Impetigo Herpetiformis: A Case Report

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Abstract

Objective: Impetigo herpetiformis is a rare and potentially life-threatening pustular dermatosis affecting mainly pregnant women. We report here a case of impetigo herpetiformis which occured in twenty-ninth week of pregnancy.

Case: A 32 year old gravida 2, para1 pregnant woman who was referred to our institution because of congestive heart failure, gestational diabetes mellitus and oligohidroamnios in 27th gestational age was hospitalized. Eruptive pustular lesions which appeared in 29th week of the gestation has spread her entire body. Her pustular cultures were negative. A punch skin biopsy from a pustule on the trunk made the diagnosis of impetigo herpetiformis. The patient who developed spontaneous uterine contractions was treated with betamethazone and tocolysis. The patient who did not respond to this treatment was taken to delivery at 30 weeks of gestation.The newborn showed no skin lesions after birth. The skin lesions of the mother improved in the second postpartum week.

Conclusion: The rates of maternal mortality and fetal mortality and morbidity due to placental insufficiency are increased in impetigo herpetiformis. To reduce the mortality and morbidity rates the antenatal management of impetigo herpetiformis should be organized with a multidisciplinary approach.

Keywords: Impetigo herpetiformis, generalized pustular psoriasis.

Impetigo herpetiformis: Bir olgu sunumu

Amaç: İmpetigo herpetiformis gebelerde görülen yaşamı riske edebilen nadir bir püstüler dermatozdur. Bu çalışmada 29.gebelik haftasında ortaya çıkan impetigo herpetiformis olgusu sunulmuştur.

Olgu: 32 yaşında G2P1 27. gebelik haftasında konjestif kalp yetmezliği, gestasyonel diyabetes mellitus, oligohidramnios tanılarıyla kliniğimize refere edilen olgu hospitalize edildi. İzlemde 29. gebelik haftasında ortaya çıkan eritemli püstüler lezyonlar tüm vücuda yayıldı. Püstüllerden alınan örneklerde bakteriyolojik üreme olmadı, histolojik tanı impetigo herpetiformis olarak konuldu. Spontan uterin kontraksiyonları başlayan olguya Betametazon ve tokoliz tedavisine başlandı. Tokolize yanıt vermeyen olgu 30.gebelik haftasında doğurtuldu. Fetal impetigo görülmedi. Cilt lezyonları postpartum 2 hafta içerisinde hızla iyileşti.

Sonuç: İmpetigo herpetiformis olgularında maternal mortalite, ayrıca fetoplasental yetmezlik nedeniyle fetal mortalite ve morbidite oranları artmıştır. Bu oranları azaltabilmek için antenatal izlem protokollerini multidisipliner bir yaklaşımla düzenlemek gerekmektedir.

Anahtar kelimeler: İmpetigo herpetiformis, jeneralize püstüler psoriazis.

Background

Impetigo herpetiformis is a rare and potentially life-threatening pustular dermatosis affecting mainly pregnant women. The disease was triggered by hormonal disorder in pregnancy.[1] It has various clinic and histological similarities to the generalized pustular psoriasis. Impetigo herpetiformis is considered as variant of pustular psoriasis that appears in pregnancy.[2]

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In the literature more than 100 cases were described.[1] Fetal mortality and morbidity risks are high because of maternal mortality and placental deficiency.[3] This case report was presented in order to highlight the importance of impetigo herpetiformis in maternal and fetal, mortality and morbidity. Although it was seen among 6-33 age group, it was also reported for menopause group.[2]

Case

Mrs. AT is 32-year-old. In 27th gestational week, she was referred to our clinic with 2 para 1 congestive heart failure, oligohydramnios diagnosis. She had diagnosed mitral stenosis, and tricuspid failure and used digoxin. The patient delivered a spontaneous vaginal unvivid birth 1 year earlier when an in utero fetal loss has been developed in her first pregnancy. She is hospitalized once she has atrial fibrillation and gestational diabetes on 27th week of current pregnancy.

In ultrasonographic monitoring, fetal biometry was consistent with latest monitoring. In Doppler measurement, dicrotic notch was monitored in bilateral arteries, while umbilical artery and middle cerebral artery were normal. The treatment arranged by regular insulin, digoxin, diltizem, acetyl salicylic acid, low molecule weight heparin.

