CASE REPORT

Unusual presentation of erythema elevatum diutinum mimicking a giant wart on the heels of a Filipino male : a case report

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Erythema elevatum diutinum (EED) is a rare condition believed to be a form of chronic recurrent leukocytoclastic vasculitis possibly secondary to vascular immune complex deposition. The disease is characterized by symmetrical, red, brownish-purple, and yellow papules, plaques, and nodules distributed mainly over the extensor surfaces of the extremities. We report a 61-year-old male with an atypical presentation of such disease as a giant warty lesion on the heels. Histologically, a spectrum from leukocytoclastic vasculitis to vessel occlusion and dermal fibrosis is seen in EED. These histological findings were present in the histopathological reading of the patient which established its diagnosis and further ruled out verruca vulgaris. The disease is associated with many disease entities, which include human immunodeficiency virus, malignant conditions, chronic infection, and autoimmune and connective tissue disorders. None of these conditions was present in the patient as manifested in the history, physical, and laboratory examinations. However, the patient has a low hemoglobin and a G6PD deficiency which makes him a bad candidate for dapsone therapy which is the main treatment for EED. Tetracycline, niacinamide and plain vaseline + salicylic acid were given initially for 4 weeks but no improvement was noticed. It was then shifted to 10mg intralesional corticosteroid and urea paste 40%. Niacinamide still was given. There was a marked thinning of the lesions. The medications were continued and were slowly tapered. More improvement of the lesions was observed.

Keywords erythema elevatum diutinum, giant warty lesion, verruca vulgaris, G6PD deficiency, dapsone intralesional corticosteroid, niacinamide

INTRODUCTION

Erythema elevatum diutinum (EED) is a rare, chronic, and recurrent dermatosis affecting adults. The disease is characterized by symmetrical, red, brownish-purple, and yellow papules, plaques, and nodules distributed mainly over the extensor surfaces of the extremities. EED is usually associated with many disease entities which include human immunodeficiency virus, malignant conditions, hematologic abnormalities, chronic infection, and autoimmune and connective tissue disorders. Dapsone is usually the treatment of choice unless there are contraindications to its use. This is a case of an unusual appearance of EED on the heels of a 61 y/o male. He has no concomitant illness usually associated with EED. The management was affected by the presence of glucose-6-phosphate dehydrogenase (G6PD) deficiency which made the drug of choice for EED, dapsone to be contraindicated. There has been no report so far in the literature of EED with a co-existing G6PD deficiency.

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CASE REPORT

A 61-year-old male family driver from Pandacan, Manila was first seen in the Department of Dermatology of Jose R. Reyes Memorial Medical Center (JRRMMC) in 1995 for multiple erythematous and hyperpigmented nodules and plaques over the elbows, right knee, and heels.

Eighteen years prior to consultation, he noted multiple papules and vesicles on both knees which were followed by erythematous macules and patches on both elbows and knuckles of both hands. These were accompanied by slight pruritus but there was no pain. The lesions on the elbows and right knee became hyperkeratotic, hyperpigmented, and nodular. No other accompanying symptoms were noted. This was followed by the appearance of erythematous, some painful pruritic papules, and plaques on the heels. There was fever but no arthralgia. The lesions on the heels increased in size rapidly and later became warty and ulcerated with foul-smelling discharge. Varied consultations were done with different private doctors and institutions but no improvement was noted.

Fourteen years ago, and with the persistence of the hyperpigmented nodules on the elbows and knees and warty plaques with ulceration and discharge on the heels, he consulted at Jose R. Reyes Memorial Medical Center (JRRMMC). A 4mm skin punch biopsy (SPB) was done on two sites (knee and heel) which revealed EED. He was started on niacinamide 30mg TID, hydroxyzine 25mg OD and oxytetracycline ointment BID for a month. There was resolution of the foul-smelling discharge and relief of the pain and the pruritus. He was then lost to follow up. Consultations were done at other institutions. He then stopped seeking medical help and opted to self-medicate with dapsone.

Eight years later, still showing hyperpigmented nodules and hyperkeratotic plaques accompanied by dizziness, he came back to our institution for follow up. A complete blood count was done which showed a decrease in hemoglobin (undocumented). G6PD test was also done which presented a decrease at 14 mU/1B of erythrocytes. He was advised to discontinue dapsone. He was prescribed with ferrous sulfate 500mg BID, clobetasol ointment BID, cetirizine 10mg 1 tab OD, and nimesulide 100mg 1 tab BID for 2 weeks. This afforded slight relief of symptoms. He was then lost to follow-up. A few consultations at other institutions were done where he was prescribed medications which he did not take. He was lost to follow up and self-medicated with dapsone.

