Journal of Pigmentary Disorders
ISSN: 2376-0427

Khanna and Khandpur, Pigmentary Disorders 2015, 2:10
DOI: 10.4172/2376-0427.1000214

Review Open Access

Pigmented Contact Dermatitis

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Rec date: August 28, 2015; **Acc date:** September 18, 2015; **Pub date:** September 30, 2015

Abstract

Pigmented Contact dermatitis is a non-eczematous variant of contact dermatitis clinically characterised by reddish-brown to slate grey pigmentation in a reticulate pattern, usually without any active or preceding clinical dermatitis. A detailed history is essential to establish the temporal correlation of exposure to a possible sensitizer, which further helps in differentiating PCD from its close mimickers such as melasma, lichen planus pigmentosus erythema dychromicum perstans, etc. Clinical variants of PCD include Riehl's melanosis, pigmented cosmetic contact dermatitis, pigmented contact cheilitis and purpuric dermatitis. Histopathology shows pigment incontinence. Patch testing should be performed to identify the causative allergen and besides a papule or vesicle, a brown pigment may develop at the patch test site. Patients should be counselled regarding complete avoidance of the causative allergen and also the resistance of this condition to various treatment modalities.

Keywords: Pigmented contact dermatitis; Hyperpigmentation; Pigmentation; Dermatitis; Melasma

Introduction

Pigmented contact dermatitis (PCD) is a non-eczematous variant of contact dermatitis which is clinically characterized by hyperpigmentation with little or no signs of dermatitis [1]. Osmundsen, a Danish dermatologist had first used the term "pigmented contact dermatitis" to describe an epidemic of contact dermatitis in Copenhagen which had occurred due to the optical whitener (Tinopal CH 3566) used in washing powders [2]. In about 8 months, 120 patients were seen with contact dermatitis due to the washing powder; furthermore there were 7 patients who showed a pronounced and bizarre pigmentation. In four cases, the pigmentation followed a contact dermatitis, but the other 3 patients had not observed any skin change or itching prior to or during the development of pigmentation. In almost all cases, the hyperpigmentation was localised mostly to covered areas, such as the chest, back, waist, arms, neck, and thighs. One of the patient wanted to conceal the pigmentation by wearing long sleeves and a high-neck sweater, which she had been washing with the same washing powder and as a result the hyperpigmentation started to extend from the neck and axillae to all over the neck, chest, and arms. The hyperpigmentation in the reported cases had varying colours ranging from brown, slate-colored, grayish-brown, reddish- brown, bluish-brown, etc. and in some cases showed a reticulate pattern. The histopathology demonstrated pigment incontinence and patch tests with the standard series of that time were negative [2]. However, patch tests with 1% CH 3566 in petrolatum finally explained the pigmentary disorder, as they showed strong positive reactions in the patients and negative results in the controls. Historically the next major series of PCD were seen in 53 workers handling azo dyes in a textile factory [3]. Among them, 12 workers developed an asymptomatic, spotted hyperpigmentation and 18 suffered from hyperpigmentation of lesser extent with morphology varying from bizarre dark pigmentation to a streaky milder pigmentation of the neck, arms, face, and in exceptional cases, covered areas. This new occupational skin disorder appeared almost 4 months after the introduction of a new dyeing process of azo-coupling on textiles.

Etiology and Pathogenesis

There are several contact sensitizers that produce secondary hyperpigmentation, the common ones being Tinopal Ch 3566, Naphthol AS, benzyl salicylate, hydroxycitronellal, D&C Red 31, Phenyl-azo-2-naphthol, D&C yellow 11&10, Carbanilides, Ylang-ylang oil, Cananga oil, Jasmin absolute, synthetic sandalwood, sandalwood oil (Table 1) [1,4]. In the Indian population, red kumkum is the most commonly implicated allergen causing PCD, although there is clear paucity of data in Indian context [1]. Commercially available red kumkum contains azo dyes, coal tar dyes, toluidine red, erythrosine, fragrances, ground nut oil, tragacanth gum, turmeric powder, paraben and cananga oil [1]. Since most cases of PCD have been found to occur in dark skinned patients there appears to be a role of pigment-genetic interaction [1]. PCD is commonly acquired due to direct contact with the allergens, although few cases have been described in the literature where it occurred after air-borne spread of the allergen due to cigarette smoke, musk ambrette or Plathymenia foliosa dust [1,5,6]. Some experimental studies have demonstrated that cutaneous inflammation increases the number and size of melanocytes and enhances their enzymatic activity, thus explaining the association of pigmentation with contact dermatitis [7]. Additionally, Nagao et al. postulated that the allergen responsible for PCD may have a special affinity for melanin, inciting an inflammatory reaction first around the melanocytes and then incorporating melanin granules around the cells [8]. Osmundsen mentioned that PCD may reflect some idiosyncrasy of the patients and/or the mode of exposure to the allergen and/or some specific peculiarity of the allergen itself, the exact patho-mechanism remaining uncertain [2].

