Hair-dye Induced Allergic Contact Dermatitis in a Young Nigerian Apprentice: A Case Report

Olaniyi Emmanuel Onayemi[1], Mufutau Muphy Oripelaye[1*], Fatai Olatunde Olanrewaju[1], Abiodun Grace Alo.[2]

[1]Department of Dermatology and Venereology, Obafemi Awolowo University, Ile-Ife, Osun state.

[2]Departmet of Dermatology and Venereology, Obafemi Awolowo University Teaching Hospitals Complex, Ile-Ife,Osun State

*_Corresponding author;_ Tel: +2347030211101; **E-mail:** mmoripe@yahoo.co.uk

Keywords: allergic, contact dermatitis, hairdressers, *p*-phenylenediamine

INTRODUCTION

Allergic Contact Dermatitis is a type IV hypersensitivity reaction induced by exposure to environmental agent and presenting with a polymorphous pattern of skin inflammation. It occurs at the site of contact to a sensitizing agent with prior exposure to the similar agent. It may assume a public health importance when the associated allergens are due to occupational exposure. This will cause a significant economic loss to those affected with associated reduced productivity. These were typified by the young apprentice presenting with allergic contact dermatitis.

CASE REPORT

A 17 year old female hair stylist apprentice presented at the accident and emergency unit of our hospital with a 4 day history of multiple fluid containing rashes of various sizes on both hands. The rash was insidious in onset and progressively worsened. There was associated itching, pain and progressive swelling of both hands. No history of fever. About 12 hours before onset of symptoms, she had contact with hair dye (containing *p*-phenylenediamine) which she applied to a customer's hair using bare hands. No history of similar symptoms on customer's scalp. Had similar rash on right ear after touching with the dye on the hand. No past exposure to the hair dye. No history of similar rash in the past. No personal or family history of atopy. Past medical history was noted but not contributory.

Examination revealed an anxious young lady in painful distress, had multiple bullae, vesicles, papules and scarification marks with background erythema on both hands. There was black discoloration of all the finger nails. There was also associated swelling of the hands up to the mid-arm with differential warmth.

An assessment of allergic contact dermatitis with secondary bacterial infection was made. Patient was given oral prednisolone 30mg daily which was later changed to topical betnovate upon improvement of lesions. Prophylactic antibiotics were also given. Patch testing was deferred in view of the severely acute nature of the presentation.

DISCUSSION

Contact dermatitis is a common skin conditions and a major cause of morbidity. Its socioeconomic impact as well as its effect on quality of life is great and often difficult to quantify. These impacts are particularly worse when the hands are involved as shown in our patient. Hair styling in Africa includes a number of traditional styles involving plating, braiding and weaving. Enhanced dexterity in using the hand form the basis for performance in this occupation. Occupational contact dermatitis has been widely reported among hairdressers accounting for about 90% of occupational dermatitis with more than half due to allergic contact dermatitis, especially in the developing countries[1].

Skin diseases are often thought to be infectious in developing countries and affected patients are avoided and stigmatized. Occupational dermatitis occurring in hairdressers also impaired their patronage leading to economic loss. This effect is compounded by the attendant disability arising

from hand involvement.[2] The psychosocial well-being is also affected. The social stigma and psychological problems aggravate their low self-esteem and worsen anxiety as well. [2]

The prevalence of Allergic Contact Dermatitis due to specific allergens depends on their sensitizing potential, the frequency and duration of exposure. It differs in various populations and occupations since allergic contact dermatitis results from specific antigenic exposure for each region and/or occupation. Allergic contact dermatitis is one of the manifestations of hand dermatitis in hairdressers, beauty therapists and barbers. Hairdressers and beauty therapists come in contact with a wide range of cosmetic products which contain potential allergens. Lee and Nixon found that para-phenylenediamine a constituents of hair dye, may be responsible for 60% of cases of allergic occupational dermatitis in hairdressers. In some other studies *p*-phenylenediamine was confirmed to have the greatest sensitizing capacity.[7] This was favoured by our patient who had severely acute allergic contact dermatitis presenting as early as eighth hour after exposure and with severe lesions. Hairdressing, being a women-dominated occupation and involving extensive wet work predispose to several skin diseases. This is because they spend a lot of time at the basin, increasing their susceptible to occupational related skin disease, especially on a background of atopy in them and if they work without protective devices.

The skin encounters physical, chemical and biological agents from the environment. The horny cell layer of the epidermis (the outermost layer of the skin) acts to prevent the penetration of these potential allergens.