Two weeks later, there was persistence of the hyperpigmented nodules on the elbows and knees as well as the hyperkeratotic plaques on both heels. The appearance of new hyperpigmented nodules on his knuckles prompted him to return to our institution for consultation.

The pertinent negatives for the review of systems include: no body weakness, no myalgia, no joint pains, no seizure, no headache, no dyspnea, no palpitations, and no abdominal pain. He is an occasional drinker of alcohol with a smoking history of 10 years. No history of intravenous illicit drug abuse or blood transfusion. There was no history of multiple sexual partners as well. Family history revealed no similar illness or history of malignancy.

On physical examination, he was conscious, coherent and oriented to time, place and person. He had a blood pressure of 110/80, heart rate of 90 bpm, respiratory rate of 19 cpm and a temperature of 37.2^{o}C. He had a pink palpebral conjunctiva and a slightly icteric sclera. He has no jaundice. There were no palpable lymph nodes nor any mass noted. Chest and abdominal examinations were normal. On dermatologic examination, there were symmetrical hyperpigmented nodules and plaques over a hyperpigmented base topped with slightly white scale on elbows, knuckles, and right knee measuring 1.5 x 1.5 cm in their widest diameter (Figures 1-3); large hyperkeratotic plaques topped with dry white to yellow scales and some topped with dark crusts measuring 6 x 2.5 cm in its widest diameter, multiple palpable purpura on both lower extremities were also noted (Figure 4).

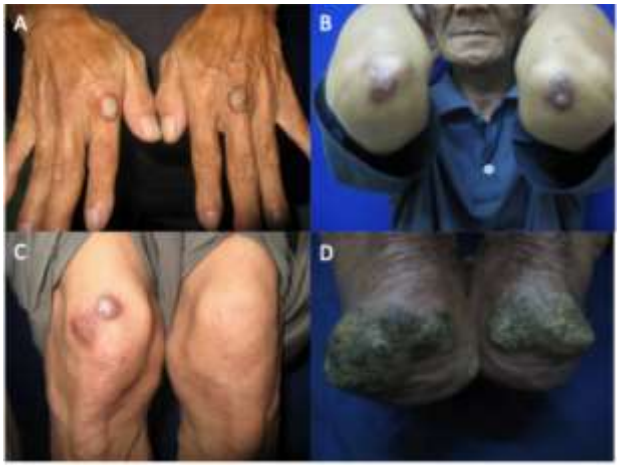

Figure 1. Nodules over a hyperpigmented base, topped with slightly white scales located A) symmetrically on the knuckles of both hands measuring 1x1.5 cm in its widest diameter; B) symmetrically on both elbows coalescing into a plaque over a hyperpigmented base, measuring 1.5x1.5 cm in its widest diameter; C) on the right knee measuring 1x1.5 cm in its widest diameter; and D) symmetrically on both heels forming into large hyperkeratotic plaques topped with dry white to yellow scales some topped with dark crusts measuring 6x2.5 cm in its widest diameter.

Laboratory investigations revealed a decreased level of hemoglobin and hematocrit of 106 g/L and 0.310, respectively. There was reticulocytosis at 4% (NV 0.5-1.5%). The uric acid, BUN, creatinine, SGPT and SGOT were all normal. Peripheral Blood smear revealed normocytic, normochromic and adequate platelets. HIV screening was non-reactive.

The findings of a 4 mm SPB on a palpable purpura on his left leg revealed leukocytoclastic vasculitis consistent with EED. Epidermis is normal (Figure 6). In the dermis are blood vessels with fibrinoid

degeneration infiltrated with neutrophils and eosinophils. There are also extravasated RBC (Figure 7).

