

PAPER DETAILS

TITLE: A Case Of Multiple Necrobiosis Lipoidica Diabeticorum: A Good Response To Acetylsalicylic Acid Treatment

AUTHORS: Çagnur ÖZCANLI,Mehmet YILDIRIM,Vahide BAYSAL,Nermin KARAHAN

PAGES: 0-0

ORIGINAL PDF URL: <https://dergipark.org.tr/tr/download/article-file/196497>

A Case Of Multiple Necrobiosis Lipoidica Diabeticorum: A Good Response To Acetylsalicylic Acid Treatment

Çağnur Özcanlı¹, Mehmet Yıldırım¹, Vahide Baysal¹, Nermin Karahan²

¹Suleyman Demirel University, Medical School, Department of Dermatology , Isparta-Turkey

²Suleyman Demirel University, Medical School, Department of Pathology , Isparta-Turkey

Özet

Asetil Salisilik Asit Tedavisine İyi Yanıt Veren Multipl Nekrobiyozis Lipoidika Diyabetikorum Olgusu

Nekrobiyozis lipoidika diyabetikorum (NLD) diyabetik hastaların %0,3'ünü etkileyen, nadir görülen, kronik granülomatoz bir deri hastalığıdır. Etiyopatogenezinin halen tam olarak bilinmemesine rağmen mikroanjiyopatinin önemli rol oynadığı düşünülmektedir. NLD tedavisinde birçok farklı ilaç kullanılmıştır. Biz salisilik asitle başarılı bir şekilde tedavi edilen bir NLD olgusu sunuyoruz.

Anahtar kelimeler: Nekrobiyozis lipoidika diyabetikorum, asetilsalisilik asit, tedavi

Abstract

A Case Of Multiple Necrobiosis Lipoidica Diabeticorum: A Good Response To Acetylsalicylic Acid Treatment

Necrobiosis lipoidica diabeticorum (NLD) is a rare, chronic and granulomatous skin disorder that affects 0.3% of diabetic patients. Although the etiology and pathogenesis of NLD is still controversial, it is thought that microangiopathy has an important role. Various medications have been previously used in the treatment of NLD. We have reported a case of NLD that successfully treated with salicylic acid.

Keywords: Necrobiosis lipoidica diabeticorum, acetylsalicylic acid, treatment

Introduction

Necrobiosis lipoidica diabeticorum (NLD) is a rare, chronic and granulomatous skin disorder that affects 0.3 % of diabetic patients (1-4). Although the lesions of NLD are usually located symmetrically in the pretibial region, they have been reported to occur on the scalp, abdomen and the upper extremities. Early lesions of the NLD present as nondiagnostic erythematous papules and evolve to annular lesions that have a yellowish-brown color, dilated blood vessels and central epidermal atrophy. They are extremely chronic and indolent, shallow, frequently painful ulcers often appear in longstanding lesions (1-5).

The pathogenesis of NLD is still controversial but it is thought that microangiopathy has an important role in the pathogenesis (1,2,6-8). The majority of the NLD patients are diabetic (3). Both insulin-dependent and insulin-independent patients are affected (3,4,7). However progression of the lesions does not correlate with the normalization of hyperglycemia (5). The

lesions of NLD are characteristic and easily recognized by well developed plaque. Sarcoidosis and granuloma annulare are considered in the differential diagnosis (2). Various treatments have been previously used without consistent benefit: these include topical, intralesional and oral corticosteroids, topical retinoids, oral fibrinolytic agents including pentoxifylline, nicotinamide, aspirin, dipyridamole mono or combination therapy, cyclosporine, chloroquine, PUVA therapy, prostaglandin E₁ and skin flap transfer combination (1-11). However there has been no statistical study comparing the effects of these treatments (2). We have reported the successful treatment of NDL with salicylic acid.

