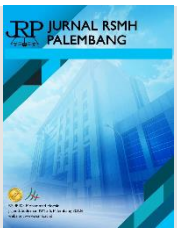
Jurnal RSMH Palembang
Journal Homepage: http://jurnalrsmh.com/index.php/JRP



Ulceration of Corticosteroid-Induced StriaeDistensae in Children with Nephrotic Syndrome

Indri Widya Sari[1*], Rusmawardiana[2], Fifa Argentina[3]

[1]Department of Dermatology and Venereology, Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia

ARTICLE INFO

Keywords:
Striaedistensae
Ulceration
Nephrotic syndrome
Side effects
Corticosteroids

Corresponding author:
Indri Widya Sari

E-mail address:
indri.widya88@gmail.com

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/JRP.v2i1.15

ABSTRACT

Striaedistensae (SD) is linear scar tissue in the epidermis and dermis due to excessive stretching of the skin.Striaedistensaeoccurs due to dysfunction of extracellular matrix components that play a role in skin elasticity increasing sensitivity to minor trauma and ulceration. One of the causes is the long-term use of corticosteroids. Systemic corticosteroids are the mainstay of treatment for nephrotic syndrome, so long-term prescribing of corticosteroids should be carried out with caution. It is reported case of a 15-year-old boy with nephrotic syndrome complains of red streaks appearing on his abdomen, back, buttocks, and lower limbs after 2 months of corticosteroid treatment. Pus-filled nodules develop which become ulcers on the red lines of the back. On physical examination found striaedistensae in the abdominal region, posterior trunk, gluteus, femoral and proximal 1/3 bilateral cruris posterior. Found 3 ulcers on the striaedistensae on the posterior trunk, oval to round shape, size 1x1x0,5 cm to 2x1,5x1 cm, base of necrotic tissue, pus contents, wall echoing, edges are not raised, surrounding tissue is erythematous-livid, tenderness, odor, and no induration. On examination of the ulcer swab with Gram stain found Gram-positive bacteria. The culture results showed Staphylococcus aureus. The patient was treated with topical 0.05% retinoic acid every night at SD, and ulcers were treatedsystemic antibiotics of 2 grams ceftriaxone per day for 1 week, as well as ulcer treatment by compressing 1%0 salicylic acid solution, hydrogel, and foam dressing. After 4 weeks of therapy, there was clinical improvement, thinning striae and reduced ulcer size.

1. Introduction

Corticosteroids are powerful anti-inflammatory agents that are used to improve the clinical conditions of various diseases. One of the diseases that require long-term oral corticosteroid management is nephrotic syndrome (NS). Long-term use of corticosteroids disrupts type I and III collagen synthesis and degradation in the dermis which causes delayed wound healing and skin atrophy, such as SD.[1]

Striaedistensae (SD)is linear scar tissue in the epidermis and dermis due to the stretching of the skin beyond its elastic limit.[2,3] There are two types of SD, striaerubra (SR) and striae alba (SA). Striaerubra is an early phase characterized by erythematous to purplish linear plaques. After a few months, the atrophic, white, and permanent lesions are called striae alba.[4] In addition to corticosteroid use, SD is also associated with various factors such as puberty, pregnancy, and rapid weight gain or loss.[2-4]

Striaedistensae can occur at any age between 5-50 years with the incidence of women two times more often than men. The incidence of SD at puberty is 25-35%, increasing during pregnancy 90%, besides that it often occurs in patients receiving long-term corticosteroid therapy.[2,5] Based on medical records at the Dermatology and Venereology Polyclinic Dr. Mohammad Hoesin Palembang, there were 8 cases from January 2015-December 2019 with ratio of

female and male is 7:1, range of age 11-33 years old.

been reported with varying success rates, both topical and laser therapy.[4,6] One of the topical therapies that can be used is retinoic acid 0.05-0.1%.[6,7] These striaedistensae can ulcerate and infected bymethacillin-resistant *Staphylococcus aureus* (MRSA) and *Pseudomonas* species. Ulceration of the striaedistensae is a rare complication. It requires antibiotics and intensive daily wound care to heal until it is closed.[8]

We report a rare case, long-term corticosteroid-induced ulceration of striaedistensae in a 15-year-old boy with nephrotic syndrome. It is necessary to understand the adequate doses of long-term corticosteroid to reduce the incidence of SD in patients with nephrotic syndrome. Combination of systemic and topical therapy, as well as ulcer treatment for 4 weeks, gave significant clinical improvement.

