

# From dermatomyositis to osteoporosis

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## ABSTRACT

*Our aim is to describe a case of a menopausal woman who started with dermatomyositis and then became a patient at endocrinology because of complicated glucocorticoid osteoporosis. This is a 63-year old female who suddenly started to accuse intense muscle pain associated with redness of the skin. She was confirmed with dermatomyositis and offered high doses of oral metilprednisolone. The thyroid ultrasound showed micronodular background and a small nodule of 0.7 cm without clinical relevance with an increase of antibodies thyroperoxidase antibody and normal thyroid function. Bone turnover markers were within normal in addition to decrease of serum 25-hydroxyvitamin D of 20 ng/ml. DXA (Dual-Energy X-Ray Absorptiometry) showed osteopenia based on a minimum T-score of -2.4 SD. The patient was offered high doses of cholecalciferol 2,000 UI per day and 500 mg of calcium daily in addition to weekly alendronate due to the high risk of fracture because of the glucocorticoid doses. 6 months later she suffered and accidental fall and a hip fracture was confirmed and required surgery. Further on the therapy was switched to zolendronic acid one injection per year. The dermatological and muscle progressively improved thus the doses of corticoids were reduced. As particular observation the presence of autoimmune thyroid condition may increase the risk of a second autoimmune disease like dermatomyositis, also the specific antibodies panel was negative. A multidisciplinary team needs to be involved in associated therapy for dermatomyositis and complications of glucocorticoids use.*

**Keywords:** skin, dermatomyositis, glucocorticoid, osteoporosis

## Abbreviations

BMD = bone mineral density                      SD = standard deviation  
DXA = Dual-Energy X-Ray Absorptiometry    TPO = thyroperoxidase antibody  
PTH = parathormone

## INTRODUCTION

Some skin conditions require high doses of glucocorticoids as dermatomyositis (1,2). This a coetaneous and muscle inflammatory disease of various causes with heterogeneous clinical and histological aspects (3,4). High doses of glucocorti-

coids causes cardio-metabolic and bone complications (5,6).

## AIM

Our aim is to describe a case of a menopausal woman who started with dermatomyositis and

then became a patient at endocrinology because of complicated glucocorticoid osteoporosis.

## MATERIAL AND METHOD

This is a case report. The clinical and endocrine data are provided.

## CASE REPORT

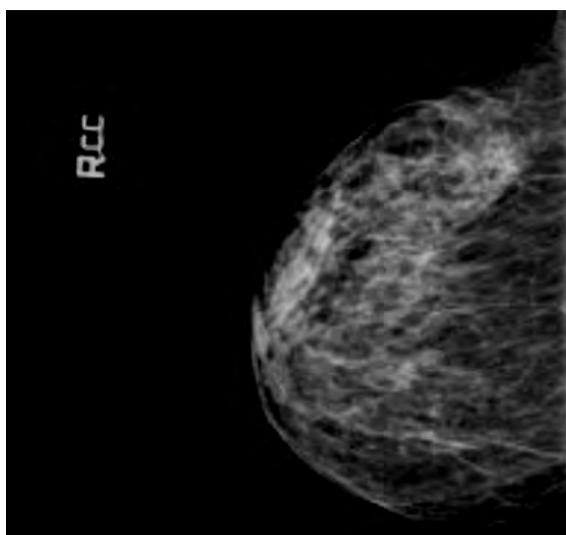
This is a 63-year old female who is non-smoker and had physiological menopause at age of 50 years. She comes from non-endemic area. Her family history is irrelevant. She associates mild increase of blood pressure which is controlled under anti-hypertensive medication. She suddenly started to accuse intense muscle pain associated with redness of the skin. She was confirmed with dermatomyositis based on muscle enzymes and inflammatory profile but all the immune profile did not conclude a particular cause. So she was offered high doses of oral metilprednisolone.