On 29th gestational week, skin lesions appeared in the pubical zone, with itchy, eritematous (Figure 1). The periphery pustules were spread to all over the body (Figure 2). Oral mucosa and soft palate 2-3 mm eritemous papules were developed. The hair with hair, plamar-plantar surfaces and fingernails were intact

Laboratory results, leukocyte 14000/mm^3, erythrocyte sedimentation rate (ESR): 29 mm/h, serum calcium: 7.3 mg/dl, serum phosphate: 2.5 mg/dl, albumin: 2.41 gr/ml. Liver, kidney function tests and parathyroid hormone levels were normal.

There was no bacteriologic reproduction in the specimens acquired from pustule and in blood culture. The histological examination of biopsy material taken from the lesion displayed that multilocular spngy intraepidermal pustules, acanthosis, parakeratosis (Figure 3).

The case was diagnosed Impetifo herepiformis based on the clinical and histopathological features. As a result of the dermatological consulta-

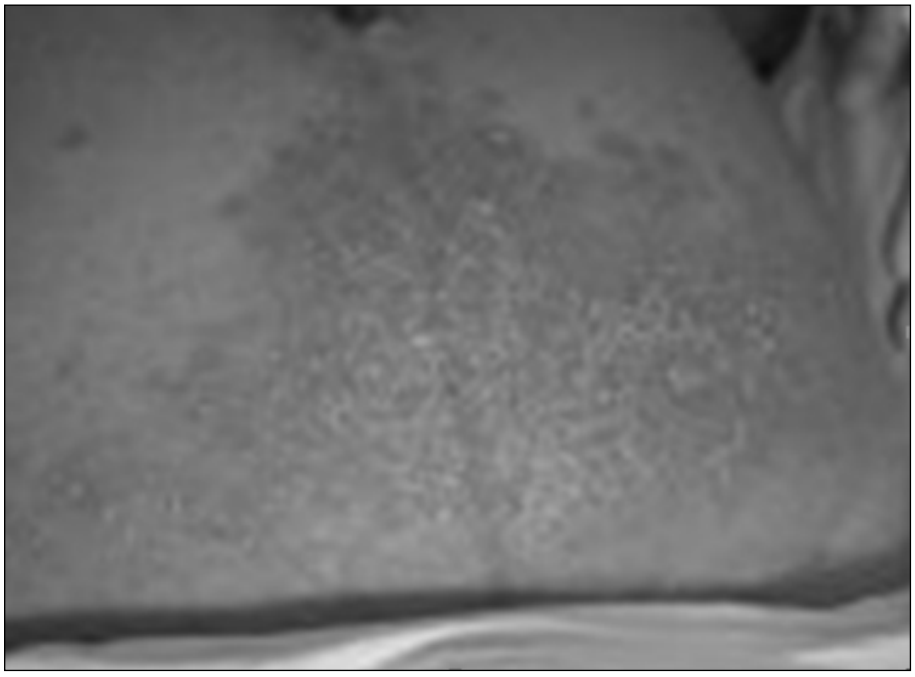

Figure 1. Eritemous dermal lesions.

