CASE REPORT

Uses of Antidepressant as Adjunct Treatment of Very Severe Atopic Dermatitis

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Abstract

This is a case of severe atopic dermatitis in a young married male age 37 years. This patient was very anxious and frustrated. The patient was using all the standard treatments for severe atopic dermatitis (A.D) but he was not getting better. The use of antidepressants is not common practice in treating atopic dermatitis. Most general practitioners and dermatologists don't pay attention to how much a patient is suffering not only physically but also mentally and because of this, they don't suggest antidepressants. The role of antidepressants as an anti-inflammatory is still needed to be educated among physicians. This is a vicious cycle. The stress causes the exacerbation of this disease in which patients start itching all over the body and because of itching, lesions all over the body start getting infected as during itching staphylococcus aureus which resides under the nail enter in the lesion and cause infection and because of infection patients have a fever, lethargy, and weakness. The use of antidepressants is a game-changer in the management of atopic eczema.

Keywords: *atopic dermatitis, antidepressants, itching, perivascular infiltrate, spongiotic vesicles, stress, superficial perivascular inflammation.*

INTRODUCTION

The use of antidepressant medication usually not being advised as a treatment of atopic dermatitis as in some studies it has been suggested that antidepressant has the anti-inflammatory role and because of this reason we are reporting this case as this might be useful for other physicians to manage severe atopic dermatitis easily [1, 2]. Usually A.D managed by topical steroids, topical use of the immunomodulators, topical antibacterial in case of infection but after all these maneuvers, patients of severe A.D usually don't get relief.

CASE PRESENTATION

A 37-year-old married male came to us for a second opinion regarding his diagnosed disease atopic dermatitis associated with severe itching and pustular thick crusted plaques *i.e.* erythroderma (**Figs. 1A-1C**).

The patient was having severe anxiety and the patient's family informed us about the suicidal thoughts the patient is talking about [3]. The patient was taking all the standard treatments of atopic dermatitis already *i.e.* azathioprine, tacrolimus, topical application (LPC 3% and clobetasol propionate 70%), antihistamine, and multivitamins.

The patient was very restless with itching rigorously in front of us. Because of severe itching, the patient's lesions were infected and having fever (100F) [4]. His whole body was erythematous and most lesions are infected lesions. He quit his corporate job as he said "I itch all the time and it's really embarrassing for me to hold the social pressure and all of my peers asking about my itch and no one wants to sit and shake hands with me as they thought this disease is communicable" and the patient was became socially isolated in his own office and company didn't want him to be in the office [5]. After quitting his job, the patient was more anxious and living his life miserably. The patient's skin biopsy microscopic picture of the lesion (**Figs. 2A-2D**).

We advised the patient and guide him to continue his former medication as this was the standard treatment he was taking. On the next visit after a month patient came in a very relaxed state and all the lesions have been miraculously subsided and left only the post-inflammatory hyperpigmentation scars (**Figs. 3A&3B**) and we were really surprised about the improvement of this patient as we were not expecting this much improvement and after taking detail history from the patient, he informed us about the addition of one more medicine that is venlafaxine that has been started by a local psychiatrist the day after he visited us one month back as his family took him to the psychiatrist because of his suicidal thoughts and continuously weeping and not getting enough sleep.

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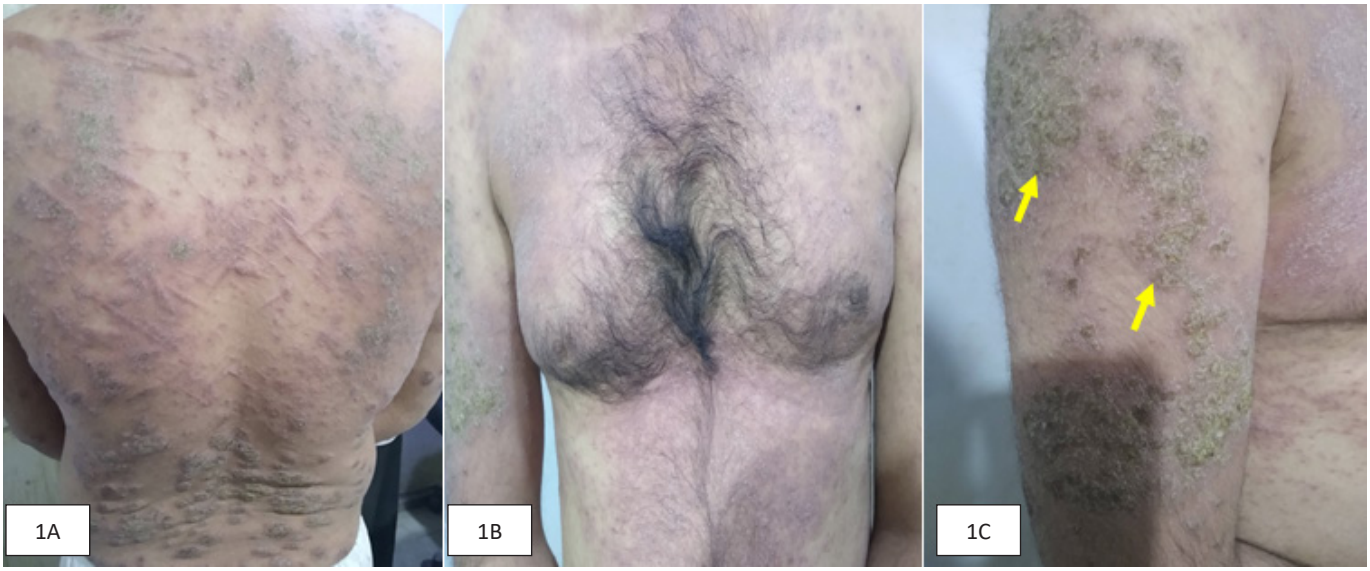