About 3000 antigens are known to be contact allergens and most of them are small substances with molecular weight less than 500 Dalton. These small molecules are known as haptens. They can penetrate through an intact skin; however certain disease state that impairs barrier function of the skin can lead to an increase risk of sensitization to allergens. The history of atopy was however not elicited in our patient.

Allergic Contact Dermatitis occurs as a result of contact allergen sensitization. The process of sensitization is complex and not completely understood. It involves 2 phases; induction (sensitization) and elicitation (challenge) phases. Both phases are dependent upon, and orchestrated by T-lymphocytes.

The induction phase includes a cascade of events following the first contact with an allergen up to sensitization. Following contact, an allergen penetrates the skin and binds to the major histocompatibility complex proteins which are encoded for by human leukocyte antigen genes located on the epidermal langerhan cells (LC). Certain epidermal cytokines play major role in LC mobilization and they include tumor necrosis factor α (TNF α), interleukin 1β (IL-1β), and IL-18. The allergen carrying langerhan cells travel via the afferent lymphatic to the regional lymph nodes. They are then recognized by specific T cells (memory and naïve T-cells) which become activated. With the help of IL-1 released by the antigen presenting cells (APC) activated T-cells produce several growth factors which cause proliferation of T-cells through an autocrine cascade of events. The expanded progeny of T-cells is subsequently released via the efferent lymphatic into the systemic circulation. The induction phase which takes about 10-14 days and is completed when an individual is sensitized and capable of giving a positive allergic reaction.

The elicitation phase is initiated by a renewed allergen contact. It depends on frequency and altered migratory capacity of the specific T-cells and their low activation threshold. Within the skin, the APCs and specific T-cells can meet, and this leads to local recruitment of pro-inflammatory cytokines and chemokines. The mediators cause a further migration of more specific T-cells, thus further amplifying mediator release. This process leads to a gradual development of eczematous reaction that reaches its peak after 18-48 hours and then declines.

The Clinical presentation of acute Allergic Contact Dermatitis was typified by our patient whose acute eczema presented with pruritus, erythema, papules, vesicles, blisters and weeping. On the contrary, in chronic allergic contact dermatitis, the skin is dry, scaly, fissured or may become lichenified. Chronic allergic contact dermatitis is either due to persistent antigen or repeated exposure to an allergen.

One of the most important steps in the management of Allergic Contact Dermatitis is allergen

identification and avoidance. But even though the patient opted to stop the handling of hair dye, she may inadvertently handle hair that has been previously dyed creating another exposure. These may lead to chronic Allergic Contact Dermatitis, with its attendant complications. Patient therefore was safely counseled on the use of protective wares such as gloves. Pre-vocational training patching testing using Patch tests standard tray will aid allergen identification. This may allow appropriate counseling to be done and actions taken to avoid exposure to implicated allergen. This was not done in the case of our patient who was not even aware of the existence of such service before taking up her vocational training.

Although the patient had allergic contact dermatitis due to *p*-phenylenediamine contained in hair dye, workplace visit may be necessary in instances where the source of the allergens is not obvious. This will help to identify the source of allergen and determine appropriate methods of avoidance. It may also be necessary to contact the manufacturers of products to determine if the allergen is present and to identify suitable substitutes.

CONCLUSIONS AND RECOMMENDATION

Allergic Contact Dermatitis is a common occupational skin disease in hairdressers, and is associated with significant economic loss and impairment in the quality of life of patients. Prevocational patch testing and appropriate counseling will prevent these complications, improve well-being of patient and promote the overall economic growth of the nation.