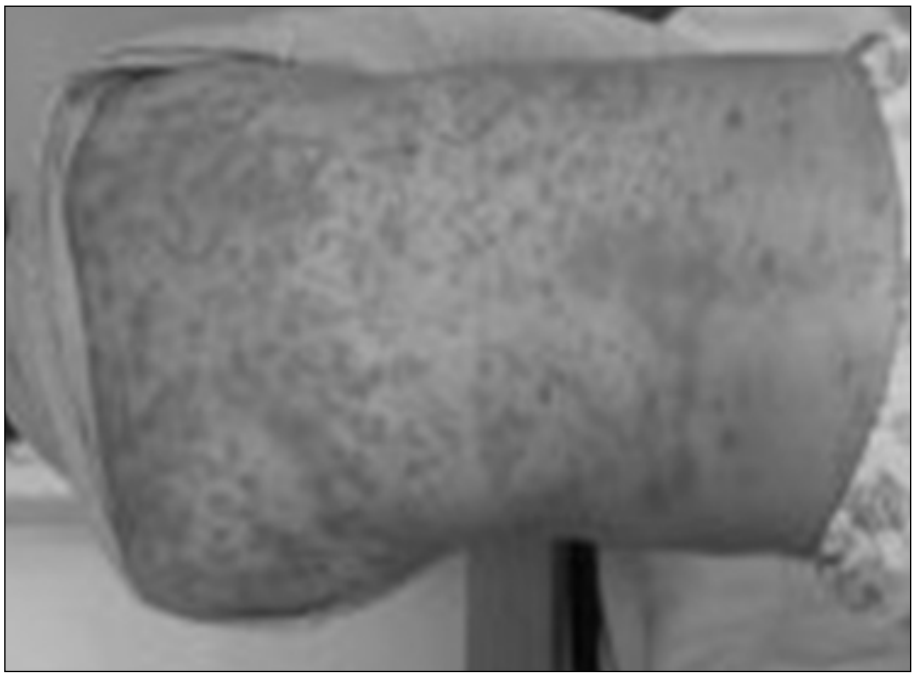

Figure 2. Pustular lesions.

tion, local betamethazon administration, 3 times per day, (Betnovate cream, Glaxo Welcome Medical Industries A.Ş, Istanbul, Turkey) has begun.

On 29th gestational week, in order to stimulate lung maturation and contribute to the treatment of the lesions, betamethazon 12 mg/24 h (Celestone chronodose; Schering Plugh Medical Products AS, Istanbul, Turkey).

In 29th gestational week, the case that developed premature membrane rupture, delivered a male infant, 1450 g, 42 cm, 12 hours later by a vaginal modality. According to Lubchenco–LO maturation curve, the weight was 50 percantile, the length 75 percantile.[4] The skin lesions were ameliorated within 2 weeks post partum.

Discussion

Impetigo herpetiformis is a dermatosis characterized by sterile pustules that occur in pregnancy. The disease appears in pregnancy. Some indicates that it has an etiological relationship with the pregnancy.[5] Even though there isn't any strict pathological mechanism, the use hypocalcaemia, hypoparathyroid, infection, oral contraceptive and stress are known to facilitate the development of impetigo herpetiformis.[5,6]

It appears in second and third trimesters but mostly occurs in third trimester. The frequency is variable and the symptoms don't completely ameliorate. In following pregnancies, it tends to appear earlier and acute.[7] In our study, lesions appeared in third trimester.

It may be life-threatening for both mother and fetus. The dermal lesions are intensified sepsis risk, while placental defect and decreasing intervillous circulation may cause fetal morbidity and mortality.[3,5]

Lesions begin with symmetrical eritemous plaques in convolution of the skin. Sterile pustules appear in the periphery of the plaques. The pustules amalgamate and expand to the periphery when the center opens and crust. Oral and pharynx lesions may be developed. General situation worsens and systemic symptoms including lassitude, headache, fever, palpitation, nausea, diarrhea, tetani as well as eruption.[7] In our case, lesions started from pubical zone and expanded to