Case

43-year old female patient. The patient was diagnosed as type II diabetes five years ago. She had a history of persistently tender plaque which was formed up from

papules at the first toe of the right foot for 4 years. A few months later, approximately 2x3 cm diameter oval plaques with an atrophic, telangiectatic center and violaceous border were developed on the front and the lateral parts of the both tibia. The patient was evaluated in the Department of Internal Medicine for regulation of blood glucose level. She had 5 oval plaques which yellow colored with violaceous border, irregularly shaped, atrophic, telangiectatic center and located in the front and the lateral part of the both tibia and on the right foot (Figure 1). The lesions were moderately pruritic.



Figure 1. Clinical appearance of the lesions before ASA treatment

No pathological parameters were observed in the hematological and biochemical values of the patient except blood glucose level: 226 mg/dl and cholesterol: 213 mg/dl. Histopathologic examination of the lesion on the lateral part of the right tibia was identified as necrobiosis lipoidica diabetica (NLD). The histologic features were mononuclear inflammatory cell infiltrations around the capillary, collagen bundle degeneration and necrosis in the middle dermis, dermal endothelial swelling and flat capillary proliferations. (Figure 2)

The patient was treated as occlusion mometasone furoate cream with intervals. While the patient was using mometasone furoate cream formation of new lesions and lesion activation (width of plaque, border



Figure 2. Histopathological appearance of lesion on the lateral part of the right tibia

elevation, plaque infiltration and erythema) continued. We couldn't prefer systemic corticosteroid and cyclosporin because our patient was diabetic. The patient was treated with oral acetylsalicylic acid (ASA) 200 mg at 48-hourly intervals and with mometasone furoate cream. After two-months of this treatment, stabilization of plaque size, decreased erythema and infiltration was occurred. She had no pruritis. The topical mometasone furoate therapy was discontinued at the second month of the treatment. ASA treatment continued for 10 months. There was no any adverse effect. At the end of the treatment there weren't any new lesions, and erythema and infiltration of the old lesions were decreased. We identified minimal atrophic scar tissue and hyperpigmentation on the old lesions. (Figure 3)

Discussion

NLD is a rare dermatologic disorder usually observed in the diabetic patients (2,3,5). Most patients with necrobiosis lipoidica are either known diabetics (60%), or develop diabetes later (25%) (12). Although the pathogenesis and etiology of NLD is still controversial, it is thought that microangiopathy has an important role (1-4,9,11,12). There is endothelial proliferation in the middle and the deep dermal vessels. The process may lead to partial and, rarely, complete occlusion of the lumen. However transcuta-



Figure 3. Clinical appearance of the lesions after ASA treatment

neous measurements of oxygen pressure shows values significantly lower than in normal skin (6,9). According to these results the decrease in the perfusion is identified. Necrobiosis of the collagen is thought to develop as an aftermath of the resultant ischemia (6,13). This supports a vascular origin of necrobiosis lipoidica, involving reduced vascular perfusion combined with a diffusion block (6). It has been hypothesized that increased platelet aggregation may be a trigger factor in the vascular changes (1,2,6,11,14). Altered platelet function has been reported in patients with diabetes mellitus, and enhanced platelet aggregation and coagulation incriminated in the pathogenesis of diabetic microangiopathy (12).

Recently, several investigations have been made treating necrobiosis lipoidica with ASA. ASA inhibits the cyclo-oxygenase converting arachidonic acid into prostaglandin in the vessel walls and thromboxanes in the platelets (11,14,15). Therefore the inflow of calcium ion into the platelets decreases and antiaggregation effect occurs (15). Thromboxane is considered to be prothrombotic as acting as a vasoconstrictor and is able to induce aggregation of the platelets. In contrast, prostacycline inhibits platelet aggregation, and is a vasodilator too. It was concluded that the missing effect probably was due to the high dose of ASA which then acted at the cyclo-oxygenase in both the