2. Case Report

A 15-year-old male complaints of erythematous striae on the abdomen, posterior trunk, gluteus, femoral, and cruris since 6 months ago (**Figure 1**). Ulcers appeared on the striae on the posterior trunk and gluteus with a history of previous pus-filled nodules about 2 weeks ago. The patient had a history

Various options for SD therapy modalities have of nephrotic syndrome 8 months ago and was taking 40 mg methylprednisolone tablets for 2 months.

In dermatological status was found 3 ulcers on the posterior trunk, oval to round in shape, size 1x1x0.5 cm to 2x1.5x1 cm, erythematous base, and necrotic tissue, pus contents, wall echoing, edges not raised, surrounding tissue erythematous-livid, tenderness, smell, and no induration.

Physical examination revealed blood pressure of 140/80 mmHg and body weight of 50 kg. Laboratory examination revealed proteinuria (urinary albumin 9.560 mg/L) and creatinine 93.13 mg/dL. Gram stain examination found Gram-positive coccus bacteria (**Figure 2**). The culture of pus specimen on the ulcer results of *Staphylococcus aureus*. The patient was treated with topical 0.05% retinoic acid every night at SD. The ulcers were treated with systemic antibiotics, injection of ceftriaxone 1x2 grams intravenously per day for 1 week, as well as wound care by compressing 1‰ salicylic acid solution, hydrogel, and foam dressing. There was clinical improvement after 4 weeks. There was thinning of the striae, 2 ulcers healed, and 1 ulcer got smaller size of 1x0.5x0.2 cm. Further therapy and observation are still being carried out.

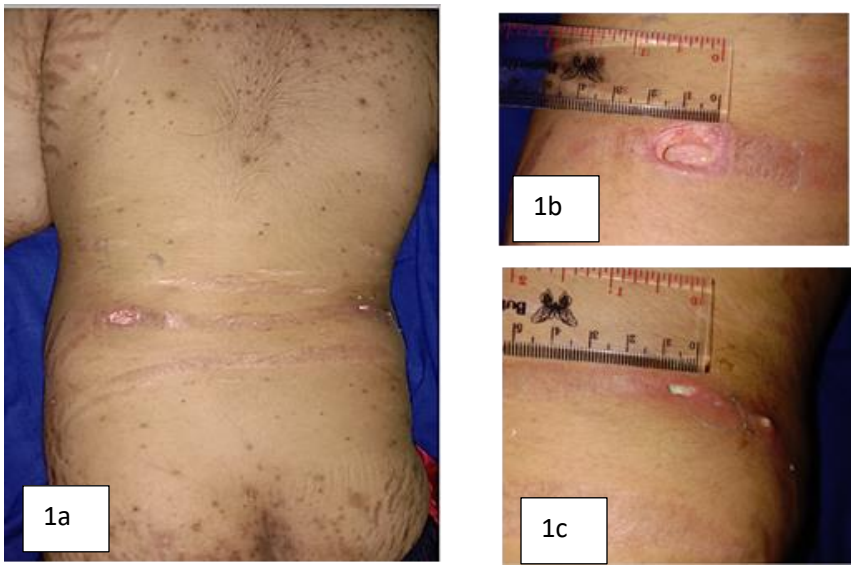


Figure 1. Clinical features patient at baseline. A) Striae and 3 ulcers on the posterior trunk. B) Ulcer measuring 2x1.5x1 cm, erythematous base C) Ulcer measuring 1x1x0.5 cm, necrotic tissue base, filled with pus

