scars on the face and limbs. The abdomen was grossly distended and after aspiration of sterile ascitic fluid a mass could be palpated. The father explained the bruises as being due to the boy’s “unsteady gait and frequent falls.”

Serum amylase was 64 C.U. (normal 8 to 64 C.U.). At laparotomy, a pancreatic pseudocyst was found and drained. Recovery was uneventful.

DISCUSSION

Blunt abdominal trauma accounts for at least half the reported cases of pseudocyst of the pancreas in children. In some of the 30 per cent of reported cases in which the cause is not identified unsuspected trauma may actually be the cause. Abdominal trauma in school age children is usually the result of accidental injury—bicycle handlebars, contact sports, or traffic accidents. The situation is somewhat different in the case of children under 3 years of age. In this age group our experience leads us to believe that when a pseudocyst of the pancreas is found, and no acceptable explanation for its occurrence is forthcoming, the possibility of child abuse should be considered. We believe that traumatic pseudocyst of the pancreas due to child abuse is more common than the literature currently indicates, and that some of the reported cases of pseudocyst of the pancreas in children, especially those reported in infants and toddlers, may in fact be the result of child abuse.

REFERENCES


Fusarium solani infection during treatment for acute leukemia

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Fusarium, a genus of the Fungi imperfecti, derives its name from the fusiform spores. Fusarium spp. are common soil saprophytes and plant pathogens; when isolated from clinical materials they are usually considered to be contaminants.

This paper describes clinical manifestations of skin lesions, panophthalmitis, subcutaneous abscess, and possible aphony and encephalopathy due to Fusarium solani infection in a leukemic child. To our knowledge, disseminated Fusarium infection has not been reported in man.

CASE REPORT

Patient M. S., a 21/2-year-old Caucasian boy, had a history of acute lymphocytic leukemia for 11/2 years. Recent admission related to exacerbation of leukemia, for which he received L-asparaginase, daunomycin, vincristine, and prednisone. Five days after he was sent home from the hospital he developed low-grade fever, hoarseness, twitching of the right side of the face, and an erythematous maculopapular skin lesion below the left knee area. This lesion progressed into a blackish necrotic center surrounded by indurated erythema. Swabs of this lesion grew Proteus sp. and Escherichia coli.

Two weeks later he was readmitted because of recurrence of fever, appearance of new skin lesions, progression of seizure activity, aphony,
Fig. 1. Skin lesions associated with *F. solani* infection. a, Early lesion with erythema and slight induration. b, Late lesion showing necrotic center (scab detached) with surrounding erythema and induration. c and d, Corresponding lesion of a and b 5 weeks after amphotericin B therapy. Erythema and induration resolved.

inability to swallow liquids, and mental disorientation. There were multiple erythematous skin lesions (Fig. 1, a) involving the buttocks, flanks, abdomen, chest, neck, and chin. Many of these lesions became indurated and formed a blackish necrosis with scab in the center. These necrotic scabs eventually detached from the lesions (Fig. 1, b). The left orbit was reddened and swollen; ophthalmologic examination revealed panophthalmitis.

Admission blood studies revealed a hemoglobin of 8.0 Gm. per cent, hematocrit of 22 per cent, white blood count of 1,100 per cubic millimeter (2 per cent neutrophils, 96 per cent lymphocytes, and 2 per cent monocytes) with a platelet count of 17,000 per cubic millimeter. Sections of the necrotic scab lesions showed numerous hyphae and chlamydospores; a fungus, later identified as *F. solani*, was isolated repeatedly from the necrotic skin lesions, eye discharges, and the needle aspirates of the newly developed skin lesions (Fig. 1, a). Blood cultures were negative for bacteria and fungi.

He was given amphotericin B intravenously with a starting dose of 0.1 mg. per kilogram per day; the dosage was gradually increased to 1 mg. per kilogram per day within a week. Then he was maintained on 1 mg. per kilogram per day on alternate days for a total of 8 weeks. In addition, amphotericin B solution (1 mg. per milliliter) was applied topically to the involved eye. Twenty-four to forty-eight hours after amphotericin B therapy was started, no additional skin lesions were observed. He began to swallow some liquid and showed some return of voice. All necrotic scabs detached from the lesions spontaneously.

There was decreasing erythema about the eye and less drainage. Progressive, but slow, improvement of skin and eye lesions was observed over the next few weeks; however, he developed increasing seizure activity and mental confusion. Smears and cultures of spinal fluid for bacteria and fungi were negative. Three weeks after initiation of amphotericin B therapy a small abscess was noted at the right knee area. Eight milliliters of thick whitish fluid was aspirated from which *F. solani* was also isolated.