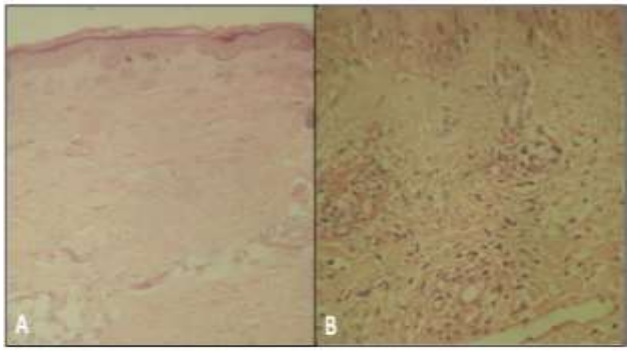

Figure 2. Skin punch biopsy done on a palpable purpura on the left lower extremity showed A) a normal epidermis on low power (x10) magnification view using hematoxylin & eosin stain. In the dermis are blood vessels with fibrinoid degeneration. B) Higher magnification view of the biopsy showed fibrinoid degeneration infiltrated with neutrophils and eosinophils. There are also extravasated red blood cells.

He was prescribed with ferrous sulfate 500mg TID and ibuprofen 200mg 1 cap prn for pain. Three percent salicylic acid + plain vaseline (PV) 100g BID was given for a month but there was no appreciable decrease in the size of the lesions. Salicylic acid was increased to 7% + PV 100g BID and niacinamide 100mg TID and tetracycline 250mg QID were also prescribed. The patient took the tetracycline for 4 weeks and the niacinamide for only 3 weeks. The surface of the lesions became slightly smooth but the size remained the same (Figure 8.A-B). The tetracycline was discontinued and replaced with intralesional corticosteroid injection. This is to avoid the possible hemolysis caused by the prolonged use of tetracycline which could aggravate the present anemia of the patient. The intralesional corticosteroid was administered at a dose of 10mg on both heels and 5mg on elbows, knuckles, and knees. On the other hand, niacinamide was continued. Salicylic acid + PV was replaced with urea paste 40% BID. After 4 weeks there was a moderate elief of pain and tenderness. Also observed were a smoother surface and a slight decrease in size and mild flattening of the lesions (Figure 8.C-F). However, after 4 sessions of intralesional injections, there was only minimal improvement. This prompted us to increase the dosage to 18mg on both heels and to just maintain the 5mg dosage on the other lesions, with a total of 23mg per session. Colchicine was also added at 0.5mg BID. After 2 weeks, a slight improvement was noted (Figure 8.G). Marked thinning of lesions were noted on the 4th and on the 6th week of treatment (Figure 8.H-I). The intralesional steroid injected on the heels was decreased to 14mg because of the decreased number of elevated plaques. On the other hand, the injection of steroid on the other lesions was increased to 10mg because apparently the 5mg being injected initially was not enough to improve the lesions. Further improvements on the elevated lesions on the heels were observed on the 8th week, so the amount of steroid injected was decreased to 8mg. On the 10th week, the lesions on the heels were almost flat and this time the few remaining lesions were just slightly elevated thus the intralesional steroid was discontinued. The lesions on the elbows, knuckles, and knees also started to flatten but not enough to suspend the corticosteroid. The colchicine and the 40% urea paste were maintained.

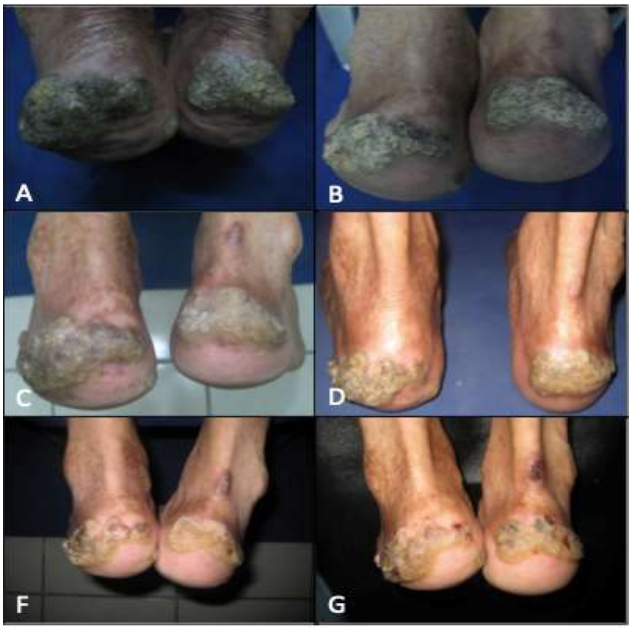

Figure 3. A) Photograph of the lesion on the heels at baseline. B) Four weeks after treatment with tetracycline, niacinamide and plain vaseline + salicylic acid. The lesions became thinner and the surface became slightly smooth. There was a slight decrease in size. C) Two weeks after shifting to10 mg intralesional corticosteroid and urea paste 40%. Niacinamide still was given. Smoother surface with slight decrease in size and mild thinning of lesions were noted. D) Four weeks after treatment with 10mg intralesional corticosteroid and urea paste 40%. Niacinamide was discontinued 2 weeks prior. Minimal improvement of lesions. E) Six weeks after treatment with 10mg intralesional corticosteroid and urea paste 40%. No significant improvement of lesions. F) Eight weeks after treatment with 10mg intralesional corticosteroid and urea paste 40%. Still no significant improvement of lesions.

