in a better balance between bone formation and resorption leading to an increase in bone mass. The lines of increased bone density are likely to be the result of an increase in both the number of trabeculae and the amount of residual calcified cartilage within the secondary spongiosa(2). This radiographic appearance is striking and will probably be seen more commonly with expanding indications for Biphosphonate therapy in the future, Other conditions where multiple dense transverse bands extending across the metaphysis of long bones are seen include chronic lead poisoning and in chronic disease or stress related to malnutrition in which case they are referred to as growth arrest lines.

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Nephrotic Syndrome in a Girl with Filariasis

Filariasis causing nephrotic syndrome is rare and reported only in adults but not in pediatric age group(1). We report here an interesting case of a girl with nephrotic syndrome secondary to filariasis. A ten-year-old girl was admitted with generalized body swelling and decreased urine output for one month. There was no history of hematuria or dysuria. On examination she had anasarca and her blood pressure was normal. She had a distended abdomen due to ascites and grossly edematous lower limbs. Investigations revealed the following: Hb 12 g%, TC 5000/cu mm, DC - N 62% E 12% L 26%. Serum analysis showed Urea 32 mg/dL, total protein 3.9 g/dL, albumin 1.1 mg/dL, and cholesterol 498 mg/dL. Urine examination revealed massive proteinuria, normal microscopy and absence of chyluria. In view of eosinophilia and longstanding pedal edema, filariasis was considered. Peripheral smear was positive for microfilaria and filarial serology was also strongly positive by indirect hemagglutination. She was treated with diethylcarbamazine for 3 weeks and she went into remission after 10 days.

In this child with filariasis, all the criteria for nephrotic syndrome namely edema, proteinuria, hypoalbuminemia and hyperlipidemia were present. A remission induced by antifilarial therapy also supports the filarial etiology for nephrotic syndrome. Nephritis due to microfilaria has been postulated as a possible mechanism for the development of nephrotic syndrome(2). Chyluria due to Bancroftian filariasis leading to hypo-albuminemia is documented in literature(3). The absence of

chyluria in this girl rules out hypoalbuminemia due to filariasis *per se*. However, microfilaria could not be documented by renal biopsy. Hence, filariasis is the probable cause for nephrotic syndrome in this girl.

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Pseudomonas Septicemia in Selective IgM Deficiency

A 7½-month-old male baby presented with cough 10 days, fever 7 days, altered sensorium 3 days, discharge both ears 3 days, and reddish lesions on body 2 days. In the past he had 3 episodes of chest infections. He was the only child born out of non-consanguinous marriage between apparently healthy parents. There was no history of intake of immuno-suppressive drugs. He was found to be very sick with marked tachycardia, respiratory distress, bilateral seropurulent ear discharge, multiple erythematous indurated patches with necrotic center, generalized lymph-adenopathy, hepatomegaly and crepita-tions in the chest. He was deeply comatose, had brisk deep tendon reflexes, extensor planters and meningeal signs. There was polymorpho-nuclear leukocytosis and thrombocytopenia. CSF was suggestive of partially treated pyogenic meningitis, however, CSF culture was sterile. Gram stain of CSF and skin lesions revealed no bacteria. Culture of fluid from skin lesions, blood culture and ear discharge grew Pseudomonas aeruginosa. Serum IgG level was 930 mg/dL, IgA was 102 mg/dL, and IgM was 12 mg/dL. Serum IgM level of father was. 19.5 mg/dL. The patient was administered appropriate antibiotics, platelet transfusion, fresh frozen plasma, IV immunoglobulins along with other supportive measures. His condition progressively deteriorated and he succumbed to disseminated intravascular coagulation and multiorgan dysfunction.

Selective IgM deficiency severe enough to cause symptoms is rare as a primary disorder though it is a common consequence of immunosuppression(1). Patients have IgM concentration <20 mg/dL with normal levels of other immunoglobulins(2). Inheritance is multifactorial, may be autosomal recessive but is presumed to be partly influenced by X-chromosomal products. Among healthy