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położnictwo

Toxic epidermal necrolysis complicating antibiotic treatment of puerperal endometritis: a case report

Toksyczna nekroliza naskórka wikłająca antybiotykoterapię połogowego zapalenia endometrium: opis przypadku

Ondrej Simetka[1,2], Igor Michalec[1,2], Zdenka Nemeckova Crkvenjas[3], Hana Klosova[3], Lenka Janackova[1], Jaroslav Klat[1,2]

[1] Department of Obstetrics and Gynecology, University Hospital Ostrava, Czech Republic
[2] Department of Surgical Studies, University of Ostrava, Czech Republic
[3] Burn Unit, University Hospital Ostrava, Czech Republic

Abstract

The aim of the study is to describe a case report of Lyell syndrome (toxic epidermal necrolysis) involving 63% of body surface which has been associated with antibiotic therapy of mild peurperal endometritis in woman 3 weeks postpartum.

Lyell syndrome is a severe life-threatening condition developing due to idiosyncrazy (alergic reaction type IV), most commonly after administration of drugs. Incidence quoted in literature is around 1:1-2000000. Illness severity can be assessed using a SCORTEN scoring system, which predicts patient mortality based on seven independent factors. Lyell syndrome is a very rare but potentially lethal complication of antibiotic treatment.

Key words: **Lyell syndrome** / **puerperium** / **exanthema** /

Address for correspondence:
Igor Michalec
Porodnicko-gynekologická klinika
FN Ostrava, 17. listopadu 1790
Ostrava, Czech Republic
e-mail: igor.michalec@fno.cz
mobile: + 420 597 371 802

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Streszczenie

Celem pracy jest opis przypadku zespołu Lyell'a (toksyczna nekroliza naskórka) obejmująca 63% powierzchni ciała, który był związany z antybiotykoterapią łagodnego zapalenia endometrium u kobiety 3 tygodnie po porodzie.

Zespół Lyell'a jest ciężkim zagrażającym życiu stanem wywołanym idiosynkrazją (reakcja alergiczna typu IV), najczęściej po podaniu leków. Częstość występowania szacowana jest na około 1:1-2000000. Ciężkość choroby może być określona przy pomocy systemu punktowego SCORTEN, który przewiduje ryzyko zgonu pacjenta w oparciu o siedem niezależnych czynników. Zespół Lyell'a jest bardzo rzadkim ale potencjalnie śmiertelnym powikłaniem antybiotykoterapii.

Słowa kluczowe: **zespół Lyell'a** / **połóg** / **rumień** /

Introduction

Lyell syndrome, or toxic epidermal necrolysis (TEN),is a rare and life-threatening condition with extensive skin and mucous membrane involvement, which develops due to idiosyncrasy (allergic reaction type IV), most commonly after drug administration [1]. The annual incidence is between 1.5–2 cases per million and the mortality rate around 30% [2,3]. The most common triggers of this autoimmune reaction are antibiotics (aminopenicillins, sulfonamides, cephalosporin, and quinolones), and anti-inflammatory and anti-epileptic drugs. Rarely, the condition is activated by infection (e.g., herpes virus, *Mycoplasma pneumoniae*) [4].

Materials and methods

In the present study, we described a case report of Lyell syndrome in a woman 3 weeks postpartum.

Results

A 35-year-old secundipara developed fever and hypogastric pain 14 days after caesarean section, and was admitted to the hospital 18 days after delivery. Antibiotic therapy (clindamycin and gentamycin) was commenced for endometritis; however, due to persistent laboratory and local clinical findings of infection, the therapy was changed to cefotaxime on day 26 after delivery. This change was followed by onset of generalized erythematous exanthema within 24 hours. Further complications – including hyperpyrexia (39.6°C), generalized exanthema, and small pulmonary embolism – resulted in transfer of the patient to the perinatal centre on day 27. Shortly after admission, generalized macular exanthema with petechiae developed, and intravenous antibiotics (amoxicillin/acidum clavulanicum) and corticoids (methylprednisolone) were administered for suspected toxic shock syndrome. Her status rapidly deteriorated further, with development of tachypnea and a requirement for circulatory system support, accompanied by progression of skin lesions to haemorrhagic blisters (Figures 1 and 2).

The patient was diagnosed with Lyell syndrome involving 63% of the skin surface, with 53% being grade I and 10% grade II (haemorrhagic blisters). The patient was transferred to the burn unit ICU, where she stayed for 10 days and continued to require circulatory system support (dobutamine).