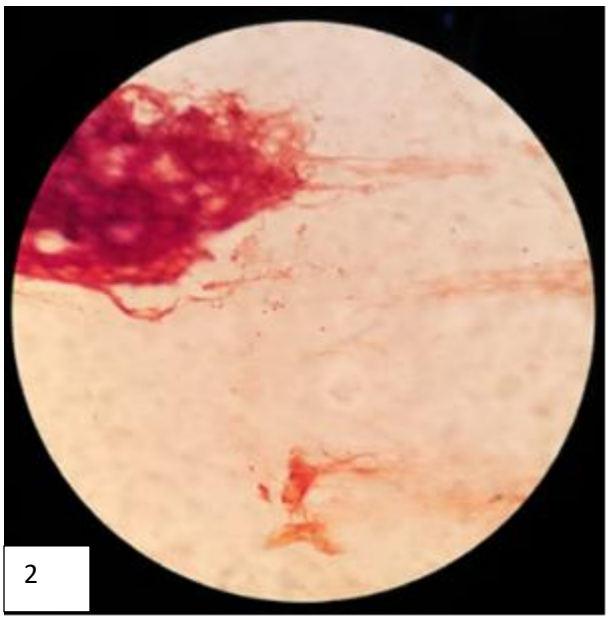


Figure 2. Gram stain examination found gram-positive Staphylococcus bacteria

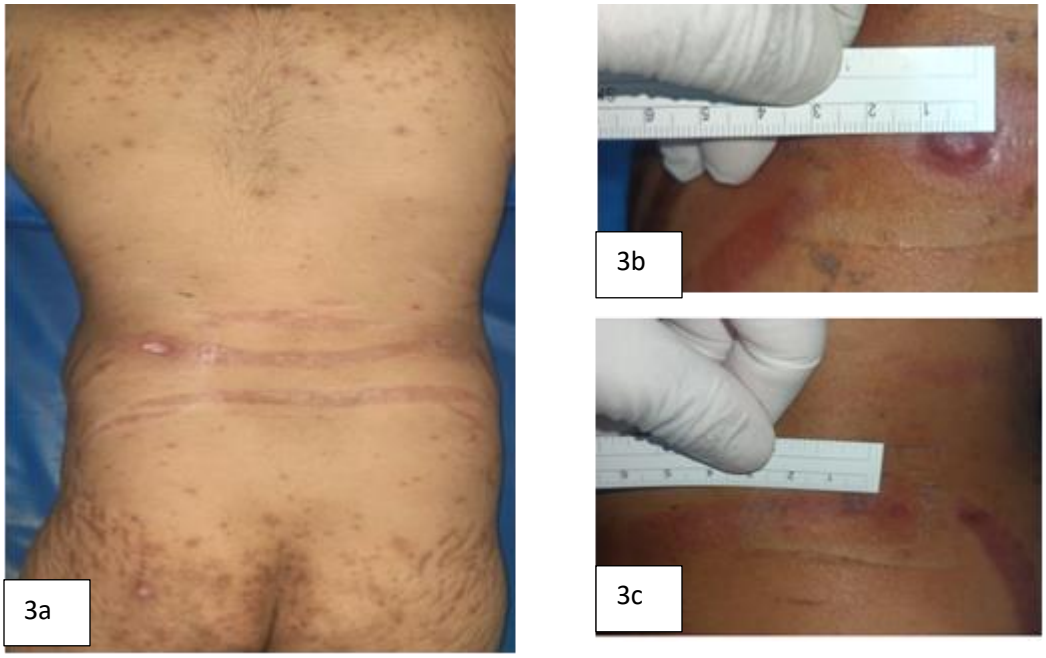


Figure 3. Clinical features patient after 4 weeks of therapy A) Striae and ulcer on the posterior trunk. B) Ulcers got smaller size, measuring 1x0.5x0.2 cm, erythematous base C) Two ulcers healed and the striaewas thinning.

3. Discussion

Nephrotic syndrome (NS) is the most common kidney disease found in children. Corticosteroids as therapy for NS is effective in achieving remission (94%). Management of NS in Indonesia bases on the Nephrology Coordination Unit of the Indonesian Pediatrician Association consensus following the empirical recommendations of the International Study for Kidney Disease in Children (ISKDC). Initial therapy was given prednisone dose of 60 mg/m2/day (2 mg/kg BW/day) for four weeks followed by dose of 40 mg/m2/day (1.5 mg/kg BW/day) every other day for

four weeks. Long-term use of corticosteroids causes the accumulation of high doses of drugs that trigger severe side effects, including cataracts, osteoporosis, hypertension, growth disorders, Cushing's syndrome, and skin disorders such as striae and acne.[11] Patients were diagnosed with NS since 8 months ago, and were given 40 mg methylprednisolone tablets for 2 months. Striae appeared after 2 months of drug administration.

Liern et al reported two groups of patients with nephrotic syndrome aged 2-11 years who were given corticosteroids for 12-24 weeks. Twenty-seven patients in the first group received methylprednisolone doses of 48 mg/m2/day (equivalent to 60 mg/m2 prednisone) for 6 weeks, followed by alternate doses of 33 mg/m2/day (equivalent to 40 mg/m2/day) for 6 weeks, then tapering off was performed until 12 weeks, with alternate doses of 9 mg/m2/day before discontinuation. Twenty-nine patients in the second group received treatment as in the first group until 12 weeks, then continued with placebo until 12 weeks. Side effects of striae were found in 25.92% of patients in the first group, and the second group was 17.24%. Most side effects were found at age > 7 years.