There was improvement of the hemoglobin and hematocrit after 5 months of using ferrous sulfate, from 106 and 0.310 to 126 and 0.37 of hemoglobin and hematocrit, respectively.

DISCUSSION

EED was first observed in 1888 by Hutchinson and in 1894 by Radcliffe-Crocker and Williams. It is believed to be a form of chronic recurrent

leukocytoclastic vasculitis possibly secondary to vascular immune complex deposition.[1] The disease is characterized by symmetrical, red, brownish-purple, and yellow papules, plaques, and nodules distributed mainly over the extensor surfaces of the extremities.[2] It can be associated with pain, itching, and/or a burning sensation. Arthralgia is the most common systemic symptom.[3] Our patient had complaints of pain and itchiness but he never complained of arthralgia.

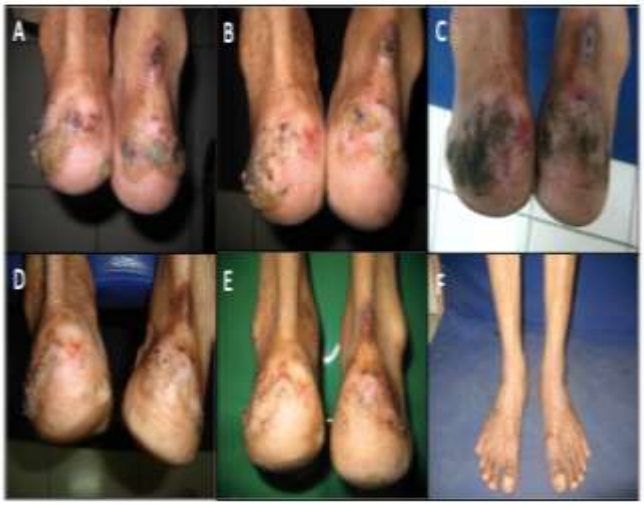

Figure 4. A) Two weeks after increasing intralesional corticosteroid to 18 mg, urea paste 40% and additional colchicine .5mg. Mild thinning of lesions. B) Four weeks after treatment with 18mg intralesional corticosteroid, 40% urea paste and 0.5mg colchicine. There was marked thinning of lesions. C) Six weeks after treatment with 18mg intralesional corticosteroid, 40% urea paste and 0.5 mg colchicine. More thinning of lesions noted. D) Eight weeks after decreasing intralesional corticosteroid to 14mg, 40% urea paste and 0.5mg colchicine. Just a few elevated plaques were observed. E) Ten weeks after decreasing intralesional corticosteroid to 8mg, 40% urea paste and 0.5mg colchicine. The lesions were almost flat. Few lesions were just slightly elevated. F) Multiple palpable purpura on both lower extremeties.

There are some rare manifestations of EED as mentioned in various literatures. One is a case of a 45-year-old woman who presented with a giant annular pattern. Clinical examination revealed red-yellow to brownish infiltrated papules coalescing into annular-shaped plaques of several sizes with a hyperpigmented center affecting the abdomen and the lower limbs, as well as multiple firm nodules on the right palm, elbows, and knees. She was misdiagnosed with a nodular form of leprosy, as this disease is very common in Brazil from which the patient resides.[1] Another case is that of a 47-year-old man who presented with 2 red-brown plaques on his back. The case was unusual in the site of presentation and in the paucity of lesions.[4] Next is a case of a 78-year-old woman with a history of symmetrical erythematous plaques on the arms, and a monoclonal gammopathy, who developed a strange striped reticulate papular dermatosis with central atrophy.[5] There is also a case of a 77-year-old man who had a 6-month history of very firm, exophytic skin nodules, some of which had ulcerated. He had severe pain on pressure and with movement which is unusual in EED patients who present with nodular lesions.[6] Another one is a case of a 77-year-old woman who developed EED with an atypical distribution on the palms, soles, and back of the hands and feet. She first noticed the keloid-like nodules and plaques on the back of some of her fingers and the filiform warty lesions that appeared subsequently.[7] Last is a case of EED simulating as Kaposi's sarcoma in a 52-year-old, HIV-infected female patient with no previous opportunistic infections and a $CD4^+$ count of 164/mm^3. She developed multiple, dusky red large nodules and plaques over the feet, ankles, soles, shins, knees, and elbows.[8]