She was referred for an endocrine check up. The clinical examination was irrelevant for a particular endocrine condition. The thyroid ultrasound showed micronodular background and a small nodule of 0.7 cm without clinical relevance with a small increase of antibodies thyroperoxidase antibody (TPO) of 70 U/l with normal levels less than 34 U/l with normal thyroid function (Figure 1). The screening mammogram did not show any condition (Figure 2).



**FIGURE 1.** Thyroid ultrasound showing a small nodule of 0.7 cm at adult menopausal woman

Also the bone turnover markers were within normal limits as well as PTH (parathormone) and a small decrease of serum 25-hydroxyvitamin D of 20 ng/ml was identified. Central DXA (Dual-Energy X-Ray Absorptiometry) showed osteopenia based on BMD (Bone Mineral Density) values according



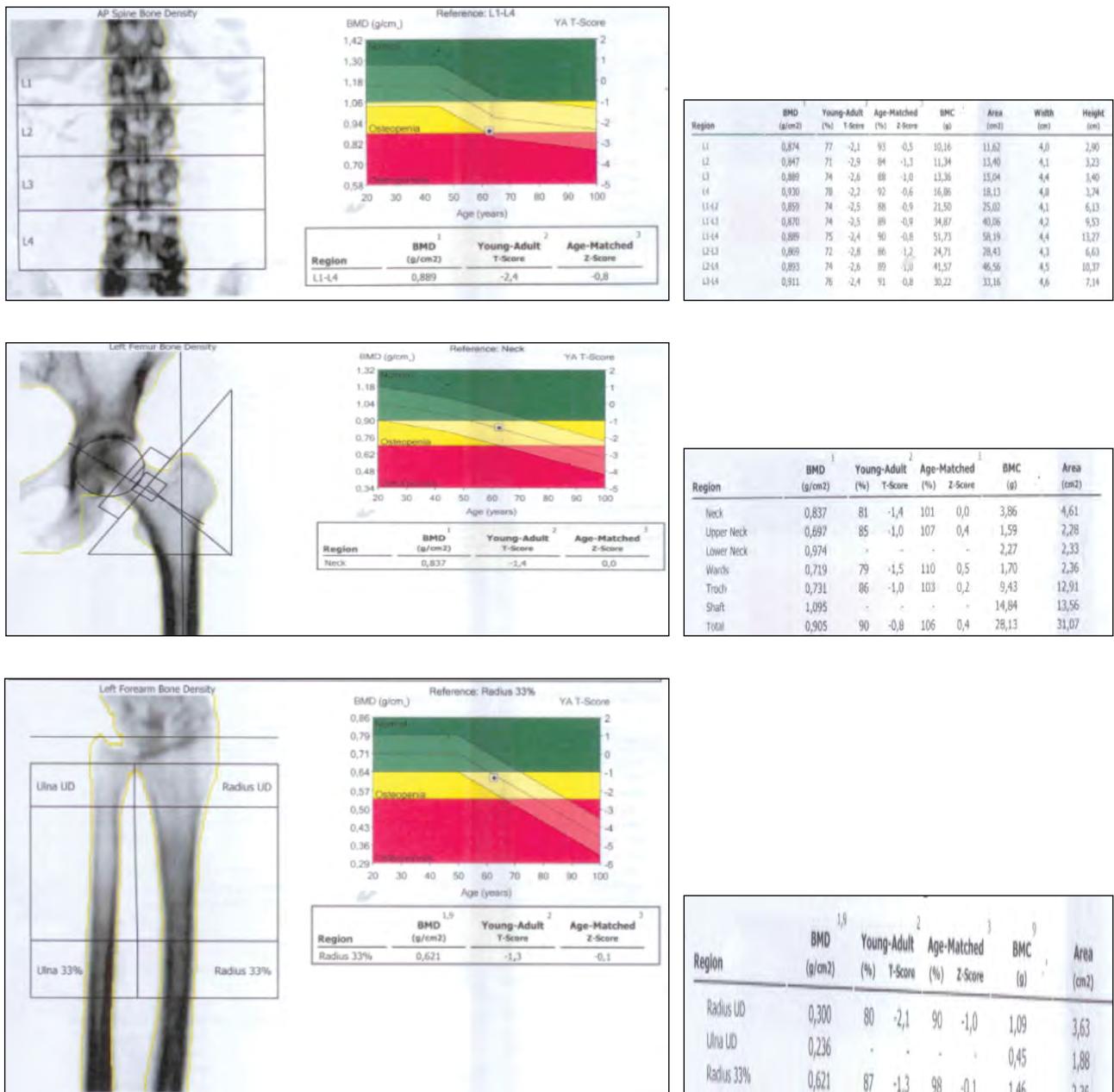
**FIGURE 2.** Screening mammogram on menopausal female with dermatomyositis of unknown cause (no malignancy was suspected)

