Pseudochromhidrosis: A rare case of yellowish discoloration of sweat in a patient with dermatitis herpetiformis and celiac disease

Introduction

Chromhidrosis is the production of colored sweat by eccrine or apocrine sweat glands. Pseudochromhidrosis is the production of colourless sweat, which then becomes coloured when it reaches the skin and combines with agents like chromogenic bacterial products (such as those produced by species of corynebacteria) , certain chemicals, paints and dyes. Only a few cases have been reported previously.[1,2] Here, we report the case of a 25-year old male with pseudochromhidrosis who was earlier diagnosed with celiac disease and dermatitis herpetiformis.

Case report

A 25-year old male was referred to the Gastroenterology OPD with presenting complaint of yellowish discoloration of sweat for the last one month. He had consulted many physicians before reaching us. He was on a gluten-free diet. There was no icterus, peripheral oedema or organomegaly. Six months ago, he was diagnosed as a case of celiac disease with dermatitis herpetiformis and was under treatment elsewhere for the same **(Figure 1)**.

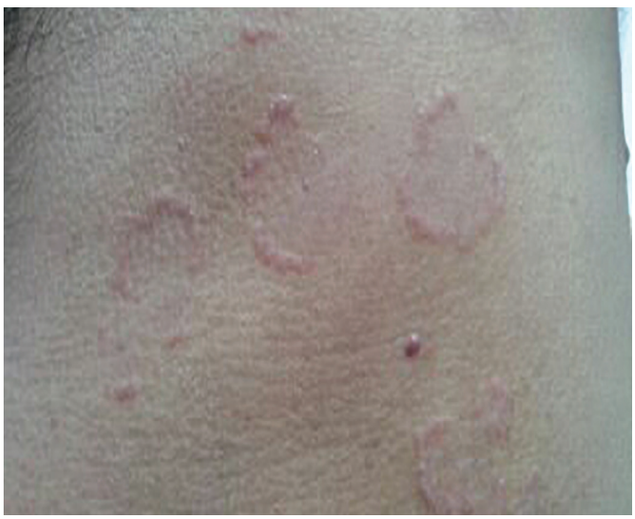

Figure 1: Skin lesions of Dematitis herpetiformis

On examination, his underclothes had deep yellow stains in the axilla, chest, back and groin **(Figure 2)**.His workup including blood counts, liver function tests, urine routine microscopy, urine homogentisic acid, ultrasound scan of the abdomen and skin biopsy were unremarkable. He was diagnosed as a case of pseudochromhidrosis and treated with oral ciprofloxacin and erythromycin for 4 weeks to which the patient responded well.

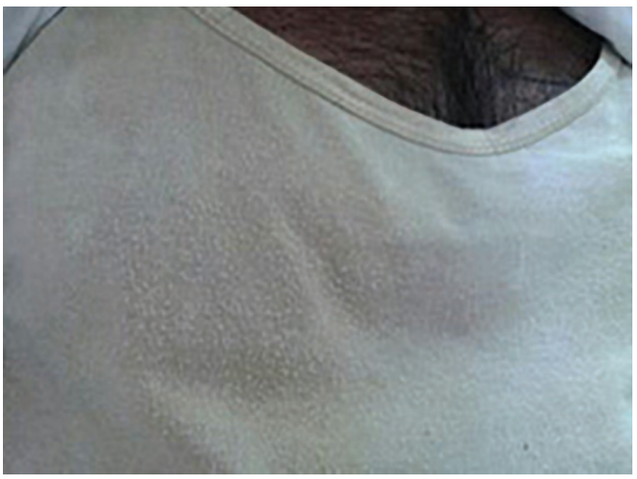

Figure 2: Yellowish pigmentation of undergarment

Discussion

Both chromhidrosis and pseudochromhidrosis are rare. The diagnosis of chromhidrosis is based on biopsy and autofluorescence of skin specimens and stained clothing, while the diagnosis of pseudochromhidrosis is based primarily on history and a good response to treatment with antibiotics[1]. Oxidized lipofuscins, which autofluoresce at 360 nm, have been detected by examining stained clothing with ultraviolet light and by autofluorescence microscopy of skin biopsies[2].