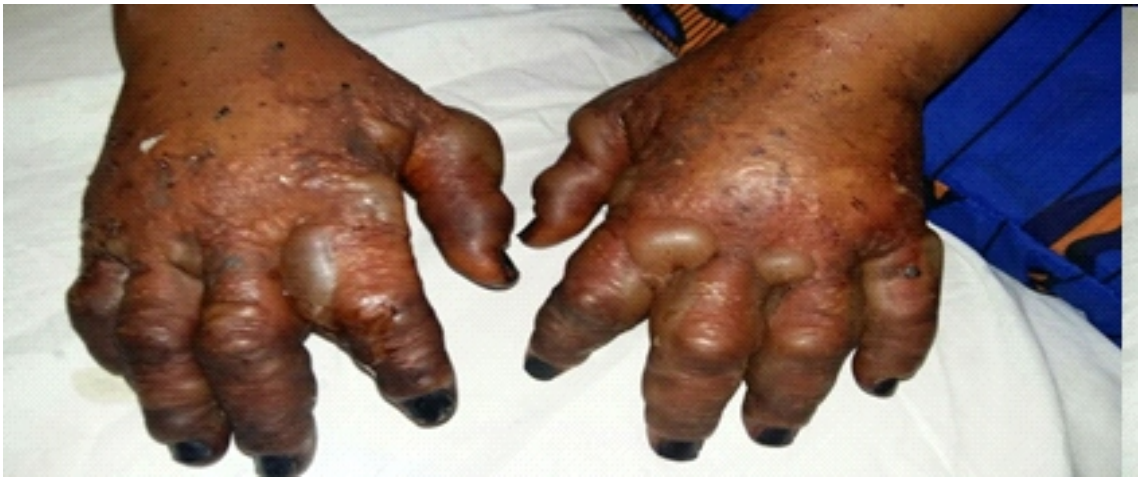

Figure 1 p-phenylenediamine induced Allergic contact dermatitis involving both hands

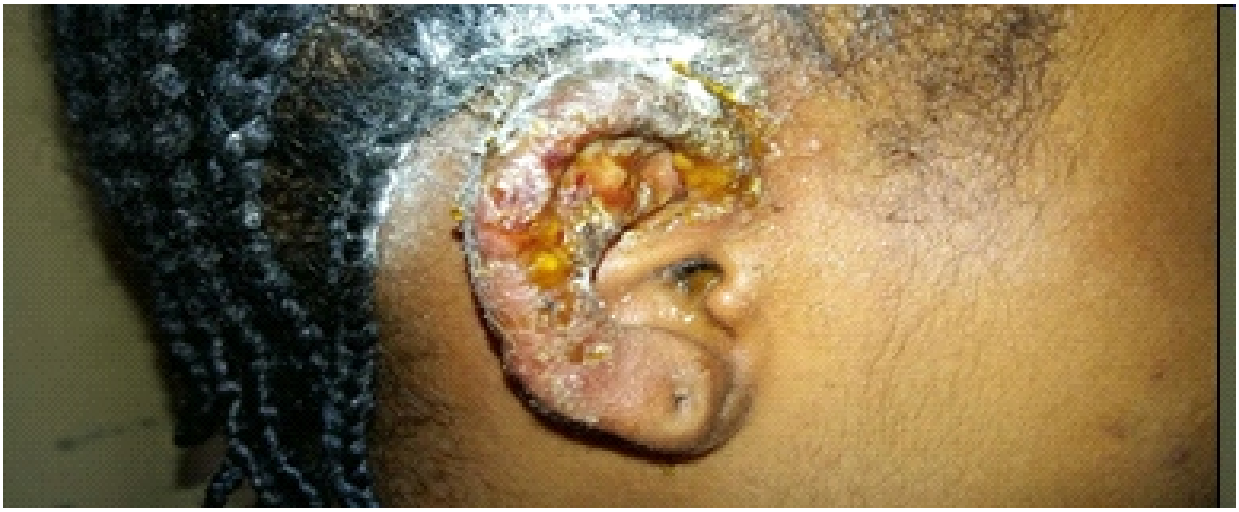

Figure 2 p-phenylenediamine induced Allergic contact dermatitis involving right

REFERENCES

1. VS B. Occupational dermatoses. Curr Opin in Allergy Clin Immunol; 2003. p. 115–23.
2. Mathias CGT. The Cost of Occupational Skin Disease _ JAMA Dermatology _ The JAMA Network. Arch Dermatol; 1985. p. 332–4.
3. A Lee RN. Occupational skin disease in hairdressers - Lee - 2001 - Australasian Journal of Dermatology - Wiley Online Library. Australasian Journal of Dermatology -; 2001. p. 1–8.
4. Ledo RVLC-SJMS. Contact Dermatitis in Hairdressers, 10 Years Later. p. 2005.
5. Occupational allergic dermatoses in hairdressers. 1998. p. 166–7.
6. Meding B. Differences Between the Sexes With Regard to Work-Related Skin Disease - Journals - NCBI. Contact Dermatitis; 2000. p. 65–71.
7. M Cumberbatch, R J Dearman IK. Langerhans cells require signals from both tumour necrosis factor-alpha and interleukin-1 beta for migration. Immunology; 1997. p. 388–95.
8. M Cumberbatch, R J Dearman, Antonopoulos C, R W Groves IK. Interleukin (IL)-18 induces Langerhans cell migration by a tumour necrosis factor-alpha- and IL-1beta-dependent mechanism. Immunology; 2001. p. 323–30.