Indian Journal of Medical Case Reports ISSN: 2319–3832(Online)
An Open Access, Online International Journal Available at http://www. cibtech.org/jcr.htm
2015 Vol. 4 (2) April-June, pp.49-55/Radhika and Lakshmi
Case Report

LIFE THREATENING INFECTIONS IN PEDIATRIC EMERGENCY

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ABSTRACT
Life threatening infections in the emergency room present with cardiovascular or respiratory compromise or with manifestations related to the central nervous system. Infections that involve the skin and have a rapid onset and a fulminant course are not very common in the pediatric emergency department. Infections that are not commonly seen are challenging in terms of recognition and management. Recognition of some of these infections might be difficult due to the rarity and it is important to have knowledge of specific management of some of these life threatening infections. We report three children who presented with fatal infections with dermatological manifestations.

Keywords: *Dermatology, Infections, Pediatric Emergency*

CASES
Case 1
3 years old female child, presented with cold septic shock and was resuscitated with fluid boluses, inotrope infusions and intubated. She had a bluish discoloration of the skin over the lateral aspect of her left upper thigh, left lateral gluteal area which extended to the lower left abdominal wall (Figure 1). There were a few blebs over the same area with serosanguineous discharge without crepitus. She had fever for 2 days and received an intramuscular injection at a local hospital. A small area of bluish discolouration was noticed over the upper left thigh 6 hours prior to admission with rapid progression. She had no significant illness in the past. The clinical picture was suggestive of necrotising fascitis probable gas gangrene with septic shock and she received an intravenous meropenem and clindamycin during resuscitation in the ER. A swab for gram stain and culture was taken from the skin lesions and she was transferred to pediatric intensive care unit where debridement and intensive care was continued. She expired 72 hours after admission due to refractory shock associated with rapid progression of skin infection involving her chest. The gram stain from her wound was positive for clostridim perfringens but wound culture was negative as anerobic culture was not done.

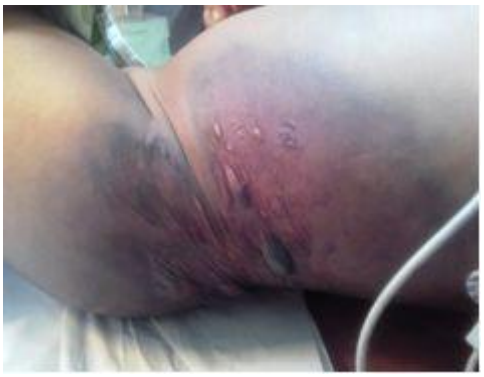

Figure 1

DISCUSSION
Necrotising Fasciitis (NF)
Necrotizing **fasciitis** is a rare, rapidly progressive and potentially fatal infection of the superficial fascia and subcutaneous cellular tissue (Kosloske, 1981). Etiology is frequently polymicrobial, and a

combination of aerobic and anaerobic bacteria causes the severe and quick progression of infection (Gluliano, 1977). Necrotizing fasciitis is rare in children and has been reported in 0.03% of hospitalization causes and in 0.08 per 100000 children per year (Wilson, 1973; Laupland, 2000). In children NF is frequently misdiagnosed and differs from NF in adults (Antonio, 2002). Though immune suppression and malnutrition are common predisposing factors, minor injuries including intramuscular injection can trigger NF (Oncel, 2010). Mortality due to gas gangrene is reported to be between 15% and 35%. NF secondary to C. perfringens (gas gangrene) is an uncommon but life-threatening and limb-threatening condition which causes progressive invasion and destruction of healthy muscle tissue. About 80 percent of gas gangrene due to traumatic injury is caused by Clostridium perfringens (Awad, 1995). The mean incubation period is less than 24 hours (6 hours to several days), depending on the size of the bacterial inoculum and the extent of vascular compromise. Traumatic gas gangrene usually presents with sudden onset of severe pain at the site of surgery or trauma due to the toxin-mediated ischemia (Weinstein, 1973). NF often initially presents as pain out of proportion to other skin finding. The skin over the infected area appears pale to start with then rapidly develops a bronze appearance, followed by purple or red discoloration and becomes tense and exquisitely tender. Overlying bullae that are clear, red, blue, or purple develop with reddish watery discharge. Signs of systemic toxicity develop rapidly including tachycardia and fever, followed by shock and multi organ failure. Shock has been reported in 50 percent of patients at the time of presentation to the hospital (Hart, 1983). Pain at a site of traumatic injury together with signs of systemic toxicity and gas in the soft tissue support the diagnosis of gas gangrene. Physical evidence of crepitus in the soft tissue is the most sensitive and specific finding on clinical examination. Complications of clostridial myonecrosis include jaundice, renal failure, hypotension, and liver necrosis (Gozal, 1983).

