Lichen Planus and Nephrotic Syndrome-Coincidence or Causation?

We describe an 8 year old boy who presented with lichen planus (LP) and minimal change nephrotic syndrome (MCNS), and discuss the possible pathogenetic links between the two disorders. A 7 year old boy presented with anasarca for 7 days in association with skin lesions over both the lower limbs. The skin lesions were bilaterally symmetrical violaceous polygonal pruritic papules present over both lower limbs, diagnostic of classical LP; and appeared simultaneously along with periorbital edema progressing to anasarca. The oral, genital mucosa and nails were unaffected. There was no causal relationship of either the skin lesions or anasarca with any drug usage or immunization. There was no jaundice, hepatosplenomegaly, lymphadenopathy or joint involvement. Urinalysis showed proteinuria (urine spot protein: creatinine ratio 3.4). Serum albumin and cholesterol were 1.8 g/dL and 340 mg/dL respectively. Serum creatinine was 0.5 mg/dL. Hepatitis serology profile, chest x-ray and complete blood counts were normal. Anti-nuclear antibody (ANA) were negative and C3 levels were normal. The renal biopsy showed minimal change disease. He was treated with prednisolone as per standard guidelines [1], and went into remission. The skin lesions were treated with 0.05% betamethasone dipropionate cream for 2 weeks and healed with patchy hyperpigmentation. Seven months later, he had a relapse of nephrotic syndrome coincident with flare of the lichenoid lesions over the same sites. He was again treated with systemic and topical steroids. Proteinuria is currently settled. Itching has subsided, however residual postinflammatory hyperpigmentation is present.

LP is a chronic inflammatory dermatological condition usually affecting adults, but rare in children [2]. The diagnosis is essentially clinical [2]. Immunological mechanisms mediate the pathogenesis of LP, as evidenced by dermal infiltrate of T lymphocytes and association with diseases of altered immunity such as vitiligo [3]. An association with hepatitis C is mentioned [2].The immune system including T cells have an important role in the pathogenesis of steroid sensitive MCNS too [4], although the mechanisms are not fully understood [4]. On literature search, only a single similar case of MCNS with LP in a 30 year old Italian woman was found [5].

Considering the temporal association of the two diseases and the flare of the LP coincident with the nephrotic relapse, we believe this may not be a mere coincidence. To our knowledge, this is the first pediatric case of LP in a patient with MCNS, and may reflect common immunological abnormalities, based on altered cell mediated immunity.

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Vivax Malaria : A Pandora's Box

As compared to falciparum malaria, the less dramatic vivax malaria was often considered benign with fewer variations. We report a case of vivax malaria in a two month old with autoimmune hemolytic anemia.

A 2 month old girl was brought to our for fever with chills and rigors for 8 days and vomiting since 2 days. Systemic examination revealed a tachycardia of 142/min, severe pallor and a hepatosplenomegaly with a liver size of 4 cm below the costal margin [span of 8 cm], a soft consistency, nontender with a soft spleen that was 6 cm below the costal margin. With a concern that we may be dealing with falciparum malaria, the child was empirically started on intravenous artesunate. She had a hemoglobin of 8 g/dL 6 days before admission which had

dropped to 5.2 g/dL 1 day before being hospitalized. Her hemoglobin on admission at our hospital was 4.1 g/dL with the peripheral smear revealing *plasmodium vivax* [few trophozoites, schizonts and gametocytes]. Initial reticulocyte count was 9.2% and LDH was 1405. In view of the rapid drop in hemoglobin, we asked for a direct Coombs test which was strongly positive. The first transfusion of packed cells raised her hemoglobin to 6.5 g/dL in a few hours while the second transfusion raised it to 8.4 g/dL. We omitted artesunate as soon as the smear report was available and initiated the child on oral chloroquine followed by primaquine once the G6PD report was normal. Subsequently, her hemoglobin stabilized at 8.8 g/dL by the 8th day. DCT became negative and the reticulocyte count also dropped [2.6%]. While AIHA is occasionally seen with falciparum malaria [1,2], there are only occasional reports of vivax induced AIHA in the world literature [3]. Thrombocytopenia which was once considered an indicator of falciparum malaria is now routinely seen with vivax malaria, albeit with a much more benign course. When faced with dropping hemoglobin in a patient with vivax malaria, the first thought that comes to mind is parasite induced destruction of red blood cells causing rapid drop in hemoglobin or drug induced hemolysis in a G6PD deficient individual. However, we must include rarer causes of drop in hemoglobin such as infection induced hemophagocytic syndrome [4] and a AIHA caused by the malarial parasite.

Such an AIHA can also be suspected when it is almost impossible to cross match blood [did not occur in our case]. Malaria induced AIHA is usually self-limiting and abates once the primary disease is treated.

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