Fig. (1): (A): Edematous and crusted papular lesions on upper back and lower back thick discrete hyperkeratotic papules and plaques. **(B)**: Erythema with non-discrete plaques. **(C)**: Yellow arrows show the ruptured vesicles with infected dry crust on them.

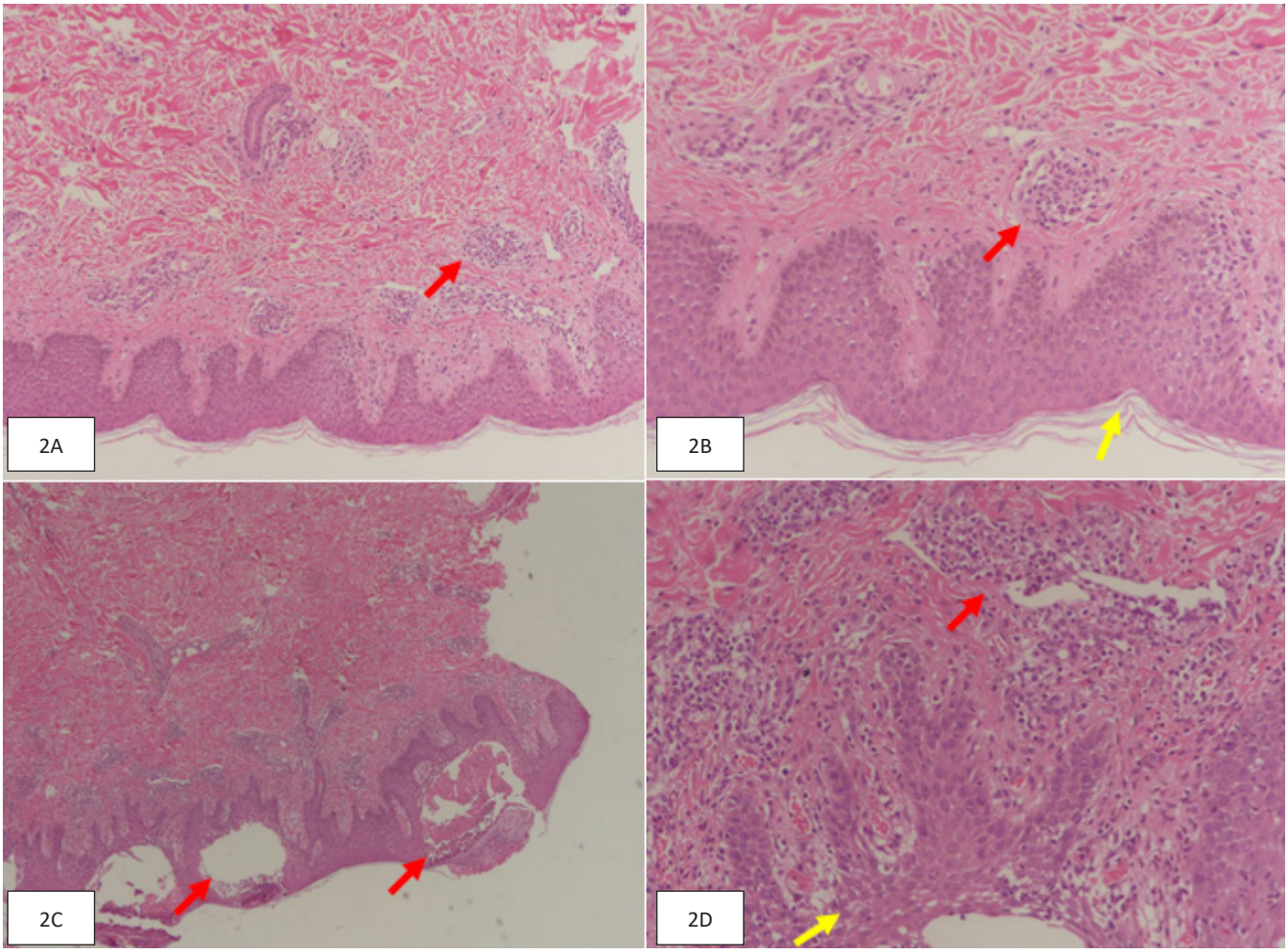

Fig. (2): (A): Hematoxylin and eosin (H & E) stained section at 100x magnification showing superficial perivascular inflammation (red arrow). **(B)**: H & E stained section at 200 x magnification depicting acanthosis (yellow arrow) and superficial perivascular infiltrate (red arrow). **(C)**: H & E stained section at 100x magnification revealing spongiotic vesicles in epidermis filled with fibrin (red arrow). **(D)**: H & E stained section at 400x magnification showing perivascular infiltrate (red arrow) and spongiosis in the epidermis (yellow arrow).

All the treatments were the same as last month that the patient was having for the last 5 months but drastic improvement, in this case, shows the effect of antidepressants drug effects in severe atopic dermatitis cases and because of this miraculous improvement we are reporting this case with documentary evidence in (**Figs. 3A&3B**).

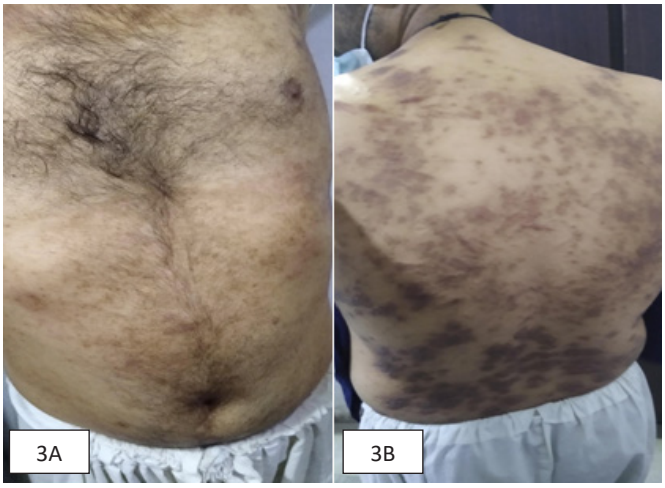

Fig. (3): (A): Post-inflammatory hyperpigmentation in confluent fashion. **(B)**: Few old keloid scars with discrete post-inflammatory hyperpigmentation.

DISCUSSION

Atopic dermatitis is very difficult to explain as it doesn't have specific diagnostic tests and can express different clinical features in every individual. Atopic dermatitis, also known as Atopic eczema. It is a chronic inflammatory and itchy skin condition that can relapse frequently. Rash of eczema is characterized by excoriation marks with lichenified skin mostly on flexural distribution. Following are the Diagnostic Criteria:

For atopic dermatitis, Hanifin and Rajka's UK refinement diagnostic criteria following [6], Atopic Dermatitis with UK diagnostic criteria qualification, the patient should have

a) An itchy skin condition (or scratching and rubbing in a child reported by parents) Plus 3 or more of the following:
b) Onset below age 2 years
c) History of skin flexures involvement
d) Generalized dry skin history
e) Diagnosed Atopic Dermatitis in 1st-degree relatives
f) History of skin crease involvement

Developed westernized countries have more prevalence of atopic eczema. Migrant studies show some fascinating designs. Populations of immigrants appear to develop atopic eczema prevalence even greater than that of the native population of their adopted country of origin [7]. Atopic asthma and eczema involve abnormalities not only of primary functions of the epidermis and bronchial epithelium but also genetic and immune dysregulations [8]. Genetic Influence: German, British, Swedish, and Danish populations reported the 4 genome screens. Loci on 1q21, 3p24-22, 3q21, and 17q25 have been identified in association with atopic dermatitis [9]. A gene on chromosome 5q encoding the cytokine gene cluster has been linked to atopic mucosal syndrome and includes polymorphism in the IL-4 gene with atopic dermatitis [10]. A gene at 16p 11.2-12 encoding the alpha chain of IL-4 receptors has been linked to atopic eczema [11]. Early-onset of atopic eczema has a worse prognosis, and persistence of disease into adulthood and it can be predicted by above mentioned genetic defects.

Mothers transfer atopic dermatitis more than fathers. Risk of a kid getting atopic eczema chances are higher if the mother is affected with atopic eczema [12]. If mothers are atopic in comparison to the paternal atopy then the cord blood IgE is high in babies.