Radiographic imaging can be useful for identifying gas in deep tissues. Blood (both aerobic and anaerobic bottles) and tissue cultures should be obtained and a definitive diagnosis of gas gangrene requires demonstration of large, gram-variable rods at the site of injury Treatment of traumatic gas gangrene consists of immediate resuscitation, appropriate antibiotic therapy, and surgical debridement and anti tetanus measures.

Surgical exploration or debridement is an important diagnostic, as well as therapeutic, procedure and typically demonstrates muscle that is grossly edematous and may have reddish-blue to black discoloration but does not bleed. Microscopic evaluation of biopsy material invariably demonstrates organisms among degenerating muscle bundles and a characteristic absence of acute inflammatory cells (Gorbach, 1992) prompt aggressive, and thorough surgical inspection and debridement of devitalized tissue are mandatory to improve survival, preserve limbs, and prevent complications (Gorbach, 1992; Lorber, 2005).

Widespread tissue necrosis and the characteristic absence of tissue inflammatory response are due to the alpha toxin. Alpha toxin potently stimulates platelet aggregation and reduces the ability of neutrophils to cross the endothelial cell barrier into infected tissue and destroys those which migrate. The anaerobic environment is facilitated by poor perfusion causing rapidly advancing margins of tissue destruction characteristic of clostridial gas gangrene.

Alpha toxin directly suppresses myocardial contractility and may contribute to profound hypotension via a sudden reduction in cardiac output (Stevens, 1988).

In the absence of a definitive etiologic diagnosis, broad-spectrum treatment with vancomycin plus either piperacillin/tazobactam, ampicillin/sulbactam, or a carbapenem is recommended for NF.

Definitive antimicrobial therapy with high-dose (250,000 U - 400,000 U/kg) intravenously administered penicillin G in combination with clindamycin should be started as soon as possible for treatment of clostridial myonecrosis (Dennis, 2014). Hyperbaric oxygen (HBO) therapy is not recommended because it has not been proven as a benefit to the patient and may delay resuscitation and surgical debridement (Stephen, 2014).

Emergency medicine physicians who treat these children should recognize this severe and potentially fatal infectious process and should not delay resuscitation and prompt surgical consultation (Temple, 2004).