to a minimum T-score of -2.4 SD (Figure 3). The screening of the vertebral fractures based on profile X-ray of the spine showed no lesion. A paraneoplastic syndrome was not confirmed, neither a potential tumour at computed tomography at the level of thorax and abdomen.

The patient was offered high doses of cholecalciferol 2,000 UI per day and 500 mg of calcium daily in addition to weekly alendronate due to the high risk of fracture because of the glucocorticoid doses. 6 months later she suffered and accidental fall and a hip fracture was confirmed and required surgery. Further on the therapy was switched to zoledronic acid one injection per year. The dermatological and muscle progressively improved thus the doses of corticoids were reduced.

## DISCUSSION

In dermatomyositis typically the inflammatory pain and weakness is proximal and symmetrical and once the diagnosis is established corticotherapy and immunosuppressive drugs are useful (1,2,3,4). Beyond muscle, skin is also involved as well as lungs and joints (polymyositis type) (1,2,3,4). A particular aspects of skin involvement in dermatomyositis is the synchronous rash with muscle involvement as seen in this case (1,2,3,4). The causes of dermatomyositis are autoimmune or cancer-related but sometimes as in mentioned case they remain as “idiopathic” (1,2,3,4). In this particular situation we avoid the use of an osteoanabolic drug as teriparatide for complicate glucocorticoid osteoporosis since an active neoplasia was not entirely excluded (7,8).



**FIGURE 3.** Osteopenia at central DXA in menopausal woman at the beginning of glucocorticoid therapy for a dermatological autoimmune condition

As particular observation the presence of autoimmune thyroid condition may increase the risk of a second autoimmune disease like dermatomyositis also the specific antibodies panel was negative. However, chronic Hashimoto thyroiditis has an increased prevalence in general population being considered the most frequent autoimmune condition which may be related to other endocrine and non-endocrine disorders (poly-glandular autoimmune syndrome) or it may be overlapped with other diseases with a high prevalence in general female adult population like metabolic complications including obesity or mammary cancer (9,10,11).

The mentioned subject developed glucocorticoid induced osteoporosis, not the entire picture of iatrogenic Cushing’s syndrome while she was priory hypertensive (12,13). Interestingly, some studies pointed that, independently of glucocorticoid iatrogenic exposure or immunosuppressive therapy, in dermatomyositis (as seen in polymyositis) there is a high risk of bone loss and osteoporotic fractures (14,15). A population-based study of cohort type showed that persons with dermatomyositis and polymyositis have a 2.99 time higher risk of osteoporosis than the subjects without this diagnosis (15). Another contributor to fall is hypovitaminosis D with a high prevalence in general

population including in menopausal women (16). This is less likely to be related with DXA result at the therapy start and also, based on the therapy with cholecalciferol which was offered to the patient, is not a major contributor to fall at values above 20 ng/mL as seen here (17,18). Generally a patient under anti-osteoporotic medication may be non-responder in cases with very low levels of vitamin D (19). But in our case the levels of 25-hydroxyvitamin D was not so low, as contributors to

fragility fracture is menopause-related estrogens deficiency, glucocorticoid exposure and the underlying dermatological condition itself (20).

## CONCLUSION

A multidisciplinary team needs to be involved in associated therapy for dermatomyositis and complications of glucocorticoids use.

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