Environmental factors are the most influencing. The level of airborne pollutants for *e.g.* sulfur dioxide, dust, nitrogen oxide, *etc* are the main influencing factors. The patient usually lives in urban cities and has more chances to get atopic eczema because of more environmental pollution [13]. Interaction with environmental microbes may be important in the causation of atopic eczema. It can happen in many ways, a) Maturation of the immune system depends upon the early life exposure so that the irregularity associated with the formation of allergies and production of IgE antibodies does not occur. b) Staphylococcal toxins or the presence of Malassezia yeasts on skin, induced or exacerbated atopic eczema. House dust mites are also the strongest factors for exacerbation of atopic eczema [14]. Differences between the western world and the developing world regarding the incidence of allergic diseases and atopic eczema could be the possible role of microbes in the early maturation of the immune system [8]. It has been reported to reduce the risk of atopy by more than 60% to toxoplasma gondii, helicobacter pylori, and some viruses [15]. Atopic eczema patients are prone to certain infections of the skin. Staphylococcus aureus is the most common skin infection, in addition few viruses (Herpes Simplex Virus 1 and 2, poxvirus of the molluscum contagiosum, human papillomavirus-induced warts) and few fungal infections, are the frequent pathogens causing, severe complications. Following are the Vascular and Pharmacological Abnormalities:

a) Atopic dermatitis shows a tendency to vasoconstriction responses in small blood vessels:
b) Skin pallor after stroking
c) Blanching delayed with acetylcholine
d) Nicotinic acid esters cause a white reaction
e) Histamine cause an abnormal reaction on the affected skin
f) Finger temperature persistently low
g) On exposure to cold pronounced vasoconstriction

Episodes of psychological stress in atopic dermatitis patients exacerbate and complicate the symptoms of the atopic dermatitis patient. The role of the mechanism of mental stress on the exacerbation of signs and symptoms of atopic dermatitis patients still needs to be unmasked. In atopic dermatitis patients, stress-induced rise in free

cortisol is reportedly lower, it could be one of the reasons for psychological stress effects on enhancement of signs and symptoms. The age of the onset is between 2 and 6 months in the majority of cases, but it may start at any age. Eruption of lesions distribution varies with age, a) Infantile Phase: Most commonly lesions start on the face but may occur anywhere on the body. When a child starts crawling the extensor area of the body mostly the elbow and knee are affected. The lesion consists of discrete or confluent papules with the erythema. The lesions are very itchy and after excessive rubbing may start bleeding. b) Childhood Phase: After 2 years, elbow and knee flexures are mostly involved with the sides of the wrist, neck and ankles. Papules mostly erythematous and edematous tend to be replaced by thickening of skin *i.e.* lichenification. c) Adult Phase: This phase is similar to the childhood phase, with lichenification, especially of the flexures and hands. This phase is most characterized by lichenification of vermillion of the lips and around nipples in young females. Asthma and Hay Fever (Allergic Rhinitis) occur in 30- 50% of cases of atopic dermatitis. Adermatoglyphia (loss of fingerprints) can also present with atopic dermatitis [16]. Transepidermal water loss through the stratum corneum is the major cause of dry skin in atopic dermatitis patients. Patients if older than 18 years or if younger than his/her family should be counseled first about that disease. In general advice, trigger factors should be reduced. Use of the emollients frequently. Bathing is helpful as long as soap and foam should be avoided and after bathing dispersible oil is very helpful [17]. Topical steroids (1% hydrocortisone) are mostly used to subside the inflammation of atopic dermatitis. For oral therapy use of antihistamine, antibiotics (against staphylococcal aureus infection such as erythromycin and flucloxacillin are usually prescribed), and use of oral steroids can be used in severe cases.Few non-conservative techniques also used for the treatment of *e.g.* wet wrap technique and topical calcineurin inhibitor (*e.g.* tacrolimus) can be used. Phototherapy and avoidance from triggering factors for *e.g.* mites, food causing allergy should be avoided. Last but not least, the use of antidepressants are always used in mild, moderate, and severe atopic eczema as they can work as an anti-inflammatory too with dealing with anxiety in atopic dermatitis patients but it is not recommended anywhere. The purpose of this case report is that every general physician and dermatologist should know the importance of antidepressants for the eruption and exacerbation and mental well-being of atopic patients.

CONCLUSION

Diagnosis of atopic eczema is not very problematic as it can be diagnosed easily by evaluating the signs and symptoms of the disease. There is no specific test for this disease. Treatment is helpful but the main factor that most physicians neglect in this eczema is the psychological stress that the patient is facing. This stress drags the patient into a vicious cycle of continuous thinking about this disease's signs and symptoms and the effects of these thoughts on the life of the patient.

CONSENT FOR PUBLICATION

Written informed consent was obtained to publish this Case report.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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