Case Report

Case 2

7 years old boy was brought to the Emergency room with fever of 5 days duration and vesicular lesions noticed by the mother on abdomen, rapidly progressing to involve his entire skin surface. He received native medication for the current illness. He had a poor oral intake with poor urine ouput for 24 hours. He had no significant illness in the past and had no history of drug ingestion or contact with varicella. On examination he was febrile, toxic, dehydrated and had extensive monomorphic vesicuar lesions from head to toe with involvement of the oral mucosa and the vesicles were filled with hemorrhagic fluid with bleeding from some of the lesions (Figures 2, 3). There were some lesions that were crusting. He was lethargic. His vital parameters were heart rate of 156/min, respiratory rate of 26/min, blood pressure of 108/76 mmHg and oxygen saturation was 98% in room air. His laboratory tests were suggestive of sepsis with dehydration. His laboratory parameters were as follows- Total WBC count of 17900 cells/cumm/, Neutrophils 83% Stab form10%, platelets 60,000 cells/ cumm, PCV 49% , blood urea 40 mg/dl, Hco3 16meq/l, Na 120meq/l, Alanine transaminase 498 IU/l, Aspartate transaminase 202 IU/l, serum creatinine, blood sugar, Prothrombin time and Partial thromboplastin time were normal. A diagnosis of hemorrhagic varicella was made based on the characteristic extensive monomorphic hemorrhagic vesicles that involved almost his entire body surface including the scalp, oral mucosa and conjunctiva. He was given treatment in the form of intravenous fluids for dehydration, acyclovir, antibiotic, care of eyes and skin. A confirmatory test could not be done as he was transferred to another tertiary care facility due to non availability of isolation in our intensive care unit. However clinically it was confirmed by the dermatologist that the presentation and lesions were consistent with a diagnosis of hemorrhagic varicella.

Figure 2

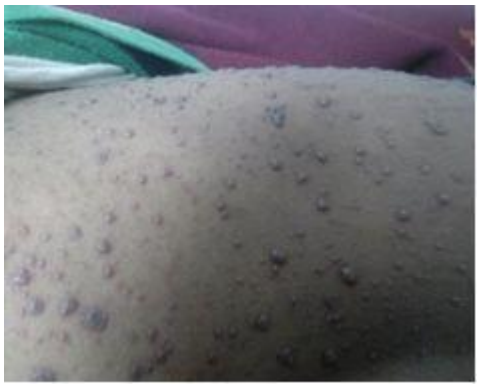

Figure 3

Discussion

Hemorraghic Varicella-zoster

Varicella is most often a relatively benign and self-limited childhood illness, however rarely it can be associated with a variety of serious and potentially lethal complications especially in immunocompromised persons and the risk of disseminated disease can be as high as 36%. Pink book 12th ed. These persons may have multiple organ system involvement, and the disease may become fulminant and hemorrhagic. Hemorrhagic varicella has however been reported in immunocompetent individuals (Niederhoff, 1976). The predominance of uncomplicated cases in children tends to overshadow the morbidity and mortality associated with severe cases (Hiroshi, 1997). Hemorrhagic varicella is a rare but fatal complication which should be recognized. hemorrhagic rash has been reported in 3.3 % of varicella infections and haemorrhage into the skin, epitaxis, malena or haematuria sometimes occur during the course of varicella and usually presents 2 - 3 days after the onset of the rash (Kole, 2014). The haemorrhage may be so severe as to be lifethreatening. Malignant varicella with purpura carries a high mortality .The pathogenesis of thrombocytopenia and DIC in varicella infections is not clear but hemorragic varicella is associated with thrombocytopenia (Moffet, 2005). Data regarding incidence of hemorrhagic varicella is extremely limited especially from low and middle income countries. Risk factors

for severe varicella and death include age, altered immunocompetence and lack of access to health care (Jane, 2014). Case reports of deaths due to hemorhagic varicella have been described (Cooray, 1960). A continuous infusion of acyclovir may be beneficial for severe, life-threatening varicella–zoster virus infections (Hiroshi, 1997).

Due to the rarity of this fatal complication of varicella and as the clinical features overlap with other dermatological conditions especially Steven Johnson syndrome a high index of suspicion is needed to not only make a diagnosis of hemorrhagic varicella but it is essential that the emergency physician is aware of the mortality associated this condition and takes appropriate resuscitative measures. Immunosuppression should be considered for choice of antibiotics in the emergency room.

Case 3

5 yr old boy, without any significant illness in the past, was brought with fever of 2 days with lethargy .He was noticed to have bluish rashes which started over his trunk and progressed rapidly all over his body and has vomiting and loose stools (Figure 4). He had hypotensive shock and had bluish black patches all over his body and oral mucosa. A diagnosis of septic shock with meningococcemia was made. He was resuscitated with fluid boluses, ionotropes and intubated. After sampling for baseline cortisol and intravenous ceftriaxone, he was transferred to PICU where he received intensive care including IV hydrocortisone. His laboratory parameters indicated multiorgan dysfunction and he expired due to refractory shock. His blood culture was negative.

