Aspergillus Appendicitis

Fungi in very rare situations, mostly immunosuppressed children, can cause appendicitis. Aspergillus is a widespread fungus identified in the environment and usually enter the human body by airborne transmission colonizing the nasal cavities or fascial sinuses. Isolated gastrointestinal aspergillosis arises from ingestion of food contaminated with Aspergillus and colonization by Aspergillus of gastrointestinal ulcers which arise from previous chemotherapy. The diagnosis of isolated (primary) aspergillus appendicitis is very rare, is delayed and associated with profound immunosuppression. This last factor of immunosuppression causes the delay in the clinical features of appendicitis. The very few cases have demonstrated a clinical pentad associated with this disease, namely: clinically-suspected appendicitis, profound neutropenia, recent chemotherapy, acute leukemia (either AML or ALL), and poor clinical course if managed solely with antibiotics or anti-candida medication. Aspergillus fumigatus is the most common and frequent species that causes infection in humans followed by A. Flavus and A. terreus. As mentioned, ingested contaminated food or mucosal ulcers from chemotherapy give rise to Aspergillus infestation. Aspergillus invade the appendiceal mucosal wall due to immunosuppression from neutropenia and acute leukemia. The child develops persistent right quadrant pain, fever and systemic GI signs unresponsive to antibacterial or anti-candida therapy. Ultrasound or with greater accuracy a CT-Scan will show appendicitis. The appendix should be removed promptly, and the specimen microscopically examined for Aspergillus with special stains. Anti-Aspergillus therapy with voriconazole or amphotericin should be instituted after removal of the appendix since the child will continue to be colonized with the organism. Surgery is crucial for removing the inflamed appendix, clearing the infection and producing tissue sampling. Positive galactomannan levels guide the decision to change the antifungal therapy regime.

References:
Eosinophilic Cellulitis

Eosinophilic cellulitis, also known as Well's syndrome, is a rare inflammatory skin disorder of unknown etiology. It is categorized as a relapsing eosinophilic dermatitis with variable clinical appearance. Cutaneous lesions are variable in appearance and may be similar to cellulitis, urticaria, insect bites, morphea or contact dermatitis. During the acute phase the child develops tender urticarial plaques, vesicles, bullae or nodules in the skin. This is usually accompanied with peripheral blood eosinophilia. Papulovesicular blistering and nodular lesions which are often painful or pruritic can also be seen. Later the skin lesions become indurated with morphea-like appearance resolving without significant scarring. Histopathologic findings of the lesion include eosinophilic granulomatous infiltration of the dermis and formation of flame figures without signs of vasculitis. Hypersensitivity due to different triggers such as insect bites, stings, drugs, infections, atopic dermatitis and contact dermatitis have been proposed. Causative medications include antibiotics, anticholinergic agents, anesthetics, non-steroidal anti-inflammatory agents, thyroid medications, chemotherapeutic agents, thiomersal containing vaccination, anti-tumor necrosis factor agents and thiazide diuretics. Children with sensitivity to thiomersal found in certain vaccines (such as influenza) can develop eosinophilic cellulitis. Diagnosis of the condition might need biopsy for histopathological identification. Other times the lesions coalesce and cause a subcutaneous abscess which need to be drained. Well's syndrome is initially managed successfully with topical or systemic corticosteroids and calcineurin inhibitors but it often relapses upon tapering. Systemic glucocorticosteroid are the most common and effective treatment modality reported.

References:
Appendicitis in the Neutropenic Child

With advancement in chemotherapy many children now survived to leukemia, lymphoma and other malignancies. The use of chemotherapeutic agents is associated with bone marrow toxicity and development of neutropenia along with infectious complications in the gastrointestinal tract. Right quadrant pain in the neutropenic child can be the direct consequence of acute appendicitis, neutropenic typhlitis, pseudomembranous colitis or obstructive ileus. Both appendicitis and neutropenic colitis are the most common surgical complications in children with leukemia. The use of steroids in these patients can masquerade acute appendicitis since the blunt the classic signs of appendicitis such as abdominal tenderness, rebound tenderness, involuntary guarding and abdominal wall rigidity. This causes confusion in the clinician trying to determine of the child has an acute abdominal condition or his symptoms are the result of side effects of chemotherapy. The incidence of appendicitis in the population of chemotherapy-induced neutropenia children is the same to the general pediatric population (2%-4%). The clinical presentation and findings on physical exam of neutropenic children diagnosed with appendicitis are often vague and atypical. Since the most important decision is to differentiate acute appendicitis from neutropenic colitis children with right lower abdominal pain, fever and CT Scan findings of cecal wall thickness carries a presumptive diagnosis of typhlitis. In the absence of these findings acute appendicitis should be suspected and managed accordingly. Limited operative morbidity in neutropenic children with appendicitis leads to favor surgery over medical antibiotic therapy with delayed intervention. Medical management of appendicitis in the child with neutropenia increase hospital stay and use of pain medication. The majority of the children managed nonoperatively had recurrence of their RLQ pain on subsequent rounds of chemotherapy. Early appendectomy once the diagnosis is confirmed is safe and eliminates the course of the disease in addition to avoiding the concern for uncontrolled intraperitoneal contamination in the immunosuppressed child.

References:

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