ID Design Press, Skopje, Republic of Macedonia
Open Access Macedonian Journal of Medical Sciences. 2019 Sep 30; 7(18):3093-3095.
https://doi.org/10.3889/oamjms.2019.560
eISSN: 1857-9655
Global Dermatology

Anetoderma Schweninger-Buzzi: Two Case Reports

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Citation: Wollina U, Mühle D, Lotti T, Vojvodic A. Anetoderma Schweninger-Buzzi: Two Case Reports. Open Access Maced J Med Sci. 2019 Sep 30; 7(18):3093-3095.
https://doi.org/10.3889/oamjms.2019.560

Keywords: Anetoderma; Connective tissue disorders; Chronic infections; Prothrombotic conditions; Histopathology

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Received: 16-Apr-2019; **Revised:** 19-Jun-2019; **Accepted:** 20-Jun-2019; **Online first:** 19-Aug-2019

Funding: This research did not receive any financial support

Competing Interests: The authors have declared that no competing interests exist

Abstract

BACKGROUND: Anetodermas are rare disorders of connective tissue with a focal loss of elastic fibres in the upper and mid dermis. Two types are separated, inflammatory and non-inflammatory.

CASE REPORTS: We report two cases of acquired anetoderma Schweniger-Buzzi type. This non-inflammatory subtype is characterised by skin-coloured or whitish atrophic sac-like protrusions of trunk skin in adult males. Chronic infections and autoimmune disorders have been excluded. The diagnosis had been confirmed by characteristic histopathology.

CONCLUSIONS: Anetodermas are symptomless disorders. They can be easily overlooked. The knowledge of such conditions is of importance to identify patients with a risk of thromboembolic events and underlying infections or autoimmune connective tissue diseases.

Introduction

Cutaneous elastic tissue anomalies are classified as those with increased or abnormal elastic tissue and those with a loss of elastic tissue. To the first group belong disorders such as nevus elasticus, pseudoxanthoma elasticum, or elastosis perforans serpinginosa. Papular elastorrhexis, mid dermal elastolysis and anetoderma fall under the second group [1].

The loss of elastic tissue can be by decreased production of elastic fibers, increased activity of elastase and matrix metalloproteinases, activation of phagocytosis of elastic fibres by macrophages or a combination of these [2]. Here we report two cases of the rare anetoderma Schweninger-Buzzi type in two adult males.

Case Reports

Case 1: A 48-year old male was referred from the rheumatologist to exclude scleroderma. The patient reported the development of asymptomatic skin lesions on his trunk. He was otherwise healthy and had no medical drug therapy.

On examination, we observed more than 100 skin-coloured herniated sac-like lesions on the trunk arranged in Langer's lines. The maximum diameter was about 1 cm. There was no erythema, no scaling, no pruritus (Figure 1).

Laboratory findings, including antinuclear antibodies (ANA), anti-cardiolipin antibodies, and Borrelia serology, were normal or negative.

A skin biopsy was obtained for histology. Narrowed collagen fibres were noted in the whole

dermis. In the upper dermis, there was a focal loss of elastic fibres. The skin appendages were preserved. An inflammatory infiltrate completely missing.

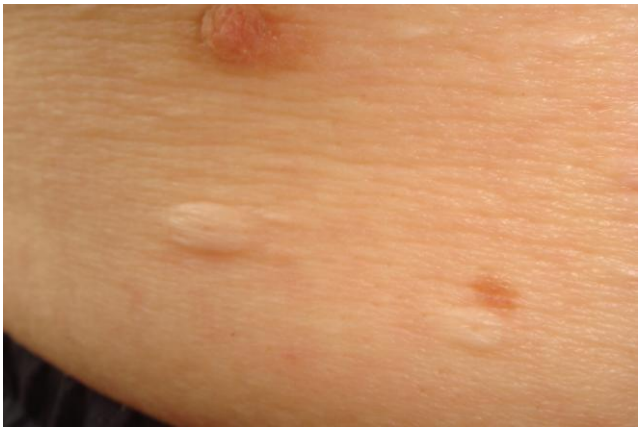

Figure 1: Anetoderma (case #1)

Case 2: A 33-year-old male presented with multiple asymptomatic lesions on the back. His medical history was unremarkable. He did not use any medical drugs.

On examination, we observed skin-coloured sac-like herniated lesions with a size of up to 7 mm arranged along Langer's lines (Figure 2).