Our case has an unusual appearance of EED with hyperkeratotic verrucous plaques on the heels. We were able to rule out verruca vulgaris because the typical history of such condition is a newly acquired, slowly expanding, persistent, and often scaly lesion of the skin. Over several weeks to months, there usually is the appearance of additional nearby lesions suggestive of human papilloma virus infection. However, this was not how our patient presented. The lesions on his heels have been existent for almost two decades and have maintained their size for several years. Histologically, a spectrum from leukocytoclastic vasculitis to vessel occlusion and dermal fibrosis is seen in EED.[9] These histological findings were present in the histopathological reading of the patient which established its diagnosis and further ruled out verruca vulgaris.

The incidence of EED is unknown. It presents predominantly in males; usually in the 4th and 6th decade of life.[10] Our case is a male who was in his 40s when his lesions first appeared. In JRRMMC, two cases were diagnosed with EED by histopathology in 2000-2008. Both were females and in their 30s when they first presented with their lesions.

Many patients experience spontaneous resolution after 5-10 years, but some have had lesions for 39 years or even longer.[11] Our patient has had his lesions for 18 years now but still without complete resolution. The disease is associated with many disease entities, which include human immunodeficiency virus, malignant conditions, hematologic abnormalities, chronic infection, and autoimmune and connective tissue disorders.[12] None of these conditions was present in the patient as manifested in the history, physical, and laboratory examinations. A peripheral blood smear was requested to evaluate white blood cells, red blood cells, and platelets. This test definitively identifies and evaluates the presence of immature and abnormal cells. The result was normal suggesting that the patient has

no chronic infections and no hematologic abnormalities either. HIV screening was done since EED is a disease usually associated with AIDS. The patient had no history suggestive of a possible HIV infection and was confirmed by a negative HIV test. Reticulocyte count was requested to know whether the anemia was really caused by hemolysis and not by any other diseases such as aplastic anemia or bone marrow failure caused by infection or malignancy. The elevated reticulocyte of the patient suggested hemolysis probably secondary to dapsone use.

A uric acid measure was requested to exclude the presence of hyperuricemia as this manifests with painful nodules on the joint area. It was ruled out because the value was normal and there is no complaint of joint pain by the patient. BUN, creatinine, SGPT and SGOT tests were also requested as the patient has a slightly icteric sclera and has been chronically using dapsone. Dapsone is metabolized in the liver and excreted by the kidney. The values were within normal limits suggesting that there was no damage yet to these internal organs.

Management of EED remains challenging due to the chronic and recurrent nature of the disease. The aim of the treatment is to alleviate the discomfort associated with the lesions, diminishing damage to the skin, and eradication of associated conditions. The treatment of choice is dapsone.[12] The responsiveness of EED to this drug is not completely known but is thought to be secondary to its inhibitory effects on neutrophil chemotaxis and function.[10] However, this was contraindicated as our patient had a G6PD deficiency. In patients with G6PD, deficiency hemolysis may be provoked by a dose less than 50mg per day of dapsone. The most important adverse effects are hemolytic anemia and methemoglobinemia.[13] Our patient had been taking dapsone against medical advice for several years . This is the reason why his CBC was always requested every follow up. Anemia had always been reflected in his blood picture since the time he started taking dapsone. Surprisingly, his anemia had not really gone down so low that he would have needed blood transfusion considering how significantly low his G6PD was. He was just maintaining on ferrous sulfate to treat his anemia. To the best of our knowledge, there has been no report so far in the literature of EED with co-existing G6PD deficiency.

