

CHROMHIDROSIS AND PSEUDOCROMHIDROSIS

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Chromhidrosis is the production of colored sweat by eccrine or apocrine sweat glands. Pseudochromhidrosis is the production of colorless sweat, which becomes colored when it reaches the skin and combines with other agents such as chromogenic bacterial products (*Corynebacterium* species), other chemicals, paints, or dyes.¹

Eccrine chromhidrosis is very rare. Water-soluble pigments are excreted by eccrine sweat glands. Apocrine chromhidrosis is seen in the axilla, facial and areolar regions and is brown, black, blue or green in color. Oxidized lipofuscins, which autofluoresce at 360 nm, have been detected by examining stained clothing with ultraviolet light or by autofluorescence microscopy of skin biopsies.¹

There have been few reports of Chromhidrosis in the literature. Cilliers and de Beer reported a 26-year-old woman who presented with a 5-month history of pink staining of her white nursing uniform and undergarments. The stains could be removed by prolonged soaking in water. On more detailed questioning the patient disclosed a 6-month history of eating a tomato-flavored prepackaged food.

Distilled water extraction was performed on samples of red-stained clothing. Ether extraction was performed on a sebum extraction. Sweat was harvested using pilocarpine hydrochloride. Additionally, a sample of early morning urine was analyzed. These samples were all analyzed using a spectrophotometer. The analysis of the extraction from the clothing matched the analysis of the urine sample. Both matched analysis of the tomato powder and paprika in the snacks. The authors determined that this was a case of true chromhidrosis by a pink lipid- and water-soluble agent in the tomato-flavored

snack, which was being excreted from the skin renal system.¹

Mali-Gerrits et al report a 35-year-old woman who presented with 10 months of black axillary sweat. The black sweat could be elicited by manual expression of the axillary skin, and fluoresced yellow under a Wood's light. The urine was negative for homogentisic acid. H & E stain of an axillary skin biopsy showed apocrine glands with predominantly apical brown granules, which were stained positively with PAS. The granules also fluoresced in ultraviolet light. The patient's sweat was maximally excited at 326 nm, and maximal emission at 514 nm. These features indicated that the pigment in the patient's sweat was not lipofuscin, which has maximal excitation between 360-395 nm, and maximal emission between 430-460 nm. This patient had axillary chromhidrosis. Brown axillary sweating can be caused by ochronosis, but this was ruled out by the negative homogentisic acid in her urine.²

Saff et al report a 15-year-old girl who presented with 3 years of brown-black chromhidrosis affecting the areolae. No autofluorescence was present on examination of the sweat with a Wood's light. Dark black and brown sweat is usually non-fluorescent, and first presents during adrenarchy, and decreases with increasing age. This patient experienced mild-moderate benefit from Capsaicin treatment.³

Diagnosis is simplified by autofluorescence of clothing affected by chromhidrosis.⁴ Cox et al present a 33-year-old woman with 6 months of green stained clothing around the axillae and breasts. Her clothing was stained lime-green, and she was diagnosed with chromhidrosis on skin biopsy, which

showed lipofuscin granules in the apocrine glands. Microscopic autofluorescence was demonstrated in an unstained histologic section. Stained clothing fibers were examined and demonstrated the same yellow-green color as the apocrine biopsy. A less intense fluorescence was observed on examination of stained clothing with a Wood's light. Autofluorescence is yellow in lipofuscin granules. The intensity of this autofluorescence increases from yellow through green to blue apocrine sweat. Autofluorescence from black sweat is weak. Lipofuscins are more highly oxidized in darker sweat.⁴

Therapy for this condition is limited. Shelley and Hurley suggested manual emptying of the glands, but this is only a temporary solution. Capsaicin depletes the neuron of Substance P and has been found to be a beneficial treatment for these patients.³ Marks reports a 30-year-old woman with an 8-year history of blue-black sweating in the malar region. She was unsuccessfully treated with topical clindamycin and topical aluminum chloride. She was then given a trial of capsaicin, which resulted in complete suppression of the apocrine chromhidrosis.⁵ Definitive treatment is surgical excision of apocrine sweat glands.²

Eccrine pseudochromhidrosis is sweat that becomes colored via surface compounds or molecules mixed with sweat. Classically this has been reported as blue sweat in copper workers.¹ Additionally, there were reports of flight attendants who experienced red discoloration of their sweat after wearing uniforms, which had been labeled with a red dye.⁹

Singal and Thami report a case of a 10-year-old girl who presented with a 15 day history of red discoloration of her neck, which was easily removable with soap and water, but would reappear in 1-2 hours. When pilocarpine was injected red sweat appeared within 1 minute. Urinalysis was normal, wood's light; gram stain and cultures of skin scrapings from affected areas were all negative. Pseudochromhidrosis secondary to bacterial infection was considered and she was treated with erythromycin 250 mg TID along with topical erythromycin gel. Complete resolution was obtained at 7 days, and she was followed for 7 years without recurrence.⁷

Thami et al report a similar case in a 9-year-old girl who presented with a 1-week history of red discoloration of her cheeks. Pilocarpine was injected into the right cheek and red sweat droplets appeared and were allowed to dry, resulting in a reddish powder. Scrapings were negative under Wood's light examination; Gram's stain and cultures were negative. She was treated for bacterial infection with erythromycin 250 mg TID along with erythromycin topical gel applied BID. Resolution was obtained at 1 week, and there was no recurrence in 3 months of follow-up.⁸

Yoshida et al report 2 cases of men who presented with brown sweating from the palms. The discoloration could not be removed with ethanol. It was later determined that these patients had inadvertently rubbed their palms with self-tanning tissues, having mistaken them for regular wet tissues. These cases illustrate the need for a detailed history of patients who present with discolored sweat, as there may be some temporal correlation with the use of chemicals or dyes that could result in discoloration.¹⁰

In contrast to chromhidrosis, pseudochromhidrosis is easily treatable. 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.

Both chromhidrosis and pseudochromhidrosis are rare. Diagnosis of chromhidrosis is based on biopsy and autofluorescence of both skin specimens and stained clothing, while diagnosis of pseudochromhidrosis is based primarily on history, and successful treatment with antibiotics. Chromhidrosis patients deserve a trial of capsaicin therapy. Definitive treatment is surgical excision.

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CHROMHIDROSIS AND PSEUDOCROMHIDROSIS – MUSEL

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