Clinical features

In PCD, reddish-brown to slate grey pigmentation occurs in a reticulate pattern, usually without any active or preceding clinical dermatitis or pruritus thus making the clinical diagnosis difficult in many cases [2]. The skin colour and nature of the allergen can modify the clinical picture. A detailed history is essential to establish the temporal correlation of exposure to a possible sensitizer [9]. PCD has been seen to occur after 2 months to 2 years of exposure to the allergen [2,3]. Face is the commonest site affected, however lips, axillary borders and thighs may be affected due to lipstick colour, shirt dye and trouser dyes respectively [1].

Textiles	Naphthol, Azo dyes, optical whiteners
Fragrances	Musk ambrette, Cananga oil, benzyl salicylate, sandalwood oil, lavender oil, cinnamic alcohol, Ylang-ylang oil, Jasmin absolute, synthetic sandalwood, sandalwood oil
Cosmetics	Hair dye, lipstick, kumkum, preservatives
Others	Para tertiary butyl phenol formaldehyde (PTBF), wood dust (Plathymenia foliosa), nickel sulphate, chromium hydroxide, cigarette smoke

Table 1: Common allergens implicated in pigmented contact dermatitis [1,5,6].

Clinical variants of PCD

Riehl's melanosis

It was first described by Riehl in 1917 during World War I, when he identified approximately 17 patients who had striking dark-brown to grayish-brown facial pigmentation, most pronounced on the lateral aspects of face and neck particularly concentrated on the forehead, ears, temple, and the zygomatic regions [10]. In few cases, pigmentation, although less pronounced and consisting of small follicular pigmented macules, was noticed on the thorax [10]. In addition, erythematous macules and papules were also identified. Riehl could not identify the exact cause of the eruption, however he speculated that it occurred due to some nutritional alteration that he attributed to wartime conditions. With the end of the war, no further cases were reported, thus supporting his hypothesis. Subsequently in World War II, 165 people in France reported a similar eruption, again associated with scarce food supplies and disappearing with the end of the war. But unlike Word War I, most cases were reported in women [11]. Later, Hoffmann and Habermann described a condition referred to as 'melanodermatitis toxica' that was hypothesized to be a form of contact dermatitis associated with the use of certain oils and hydrocarbons [11]. They emphasized the clinical similarities between Riehl's melanosis and melanodermatitis toxica, but Riehl could not accept that the melanosis he had described was due to a local chemical irritant and considered the two conditions to be separate entities [11]. Riehl's melanosis was subsequently described in the nutritionally deprived Bantu people of South Africa; however, no further reports in the literature have linked it to nutritional deficiencies [10]. Histopathology shows liquefactive degeneration of the basal cells accompanied by melanophages in the dermis. A moderate cellular infiltrate of lymphoid cells and histiocytes is also usually present in the papillary dermis. However, other than pigment incontinence, other features may be very suble [11].

Pigmented cosmetic contact dermatitis (PCCD)

After the Word Wars, Minami and Noma described a pigmented dermatitis in Asian women unrelated to the war and named the condition 'melanosis faciei feminae', which subsequently was attributed to the use of aniline dyes in cosmetics [12]. This entity has been differentiated from PCD by predominant involvement of the face, cosmetics being the causative agents and PCCD being more symptomatic, with many cases reporting a slight dermatitis preceding or accompanying the hyperpigmentation [11]. Clinically it manifests as diffuse or reticulate, black or dark brown hyperpigmentation of the face with ill-defined margins [11]. In some cases, the dark brown or black hyperpigmentation occurs at extrafacial sites involving the neck, chest, and back and in few exceptional cases, it may extend to the whole body [11]. Extensive PCCD has been reported with the allergen cinnamic alcohol which can sensitize the patients first to cosmetics and then provoke allergic reactions to soaps, domestic fabric softeners and food that contain cinnamic derivatives [13]. Histopathology of PCCD is similar to PCD. Epidermis may sometimes be atrophic, presumably due to the effect of frequently applied corticosteroid ointments for the treatment of itchy dermatitis of the face which precedes or accompanies the pigmented cosmetic dermatitis [14].

Pigmented contact cheilitis

Hemmer et al. reported a Philippino patient with hyperpigmentation of her lips from the use of lipstick [15]. This patient's condition improved upon avoidance, but exhaustive patch testing failed to elucidate the exact allergen [16]. Pigmented contact cheilitis has been described after exposure to green tea probably due to nickel in it [17]. Mehta et al. reported a case of pigmented contact cheilitis due to Paraphenylenediamine PPD following hair dye application to the moustaches [18].

Purpuric Dermatitis

It can also be a manifestation of PCD. This variant was observed in many British soldiers during World War II, especially in those who sweated profusely or experienced friction with their khaki shirts, woolen socks and elastics in undergarments [19,20]. Allergens implicated include rubber ingredients such as N-phenyl-N´-isopropyl-p-phenylenediamine (IPPD), N-phenyl-β- naphthylamine (PNA), 2-mercaptobenzothiazole (MBT), dibenzothiazole disulfide (DBD), textile finishes and dyes (blue 85) [20,21].