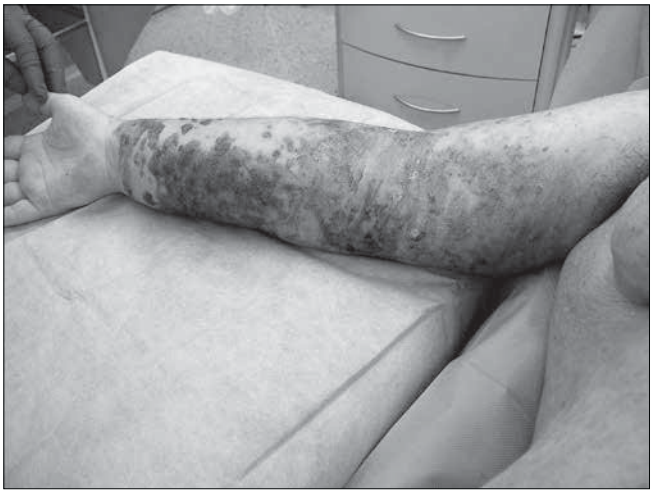

Figure 1. Lyell syndrome in patient with postpartum endometritis; hemorrhagic blisters.

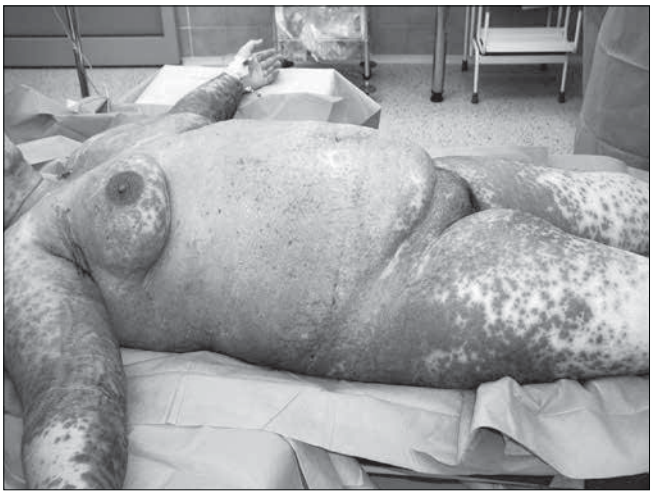

Figure 2. Lyell syndrome in patient with postpartum endometritis; skin lesions covering over 60% of body surface.

After three days in the ICU, pulmonary oedema developed, which required oxygen and aggressive oncodiuretic therapy for 72 hours. Regular dressings were applied, and meropenem, antimycotics, and other supportive treatment were given.The patient's status slowly improved, and she was discharged on day 49 after delivery in good clinical condition.

Discussion

TEN usually starts with flu-like symptoms (fever, rhinitis, conjunctivitis, or dysuria) followed by development of a rash that extends into confluent erythema, blister appearance, and epidermis exfoliation. Skin lesions are caused by eosinophilic necrosis of epidermis, with blisters developing after keratinocyte apoptosis. In more than 90% of patients, TEN also involves the mucosa of the mouth, genitals, and eyes. Mucous lesions are very painful and look like ulcerations or haemorrhagic crusts. The general condition usually deteriorates and resembles burn symptoms (fever, fatigue, electrolyte imbalance, and multiorgan failure).

Many patient recover with permanent damage, such as skin hyper- or hypopigmentation, eye complications, or mucosal lesions [5].

Illness severity can be assessed using a SCORTEN scoring system, which predicts patient mortality based on seven independent factors that are scored as 0 or 1 within the first 24 hours of admission [6]. Higher scores indicate higher mortality risk, with SCORTEN scores of 0–1, 2, 3, 4, and 5 associated with mortality risks of 3.2%,12.1%,35.3%, 58.3% risk, and 90%, respectively [7, 8]. TEN treatment includes early withdrawal of culprit drugs, complex supportive management in a burn or intensive care unit, nutritional support, and wound care. Use of corticoids is associated with higher incidence of wound infection and prolonged healing [9]. A second-line treatment is intravenous immunoglobulin (IVIG), which shows promising effects in uncontrolled trials and case reports [10, 11, 12] however, a randomized control trial remains needed to determine its efficacy. Third-line treatments include cyclosporine, cyclophosphamide, plasmapheresis, pentoxifylline, N-acetylcysteine, ulinastatin, infliximab, and/or granulocyte colony-stimulating factors (in cases with TEN-associated leukopenia) [13].

Conclusion

TEN is a very rare but potentially lethal complication of antibiotic treatment. General knowledge about this disease is vital for saving the patient.

Authors' contribution:

1. Ondrej Simetka – main author, concept, final draft.
2. Igor Michalec – clinical management, literature research, corresponding author.
3. Zdenka Nemeckova Crkvenjas – clinical management, acquisition of data, draft co-author, revised article critically.
4. Hana Klosova – clinical management, acquisition of data, draft co-author.
5. Lenka Janackova – literature research.
6. Jaroslav Klat – clinical management, revised article critically.

Authors' statement

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