. Targetoid lesions, diffuse erythema, epidermal necrosis, and sloughing of the distal extremities, including palms and soles, can occur (but are rarely involved). Classically, lesions start on the face and thorax before spreading and have a symmetrical distribution. Lesions affect <10% of the body surface in SJS and >30% of body surface in TEN.[16] |

has had outpatient follow-up with ophthalmology which was unremarkable, and no ocular manifestations of syphilis were noted. She has had normal hearing on subsequent audiology evaluations. Repeat serum RPR has remained reactive, with a downtrended titer most recently to 1:4 and CSF VDRL that was negative 6 months after initial diagnosis and treatment.

Interestingly, the twin sibling was also subsequently admitted for evaluation and treatment of presumed congenital syphilis. He was noted to have worsening congestion and rhinorrhea for 1 to 2 weeks prior to presentation as well as questionable decreased movement of 1 arm that had been gradual and less severe. He did not have a rash. His admission physical examination was notable for nasal discharge, a small reducible umbilical hernia, and decreased use of the left upper extremity with an asymmetric Moro reflex due to the decreased response on the left side. He had a peripherally inserted central catheter line placed for planned 10- to 14-day treatment with IV penicillin G and similarly developed the Jarisch-Herxheimer reaction after initiation of penicillin. His serum FTA-ABS and RPR were reactive (initial RPR titer of 1:512). CSF VDRL was negative. Other laboratory test results of interest included negative HIV and nonreactive hepatitis B as well as anemia and elevated transaminases (ALT 235, AST 245). A skeletal survey was obtained and revealed diffuse, symmetric long bone periostitis. Results of ophthalmology and audiology evaluations were normal during his hospitalization. Despite negative CSF VDRL, given his high RPR titer and twin sibling with neurosyphilis, he was treated with a 14-day course of penicillin. He has had an uneventful outpatient course with downtrended serum RPR (most recently 1:64) and normal ophthalmology and audiology evaluation results.

## CASE DISCUSSION

Over the past decade, national trends have demonstrated increased rates of primary and secondary syphilis in the United States, particularly in Western states.[6] Syphilis is caused by the spirochete *T pallidum*, which is usually sexually transmitted but can be spread by vertical transmission, transplacentally from an infected mother to her unborn fetus at any stage of pregnancy, resulting in congenital syphilis in infants. Antenatal treatment with a single dose of penicillin G is >98% effective at preventing congenital syphilis.[7] About 40% of pregnancies in women with untreated syphilis end in perinatal death,[8] highlighting the importance of prenatal testing and maternal treatment with penicillin. Rising rates of adult syphilis with poor prenatal care and low treatment rates have resulted in a recent rise in cases of congenital syphilis.[9] In 2017, there were 918 cases of congenital syphilis, the highest number of reported cases in the past 20 years.[10]

Clinical manifestations of *early* congenital syphilis typically present between 3 weeks and 3 months of age but can manifest at any point before 2 years of age. Infants may develop syphilitic rhinitis (the "snuffles") as early as the first week of life. Approximately 1 to 2 weeks after onset of rhinitis, a nonspecific red/pink rash develops which may progress to a copper/brown color with or without desquamation or scaling, involving the palms and soles, buttock, and thighs. Both syphilitic rhinitis and cutaneous lesions contain spirochetes and are highly contagious through direct contact exposures. Diffuse osteopenia and fracture patterns as demonstrated in this case are a common finding in congenital syphilis, resulting in pain to the affected infant. Pseudoparalysis of Parrot (lack of movement of an extremity secondary to pain from bony lesions) is less common but pathognomonic for the disease. Pseudoparalysis of Parrot more commonly involves upper extremities, is usually unilateral, and is poorly correlated with radiographic abnormalities. Other early findings in congenital syphilis include lymphadenopathy, hepatosplenomegaly, jaundice, anemia, or CNS involvement.[11]

*Late* congenital syphilis is characterized by Hutchinson's triad which includes interstitial keratitis, sensorineural hearing loss (cranial nerve eight deafness), and Hutchinson teeth (blunted upper incisors). Interstitial keratitis involves bilateral corneal scarring and secondary glaucoma, resulting in blindness that often manifests around puberty. Sensorineural hearing loss is most common between ages 8 and 10, with hearing loss of high frequency sounds occurring first. In addition to Hutchinson teeth, patients may have mulberry molars or perforation of the hard palate (which is virtually pathognomonic for congenital syphilis). Characteristic facial features of late congenital syphilis include frontal bossing and saddle nose deformity. Skeletal deformities, such as anterior bowing of the shins (saber shins), are also commonly seen.[11]

The classic radiographic finding in congenital syphilis is periostitis (irregular periosteal thickening) that is symmetric and bilateral and typically involves multiple long bones. Other radiographic findings of congenital syphilis include Wegner sign (metaphyseal serration or "sawtooth metaphysis") and Wimberger sign (demineralization and osseous destruction of the upper medial tibia). The bony lesions described above are considered pathognomonic for congenital syphilis.[12,13] There is no callus formation to suggest healing fractures, which further helps delineate radiographic findings from NAT.

Laboratory workup for congenital syphilis includes serum RPR and FTA-ABS testing as well as CSF VDRL. Reactive CSF VDRL dictates neurosyphilis. Congenital syphilis is treated with IV penicillin G, with a longer duration (14 days) of treatment for cases of neurosyphilis. The Jarisch-Herxheimer reaction is observed in 50% of patients with primary syphilis and in about 90% of patients with secondary syphilis.[14] The reaction is associated with initiation of antimicrobial treatment, which results in lysis of bacterial cell membranes and release of bacterial toxins into the bloodstream creating a systemic inflammatory response. Although the reaction is usually not life-threatening and is self-limiting, it may resemble bacterial sepsis. Patients may develop fever, chills, rigors, tachycardia, hypotension, hyperventilation, flushing of skin, myalgias, and exacerbation of skin lesions. Treatment is often supportive, primarily involving the use of antipyretics.

## SUMMARY

This was the case of an infant with newly discovered radiographic findings, including metaphyseal corner fracture, concerning for child maltreatment. Pediatric emergency care providers must quickly recognize clinical findings and diagnostic workup that support a diagnosis of NAT, as failure to recognize NAT can be life-threatening for an infant or child. It is important to cast a wide net and broaden the differential beyond NAT when other symptoms or signs are present, as was the case for this infant. Given the constellation of symptoms including persistent copious rhinorrhea and new rash involving the palms and soles, congenital syphilis became the leading diagnosis. Rates of congenital syphilis are increasing in the United States, and what was once considered a rare diagnosis in pediatrics is becoming far more commonly encountered in practice. Providers in the pediatric ED should be able to recognize key features of this disease, understand the diagnostic process, and be comfortable initiating treatment for congenital syphilis.

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