Treatment options for chromhidrosis is limited. Capsaicin depletes the neurons of substance P and has been found to be beneficial in the treatment of these patients. The other options are topical clindamycin and topical aluminum chloride. The definitive treatment is surgical excision of the apocrine sweat glands[3]. In contrast to chromhidrosis, pseudochromhidrosis is easily treatable[4]. Pseudochromhidrosis due to bacterial causes can be treated with systemic and topical antibiotics. Pseudochromhidrosis due to exogenous dyes, paints or other chemicals can be remedied by avoiding these compounds.

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Hepatocellular carcinoma presenting as liver abcess

Introduction

Hepatocellular carcinoma (HCC) is the commonest primary malignant tumor of the liver.HCC presenting as a pyogenic liver abscess is reported but is uncommon and rarely addressed in literature[1]. It is challenging to diagnose HCC presenting as a pyogenic abscess.[2]

Case Report

A 38 year old male patient presented with the complaint of continuous, dull aching pain in the right hypochondrium for 2 months. The pain was of moderate intensity,non radiating and associated with high grade fever with chills. There was history of loss of appetite and weight loss of approximately 3 kilograms over two months.On examination he was febrile and hemodynamically stable. He had tender hepatomegaly 6 cm below the costal margin in the mid-clavicular line. The liver was firm in consistency, had rounded margins and a smooth surface.He had pallor and the rest of the general and systemic examinations were normal. Lab investigations revealed a TLC of 24,600 /cu.mm with 83 percent polymorphs,hemoglobin was 9.7 g/dl and platelets were 3.99lacs/cu.mm.His liver function tests were normal except for a raised alkaline phosphatase (ALP), with total bilirubin of 1.2 mg/dl, Direct:0.8 mg/dl,Indirect:0.4 mg/dl,aspartate aminotransferase:35 IU/L,alanine aminostransferase:12 IU/L, ALP:260 IU/L (Normal upper limit:110), total protein:7.6 g/dl,albumin: 4.6 g/dl,globulin:3.0 g/dl, INR: 1.06.Malaria antigen, Widal test,urine R/M,blood culture and urine cultures were all negative.He was positive for HBsAg and negative for HBeAg with HBV DNA titer of 1180 IU/mL.HIV and antiHCV antibodies were negative. Ultrasound abdomen revealed hepatomegaly (18.1 cm),spleen 10.1 cm.The gallbladder,portal vein and common bile duct were normal while minimal free fluid was found in the abdomen.A 9.9 x 6.8 x 9.4 cm sized mixed echogenic, predominantly hypoechoic collection was noted in seg V and VI of the liver, abutting the right kidney.Impression of a liver abscess was given. Intravenous antibiotics were started and ultrasound guided aspiration of the lesion was done. The cellular smears revealed innumerable neutrophils admixed with several foamy macrophages,bare nuclei and degenerated cells over a necrotic background. No amoebea, scolices or hooklets, epitheloid cell clusters or multinucleated giant cells were seen.No atypical or malignant cells were detected. Diagnosis of pyogenic liver abscess was suggested.Aspirate culture showed no growth,Indirect hemagglitination test for anti amoebaantibodies was negative and serum alfa fetoprotein was within normal limits (4.11 IU/ml).

An ultrasound guided liver biopsy findings showed fibrin plugs enmeshing few cords and ribbons of partly degenerated cuboidal cells. The cells possess granular acidophilic cytoplasm and rather monotonous round nuclei with minimal anisonucleosis. At places, "ghost outlines "of similar cells were seen. The polyhedral cells could represent a necrotic neoplasm however,the accompanying smears were entirely inflammatory. To further characterize the lesion dynamic C.T. abdomen **(Figure 1)** was done. It showed 12 x 7 x 7.5 cm