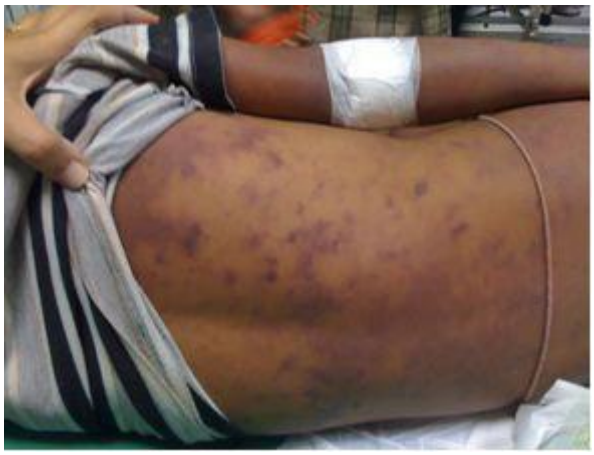

Figure 4

Discussion

Diffuse petechiae/purpura in a child with septic shock can indicate fulminant infection such as Rocky Mountain Spotted Fever / Meningococcemia or DIC. Meningococcal disease is endemic to specific geographical areas and is uncommon in south India (Basu, 1985; Kirubah, 2014). Meningococcemia characteristically has a very rapid onset and progression to death within hours (Akley, 1979). Meningococci may cause different clinical syndromes and invasion is a rare phenomenon but recognition of the critical condition and immediate resuscitation is crucial. Recognition of meningococcal sepsis is difficult due to a lack of awareness among health professionals in recognizing meningococcal purpura (Hazelzet, 2005). Delays in treatment occur when the rash is atypical of meningococcal septicaemia, and therefore decreases survival (Slack, 1978). Laboratory investigations are in the initial phase not helpful. The patients with fever, looking sick and a rapid onset of skin hemorrhages all over have a likelihood of >97% of having a meningococcal infection. The rash is characterized by dermal microvascular thrombosis and perivascular hemorrhage (Van Deuren, 2000). Recognizing the rashes of meningococcal septicaemia and initiating appropriate antibiotic without delaying treatment by looking for signs of meningitis by the emergency physician is vital (Farmer, 1993). Rocky mountain spotted fever (RMSF) may also mimic meningococcemia and toxic shock syndrome (Kristina, 2000).

RMSF usually presents with a prodrome of malaise, headache, and myalgia, with abrupt onset of high, oscillating fever and blanching macules concentrated on the peripheral extremities. Lesions on the palms and soles occur in 49 to 82% of cases (Thorner, 1998). The rash spreads centripetally, usually sparing the face, and may become petechial, progressing to ecchymosis and distal necrosis (Kirkland, 1995). Delayed diagnosis and late initiation of specific antirickettsial therapy has a higher risk of mortality. Most broad-spectrum antibiotics, including penicillins cephalosporins, and sulfa-containing antimicrobials, are ineffective treatments for RMSF (Dalton, 1995). Rapid laboratory confirmation of RMSF infection is not available hence clinicians should consider initiating empiric therapy in patients with a compatible clinical presentation and epidemiologic circumstance that could have exposed them to ticks , to reduce morbidity and mortality resulting from delayed diagnosis (O'Reilly, 2003). Serologic confirmation is generally not possible before the second week of illness and since delays in initiation of antibiotic therapy beyond the seventh day of illness are associated with increased mortality, empiric treatment of suspected RMSF with doxycycline is essential (Kirkland, 1995). Meningococcemia and RMSF are life threatening infections that need to be recognized by emergency physicians working in regions where they are uncommon to help early and appropriate resuscitation.

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