platelets and the endothelial cells (14,15). In the literature, different dosage schedules have been discussed at which ASA selectively inhibits platelets thereby preventing the prothrombotic effect of thromboxane (14). Recently evidence has suggested that there may be a difference in the sensitivity of the platelets and vessel walls cyclo-oxygenase to ASA, and that a lower dose given less frequently may well have a beneficial effect on the prostacyclin/thromboxane balance (16). Karkavitas and Beck have suggested that low dose oral ASA treatment has been successful in the NLD by inhibition of platelet aggregation and prevents some microvascular occlusion (11,14). We used ASA with an amount of 3,5 mg/kg at 48-hourly intervals and for optimal antiaggregation treatment. At the second month of the treatment, topical mometasone furoate was discontinued. The patient took 200 mg ASA for a period of 10 months and no new lesion formation was observed after two months of treatment. No side effects were observed during the treatment. After ASA treatment minimal hyperpigmentation and atrophic scar has been observed in the previous lesion localizations. According to this result, ASA can be regarded as an effective and safety method of NLD treatment. However this result should be confirmed in randomized, double-blind comparative clinical trials.

References

1. Nguyen K, Washenik K, Shupack J. Necrobiosis lipoidica diabetorum treated with chloroquine. *J Am Acad Dermatol* 2002;46(2): 34-6.
2. Freinkel RK. Diabetes Mellitus. in Fitzpatrick TB, Eisen AZ, Wolf K, Freedberg IM, Austin KF (eds): *Dermatology in General Medicine*, New York: McGraw-Hill 2000; 1969-75.
3. Jabbour SA, Miller JL. Endocrinopathies and the skin. *Int J Dermatol* 2000;39: 88-9.
4. Boyd SA. Tretinoin treatment of necrobiosis lipoidica diabetorum. *Diabetes Care* 1999;22(10): 1753-4.
5. Boulton AJM, Cutfield RG, Abouganem D, Angus E, Flynn HW, Skyler JS, Penneys NS. Necrobiosis lipoidica diabetorum: A clinicopathologic study. *J Am Acad Dermatol* 1988;18(3): 530-7.
6. Noz KC, Korstanje MJ, Vermeer BJ. Ulcerating necrobiosis lipoidica effectively treated with pentoxifylline. *Clin Exp Dermatol* 1983;18: 78-9.
7. Gebauer K, Armstrong M. Koebner phenomenon with necrobiosis lipoidica diabetorum. *Int J Dermatol* 1993;32: 895-6.
8. Erbağcı Z, Erbağcı İ, Özgöztaş O. Tip II Diabetes mellitusta dermatolojik mikrovasküler komplikasyonlar ve diabetik retinopati. *T Klin Dermatol* 1999;9:68-74.
9. Sawada Y. Successful treatment of ulcerated necro-

biosis lipoidica diabeticorum with prostaglandin E1 and skin flap transfer. J Dermatol 1985;12: 449-54.

10. Ling TC, Thomson KE, Goulden V, Goodfield MJD. PUVA therapy in necrobiosis lipoidica diabeticorum. J Am Acad Dermatol 2002;46(2): 319-20.

11. Karkavitas K, Miller JA, Dowd PM, Kirby JD. Aspirin in the management of necrobiosis lipoidica. Acta Derm Venerol 1982;62: 183.

12. Colwell JA, Haluska PV, Sarji K. Altered platelet function in diabetes mellitus. Diabetes 1976;25: 826-31.

13. Brüngger A. Transkutane sauerstoff- und kohlendioxiddruckmessung bei necrobiosis lipoidica. Hautartz 1989;40: 231-2.

14. Beck HI, Bjerring P, Rasmussen I, Zachariae H, Stenbjerg S. Treatment of necrobiosis lipoidica diabeticorum with low-dose acetylsalicylic acid: randomized double blind trial. Acta Derm Venerol 1985;65: 230-4.

15. Mycek JM, Harvey RA, Champe CP, Fisher BD, Cooper M. Anti-inflammatory drugs. Pharmacology, Lippincott- Raven Publishers 1997;401-19.

16. Massoti G, Poggesi L, Galanti G, Abbate R, Neri Serneri GG. Differential inhibition of prostacyclin production and platelet aggregation by aspirin. Lancet 1979;8(2):1213-7.

Yazışma Adresi:

Email: cagnurozcanli@hotmail.com