In corticosteroid-induced striae, there is disruption of type I and III collagen synthesis and degradation in the dermis resulting in linear scarring of the epidermis and dermis due to stretching of the skin beyond its elastic limit or known as striae distensae.[5,12,13] Striaedistensae occur due to component dysfunction. The extracellular matrix plays a role in elasticity, namely fibrillin, elastin, and collagen. This results in increased sensitivity to minor trauma and ulceration. Overstretching of the striae due to anasarca and some hormonal factors alters the proportion of cross-linked collagen so that the elastic collagen breaks and causes rupture.[14]

Case reports regarding SD ulceration are limited. In 1954, Lawrence et al reported a case of a patient with tuberculosis, treated with systemic antibiotics and corticosteroids, resulting in ulceration of an abdominal SD. Stroud and Van Dersarl reported two cases of striae ulceration due to the use of topical and systemic corticosteroids. Singh and Talpur reported psoriasis patients treated with systemic corticosteroids showed SD ulceration. Lokhande reported that a patient with nephrotic syndrome was treated with systemic corticosteroids, only the striae were distended with edema occurring.[14,15]

The diagnosis of SD is based on history, physical examination, and investigations. The important history was asked for SD location, color, duration, progression, history of excess weight gain, physical activity, history of long-term use of topical and systemic corticosteroids, history of taking hormonal drugs, and family history of SD.1,3 In this case, the patient was a 15-year-old male, with SR in the abdominal region, posterior trunk, gluteus, femoral, and cruris with a history of NS and long-term corticosteroid treatment since 8 months ago. The clinical manifestations of SR are multiple, symmetrical, well-defined, irregular, red striae that appear 2 months after treatment.

Ulcerations in SD are generally caused by infection with Staphylococcus aureus and Pseudomonas species.8In this case, Gram-staining examination of the posterior trunk ulcer lesion with a microscope magnification of 100x found Gram-positive cocci. The culture results from the pus specimen on the ulcer revealed Staphylococcus aureus.

Topical retinoic acid therapy is a therapy that has shown clinical improvement of SD. Topical retinoic acid 0.05-0.1% showed therapeutic efficacy at SD.[3,7] The mechanism of action of topical retinoic acid is still unclear, but several studies have found a role for retinoic acid in stimulating fibroblasts thereby increasing type I collagen in tissues and inhibiting enzyme activation. degradation of the extracellular matrix so that it can protect the skin from inflammatory mechanisms.[3,7,16] In SD, retinoic acid works to inhibit the inflammatory process by stimulating fibroblasts.3 Treatment can be continued until a satisfactory response has been achieved. Treatment is discontinued if there is no improvement after 3-6 months of treatment. RCT study by Al-Himdani in 2014 on 26 SD patients, 10 patients were given retinoic acid 0, 1% topical and 16 patients were

given the placebo. Clinical improvement occurred in 8 patients who received topical 0.1% retinoic acid and 2 patients did not respond. In the placebo group, only 1 patient experienced clinical improvement.[12] In this case, the patient was treated with topical 0.05% retinoic acid at night. It appears that the striae are thinning after 4 weeks of therapy.

Ulcer management, in general, is to identify and treat infection, maintain cleanliness and hydration of the ulcer base, ulcer protection to prevent secondary infection and dehydration.[17] In this case, systemic antibiotics of intravenous ceftriaxone injection of 2 grams doses for 7 days, accompanied by ulcer treatment with compresses using 1%0 salicylic acid solution for 10 minutes every 12 hours. Solution of 1% salicylic acid functions as an antiseptic, cleans wounds and keeps ulcers moist, vasoconstriction, and reduces inflammation. In addition to compresses, ulcers are treated with modern wound dressings of hydrogel and foam dressing. Combination therapy aims to treat infection, maintain ulcer hydration and ulcer protection so that the healing process occurs better.[17]

The prognosis depends on the type, the patient's condition, the choice of therapy, and the patient's adherence to therapy.[3,4] After 4 weeks of therapy, the ulcer size is reduced and the striae are thinned. Therapy was continued and further observations were made until the ulcer lesions healed completely and the patient's clinical condition improved. Cosmetic problems and the psychological impact of patients due to SD also need further attention.

4. Conclusion

We report a case of ulceration of SD induced by long-term corticosteroid use in child with nephrotic syndrome. Long-term use of corticosteroids for more than 8 weeks needs to be watched out for as a SD trigger. Complications of SD ulceration due to Staphylococcus aureus infection can occur. Systemic therapy of ceftriaxone injection, accompanied by 0.05% retinoic acid topical therapy for SD and ulcer treatment with 1%0 salicylic acid solution compress, hydrogel, and foam dressing showed clinical improvement after 4 weeks of observation. The ulcer size is reduced even though the SD has not been lost. A longer therapy period is needed along with monitoring the patient's quality of life due to the influence of SD to get maximum results. This case is still under observation, the patient's therapy is continuing.

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