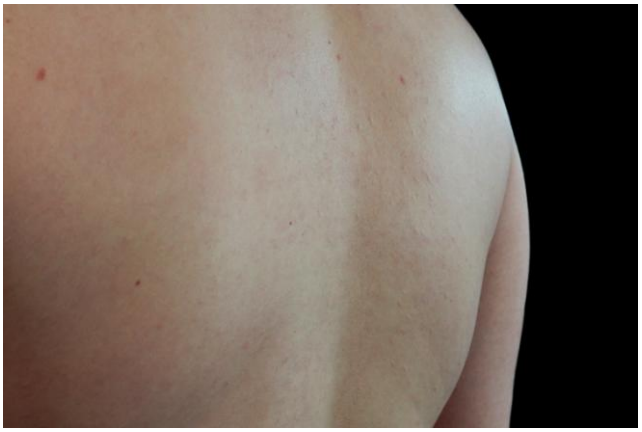

Figure 2: Anetoderma (case #2)

Laboratory investigations demonstrated antimitochondrial antibodies 1:320, mild eosinophilia of 9.6%. Borrelia serology and ANA screening remained negative.

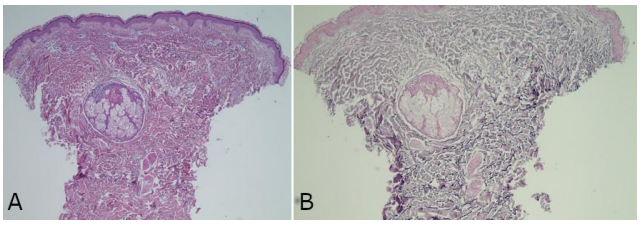

Figure 3: Histopathology of anetoderma Schweninger-Buzzi, case #2; A) Hematoxylin-eosin; B) elastica stain (x 4)

A skin biopsy revealed the same pattern as in the first patient (Figure 3).

In both patients, the diagnosis of anetoderma Schweninger-Buzzi type was confirmed by clinical appearance and histologic findings.

Discussion

Anetoderma is a rare disease. It belongs to the acquired connective tissue disorders and is characterised by localised skin atrophy and loss of elastic fibres [3]. In electron microscopy, fibre fragments have been described [4].

Traditionally, anetoderma has been differentiated into an inflammatory subtype Jadassohn-Pellizari with prodromal urticaria-like lesions, followed by erythematous atrophic lesions [5]. The type Schweninger-Buzzi develops spontaneously without inflammation [6]. Lesions are either skin-coloured or whitish [7]. Hereditary antoderma is extremely rare [8].

Primary anetoderma can be a precursor of autoimmune disorders and a marker for prothrombotic conditions like anti-phospholipid syndrome [7], [9], [10]. Secondary anetoderma has been reported after granuloma annulare [11], secondary syphilis [12], pilomatricoma [13], and bullous Sweet syndrome [14]. Acquired anetoderma following folliculitis is known in patients with Down syndrome [15].

Infection with *Borrelia afzelii* has been considered a possible cause of secondary anetoderma as well [16], [17]. Since Lyme borreliosis is common in Southern Germany [18], it was of particular importance to rule out Borrelia infection in our cases (Table 1).

Table 1: Anetoderma – underlying pathologies

Type	Pathologies
Primary anetoderma	Autoimmune inflammation (connective tissue disorders) Thromboembolic events (anti-phospholipid syndrome, anti-thrombin III deficiency)
Secondary anetoderma	Infectious diseases (Lyme disease, syphilis, HIV, leprosy) Inflammatory disorders (granuloma annulare, Sweet Syndrome, Stevens-Johnson syndrome, folliculitis) Drug induced (penicillamine, penicillin) Metabolic disorders (Wilson's disease) Tumor-associated (Reed syndrome, pilomatricoma, cutaneous lymphomas, xanthogranuloma)

Differential diagnoses include morphea, cutis laxa, pseudoxanthoma elasticum-like papillary elastolysis, lichen sclerosus et atrophicus, lepromatous leprosy, and chalazodermic amyloidosis [1], [19], [20].

Treatment is dependent upon underlying

pathologies. Infectious disorders need antibiosis; inflammatory disorders require immune-modulating drugs and tumours warrant surgery, radiotherapy or anti-tumour drugs. Successful treatment of the underlying pathology may result in the prevention of the development of additional lesions. Once anetoderma has developed, there is no specific drug therapy for the condition available. Corticosteroids, dapsone, colchicine, aminocaproic acid, vitamin E, and niacin yielded meager improvement. Peels and radiofrequency devices have occasionally been employed [21]. Laser therapy has been used to improve the appearance by stimulation of the production of elastic fibres and a decrease in fibre fragmentation after combined 595 nm pulsed-dye laser plus 1550 nm non-ablative fractionated laser [22] and 10,600 nm CO_2 laser [23] [24]. In analogy to depressed scars, dermal filler injections may have a lifting effect [25].

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