Healing processes were extremely slow; however, all erythematous lesions resolved and granulation tissues formed (Fig. 1, c and d). After 5 weeks of therapy *F. solani* was no longer isolated from the skin and eye lesions. At the end of
amphotericin B therapy his voice remained hoarse; extensive injury to the left eye hindered functional recovery; seizure activity and mental confusion remained unchanged and he continued to do poorly. Upon request of the parents he was transferred to a local hospital closer to the family. The patient died 2 weeks after the transfer. No postmortem examination was performed.

**MYCOLOGIC STUDIES**

Mycelium and chlamydospores of *F. solani* were easily seen in the sections of scab lesions (Fig. 2, a) and in preparations of skin from the erythematous area around the lesion or scabs hydrolized with 20 per cent potassium hydroxide solution. The fungus was easily isolated from the scabs or debris from the lesions (Fig. 2, b) but was less readily isolated from the exudate swabbed from the necrotic lesions. It grew on all isolation media not containing cyclohexamide and grew well but more slowly at 37°C. This fungus was readily identified to genus, but it was not easily differentiated to species even using the pictorial guide of Toussoun and Nelson.1

In vitro amphotericin B sensitivity tests indicated that the minimal inhibitory concentration for this fungus was 3.0 µg per milliliter. Amphotericin B blood levels at 24 hours and 48 hours were 0.59 µg per milliliter and 0.24 µg per milliliter, respectively. Methods for drug sensitivity test are similar to those described previously.2

**DISCUSSION**

Demonstration of *F. solani* in open wounds and dead tissue does not establish its pathogenicity. However, recovery of the organism from needle aspirates of carefully cleansed nonulcerated skin lesions and from an enclosed abscess suggests that the fungus is responsible for the disease in this patient. Early dramatic clinical responses to amphotericin B therapy indirectly supports our assumption. *Fusarium spp.* are opportunistic organisms. Recently *Fusarium spp.* have been associated with onychomycosis and keratitis in persons who had suffered accidental cornea injury.4 It has also been isolated from ulcerated legs,5 burn lesions,6 necrotic skin lesions of patients with diabetes mellitus,7 and facial granuloma of a child with phagocytic dysfunction.8 The pathogenetic role of *Fusarium spp.* in these skin lesions has not been established. The disseminated disease observed in our patient suggests that hematogenous spread was preceded by an initial colonization in the primary lesion. Compromised host resistance might have been responsible for the infection to become established.

Mycotoxins are known to be produced by *Fusarium spp.* Ingestion of contaminated grains may cause abdominal pain, nausea, diarrhea, vomiting, marked ataxia, bone marrow depression, convulsions, pharyngitis with ulceration resulting in aphonia, and death in man.5,6

The skin lesions of *F. solani* infection demonstrated in our patient were quite characteristic; they progressed slowly over a period of days from erythema and induration to blackish necrosis in the center. Such lesions should be differentiated from lesions of Pseu-
Glycogen storage disease
type IX: Benign glycogenesis of liver and hepatic phosphorylase kinase deficiency

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Increasingly, heterogeneity is being found among the various inborn errors of metabolism. The pedigrees of the initial patients with phosphorylase kinase deficient glycogenesis (glycogen storage disease type IX [GSD IX]) were consistent with autosomal recessive inheritance of this entity. Subsequently, a study on leukocytes appeared in which X-linked transmission was deemed more appropriate. In this report we present two male patients with GSD IX whose mothers are sibs, a feature consistent with X-linked inheritance. The diagnosis was based on the demonstration of increased hepatic glycogen concentration and deficient activity of phosphorylase kinase in the liver. Evidence also will be presented that this form of glycogen storage disease is a hepatic abnormality and not a generalized disorder.

CASE REPORTS

Case 1. Patient D. B. (KUMC 7201669), weighed 3,346 Gm. at birth and was noticed to have hepatomegaly. The developmental history was normal for the first few months of life; however, growth slowed and he was hospitalized at the age of 7 months for evaluation. At that time he was found to have a mild anemia and an enlarged liver, without abnormal liver function studies. No diagnosis was established. He continued to grow poorly over the next few months, and the asymptomatic hepatomegaly persisted. He was subsequently admitted to another hospital at the age of 3 years for further evaluation.

Physical examination at that time revealed a