Differential Diagnosis

Various inflammatory processes may disturb the dermo – epidermal junction and melanin may pass into the upper dermis to produce persistent pigmentation (post-inflammatory hyperpigmentation) [2]. Hyperpigmentation may occur in the course of irritant and allergic

contact dermatitis, though it is not a common feature of these disorders [2]. It may however commonly follow many acute and chronic inflammatory processes in the skin such as lichen planus, lichenoid drug eruption, fixed drug eruption etc [2]. Other differential diagnoses of PCD includes melasma, lichen planus pigmentosus (LPP), addison's disease, friction melanosis, amyloidosis cutis, drug eruption or atopic dermatitis with pigmentation [11]. Melasma is characterized by symmetrical brown to grey brown macules, which may be blotchy, irregular, arcuate, or polycyclic distributed predominantly on the sun exposed areas such as the face (predilection for malar area, periorbital and perioral area, nose), 'V' area of the neck and forearms [22]. LPP is also a close clinical differential of PCD which is characterized by generally asymptomatic diffuse dark-brown to slate-gray macules present mostly over exposed areas and flexures. Less commonly the lesions may be itchy reticular, blotchy, or perifollicular. The lesions lack the erythematous border of EDP (a feature usually appreciated in lighter skin types) [22]. Friction melanosis manifests as asymptomatic dark brown to black hyperpigmentation over bony prominences such as clavicles, scapulae, knees and elbows. The histopathology of friction melanosis shows pigment incontinence similar to PCD [23]. Tienthavorn et al evaluated the histopathology and patch test results of 43 Thai patients of skin types IV- VI with clinical differential diagnoses of EDP, LPP and PCD [24]. The histopathology showed overlapping features, with melanin incontinence being observed in almost all cases. The additional histological feature of lichenoid inflammatory reaction pattern was more pronounced in EDP and LPP, while in PCD, liquefactive degeneration of the basal layer was seen in addition. In our experience, it is difficult to differentiate between these three entities based on histopathology. In the Thai study, 80% of PCD cases showed patch test positivity. Interestingly as many as 40% and 36.3% patients with clinical diagnosis of EDP and LPP respectively also showed a positive patch test [24]. Thus, on several occasions histopathology and patch testing alone may not help in differentiating PCD from its close mimickers.

Investigations

Patch test is of immense value in the diagnosis of PCD. Closed patch testing should be carried out with the standard, cosmetic and fragrance series and personal products of the patients. Photo patch test should also be undertaken as part of the evaluation [1]. A study from Israel on the utility of screening patch test in PCD revealed highest yield with the European standard series and the Scandinavian photo series. The cosmetic and fragrance series gave a low yield, with only 2 patients showing a relevant result to at least one allergen [24]. Besides evidence of allergic dermatitis in the form of a papule or vesicle, a brown pigment may develop at the patch test site. Osmundsen in his report of 7 patients of PCD, on patch test revealed an intense, bluish-brown pigmentation at the site of patch test in about 3 weeks in 3 patients; in the fourth patient, a relatively weak pigmentation developed [2]. The provocative usage test (PUT) or repeated open application test (ROAT) may identify a reaction if closed patch test reaction is equivocal [1]. Unlike the usual cases of contact dermatitis where the eczematous reaction resolves with the removal of the allergen, in PCD, pigmentation tends to persist for years even after withdrawal of the implicated allergen [11].

Prevention and Treatment

It is most important to avoid using textiles and washing powders which contain potent contact sensitizers. In many instances the impurities in the colouring dyes are the primary cause of PCD [11]. For instance, in 1985 the very commonly used CI Blue 19 (or Brilliant Blue R) turned out to be allergenic and caused some patch-test-positive cases of PCD [26]. In contrast, Purified CI Blue 19 never produced positive patch test reactions at the same 5% concentration [11]. Minimum safety evaluation tests such as LD50, Ames test and skin irritation test as well as tests to evaluate the sensitising potential of chemicals should be performed on the new textile finishes introduced in the market [11]. Identifying the contact allergen is most important, for which the manufacturers of the implicated product should cooperate with the dermatologists by providing a comprehensive list of all ingredients present in the product in question. Inspired by the early success of allergen controlled cosmetics, in 1985 allergen-free undergarments were introduced called allergen-controlled wearing apparel (ACW) that successfully helped to counteract PCD in Japanese patients [11]. The persistent secondary hyperpigmentation disappears only very slowly when the causative contact allergens are completely eliminated from the patient's environment for a long period, as the hyperpigmentation is considered to be brought about by frequent and repeated contact with a very small amount of contact sensitizer in the textile or washing material [2,11]. Patients should be advised to use allergen- free soaps and allergen-eliminated washing materials for their clothing at the same time [11].

Conclusion

PCD is a distressing clinical condition and poses a diagnostic challenge if temporal correlation regarding allergen exposure is not established. Histopathology shows overlapping features with other dermatoses. In a suspected case, patch and photopatch testing is an important tool to establish the diagnosis and identify the causative allergen. Identification of the causative agent and complete avoidance of subsequent exposure forms the mainstay of treatment. The discoloration is persistent and only lightens over a long time since the pathology involves incontinence of pigment into the dermis.

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