G6PD deficiency is an X-linked recessive hereditary disease characterized by abnormally low levels of G6PD or G6PDH, an enzyme especially important in red blood cell metabolism. Individuals with the disease may exhibit non-immune hemolytic anemia in response to a number of causes, most commonly infection or exposure to certain medications or chemicals. These medications include antimalarial drugs (primaquine and chloroquine), sulfonamides, thiazolesulfone, methylene blue, naphthalene, certain analgesics (phenazopyridine and acetanilide) and a few non-sulfa antibiotics (nalidixic acid, nitrofurantoin and furazolidone). On the other hand, drugs that are safe to take are the following: Acetaminophen, Aspirin, acetylsalicylic acid, ascorbic acid, chloramphenicol, ciprofloxacin, diphenhydramine, isoniazid, phenytoin, quinidine, and vitamin K analogues. Foods to be avoided are fava beans, red wine, legumes, blueberry, soya food, tonic water, and bitter melon (ampalaya).

Signs and symptoms to watch out for caused by the complications of taking drugs and food that are not safe for patients with G6PD are the following: dizziness, headache, seizure, restlessness, difficulty breathing, palpitation, body weakness, icteric sclera, jaundice, pallor, abdominal pain, and hepatomegaly. The patient only complained of occasional dizziness and had a slightly icteric sclera.

Several other treatment modalities have been reported to be of benefit for EED.[12] Other reported treatments with variable success in EED include niacinamide and tetracycline, colchicines, intralesional, potent topical or oral corticosteroids, phenformin, clofazimine. and cyclophosphamide.[10] Our patient was treated with 10mg intralesional (IL) triamcinolone injection for both heels and 5mg for the other smaller lesions, niacinamide, and urea paste 40%. The IL triamcinolone is used for chronic inflammatory processes and hypertrophic skin disorders. In addition to anti-inflammatory properties, the atrophogenic side effect of corticosteroids also can be used advantageously when treating hypertrophic types of lesions such as the patient's skin lesions. The reasonably safe dosage is less than 25mg to avoid complications.[14] After 8 weeks of 10mg of IL triamcinolone on both heels, only minimal improvement was noted. The patient was then shifted to 18mg of IL triamcinolone for his heel lesions and maintained 5mg for the lesions on knuckles, elbows, and knees. After using 18mg on both heels for only 4 weeks, a marked improvement of the lesions was already noted. After 6 weeks, the IL on both heels was decreased to 14mg because of the decreased number of elevated plaques. Further thinning of lesions needed only 8mg on the next visit of the patient. On the 10^{th} week, the lesions were almost flat with very few plaques that were slightly elevated and did not need additional injections.

The niacinamide is responsible for the suppression of antigen-induced lymphocyte transformation. It might have an effect similar to dapsone which is to suppress the overly active neutrophil chemotaxis present in EED.[14] It was

discontinued in this case as the patient complained of flushing whenever he took the medication. It caused discomfort to him so he opted to stop using it. The Urea paste is used to soften thick, rough, or dry skin. More effective skin softening-properties are achieved with the 40% concentration.

The patient was also given colchicine to control the pain on his heels that he complained about. This drug is also one of the recommended treatment for EED. The mechanism by which it affects the vasculitic lesions of EED is attributed to the antitubulin effect of the drug, which inhibits chemotaxis, phagocytosis, and lysosomal degranulation of polymorphonuclear cells.[16]
Had the lesions of the patient been non-responsive to non-invasive treatment then the option of combining intralesional corticosteroids and surgical excision to treat late stage lesions could have been an alternative. Surgical excision is a treatment option that has been recently used and has shown excellent results.[17]

CONCLUSION

EED is a rare skin condition which is considered distinct for its unique clinical pattern and chronicity. Its rarity coupled with the possibility of encountering a bizarre appearance may compromise an accurate identification of the disease as exemplified by our case which is quite unusual for its atypical presentation as a giant warty lesion. This warrants the use of a biopsy in making the correct diagnosis and not just relying on clinical manifestations. It would help in the timely management of the illness thus preventing long standing lesions which are more difficult to manage.

With the infrequent occurrence of EED, its incidence is unknown and reports in the literature on treatment options are primarily anecdotal or based on experience on case series.
For almost two decades, our case never had a complete resolution of his symptoms due to his non-compliance and failure to follow up, not to mention the resistance of EED to treatment. Dapsone, the drug of choice for EED which afforded him significant relief from pain was even contraindicated because of his G6PD deficiency which made us resort to other treatment modalities.

The use of intralesional corticosteroid may not have been effective initially to our patient but with the right dosage, a substantial improvement of lesions was observed after only four weeks. After 10 weeks, the lesions were almost flattened without the need for further intralesional triamcinolone. The concomitant use of colchicine might have contributed in the improvement of the lesions as it was studied already in one case report to have beneficial effects for EED lesions.

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