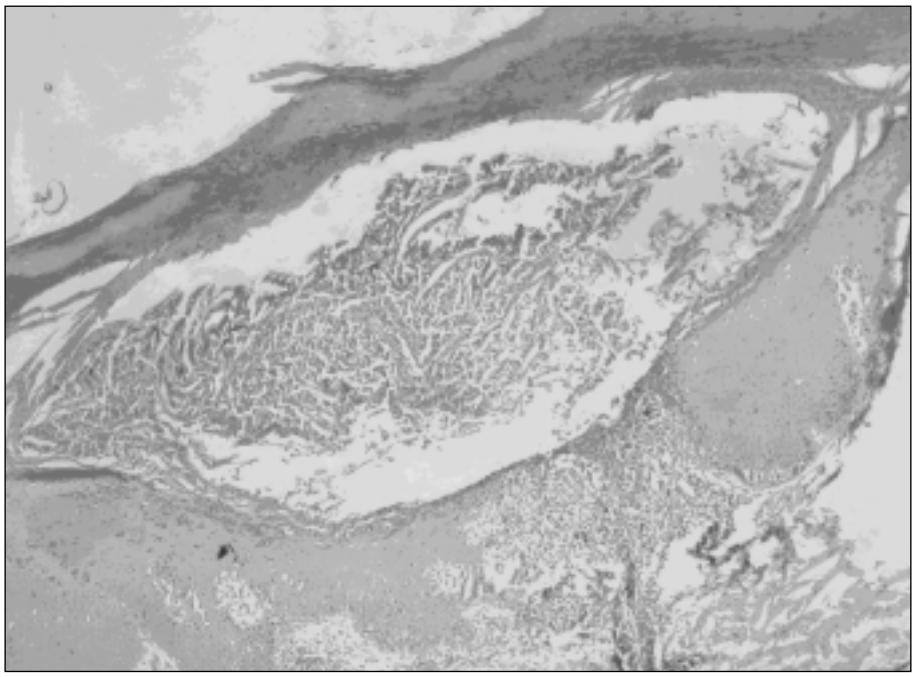

Figure 3. Epidermis multiocular spongious intraepithelial pustular, acanthosis, parakeratosis structure.

the body. The lesions are seen in oral mucosa and soft palate tissue. In lesion samples, no bacteriological reproduction has been observed. Further systemic findings haven't been diagnosed in our study. The lesions are ameliorated in postpartum period, leaving a postinflammatory pigmentation, in a couple of weeks.[7] In our case study, the lesions were ameliorated within 2 weeks.

Leukocytosis, hypocalcaemia, hyperphosphatemia, hypoproteinemia, ESR increase are most frequent laboratory findings. It is noted that hypoalbuminemia develops as a result of albumin loss from large exudative zones, and hypocalcaemia appears as a secondary effect of the hypoalbuminemia.[7] In the differential diagnosis of the cases, subcorneal pustular dermatosis may be considered. But, the general situation doesn't corrupt in subcorneal pustular dermatosis.1 In our case, leukocytosis, hypocalcaemia, hypoalbuminemia, ERT increases were diagnosed. Parathormone levels were normal.

In impetigo herpetiformis cases, as a histopathological symptom in a lesion, polymorphic leukocyte accumulation at epidermis spongious zones, are characteristic, and they are accepted similar to the spongioform pustules of Kogoj specific to the psoriasis. Extension and parakeratosis are frequently monitored.[3] In our case, prolongation in spongioform pustular papillary and parakeratosis have been diagnosed.

Most important complication of the disease is placental defect, and intrauterine fetal mortality.[6] In our case, while impetigo herpetiformis related placental defect develop as well as maternal heart failure related intrauterine maturation latency, gestational diabetes ensured that intrauterine maturation develops in normal limits.

For treatment in less severe cases, topical steroid applies, besides, in severe cases systemic steroid is in use.[5,8] For secondary infections, antibiotics, fluid, electrolyte and calcium replaced. In hyperparathyroidism cases, phosphate limitation, calciferol and dehydrocolecalciferol applications are suggested. For treatment resistant cases, cyclosporine and phototherapy treatments are in use.[5] In our case, topical steroid wasn't administrated since systemic symptoms haven't been diagnosed. In addition, systemic parenteral corticos-

teroid was administrated in order to stimulate lung maturation. In post partum period, systemic corticosteroid administration wasn't continued since a diminution was expected in lesions. Parenteral calcium replacement was used.

Since it is similar in clinic and histopathological regard, impetigo herpetiformis may be a variant of the generalized pustular psoriasis.[3,8] Impetigo herpetiformis is seen in the persons that don't have psoriasis in their personal or family history, and it is induced after the birth.[5,9] It is known that it repeats in successful gestations, more severe and earlier.[9] In our case, it appears the first time in second gestation. The psoriasis wasn't reported in personal or family history.

The symptoms of the psoriatic cases are reduced at 30-65% when the patients are conceived. The symptoms worsen only in 10-20% of the cases.[9] The amelioration occurs generally in first trimester. The gestational response remains the same in following gestations. The amelioration in psoriasis table for the pregnant women is related to the immune system down regulation.[10,11] 87.7% of the cases are involved after the pregnancy. In our case, lesions are rapidly induced after the gestation. Our case was diagnosed in clinic development as impetigo herpetiformis as it was similar in regard of morphologically and histological appearance. This rare entity is important for both maternal and fetus mortality and morbidity.

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