

# Steven Johnson syndrome in pregnancy

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## Abstract

Cutaneous eruption is one of the common forms of adverse drug reaction manifestations. Stevens– Johnson syndrome (SJS) is one such manifestation that represents severe form of cutaneous eruptions. It is a life threatening allergic reaction to medications affecting the skin and mucous membranes, characterized by severe purulent conjunctivitis, severe stomatitis with extensive mucosal necrosis, and purpuric macules. The common culprits are antimicrobials like sulphonamide followed by nonsteroidal anti-inflammatory drugs (NSAIDs), anticonvulsant drugs, and anti-gout drugs. A 30 year old G4P1L1A2, 8 weeks gestation, reported to us with features of SJS after receiving Tablet containing Amoxycillin and clavulonic acid. She was put on topical steroids, analgesics and antacids. She delivered at 36 weeks of gestation by LSCS in view of fetal distress, delivering 2.7kg healthy female child. Both mother and baby were discharged in a good condition. Case managed properly during acute phase. Perinatal outcome was good in this case.

**Key Word:** Steven Johnson syndrome, pregnancy.

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## INTRODUCTION

Stevens-Johnson syndrome (SJS) was first described in 1922, as an acute mucocutaneous syndrome. It is thought to be a hypersensitivity reaction to certain drugs and vaccines affecting the skin and the mucous membranes. Altered drug metabolism in some patients causes formation of reactive metabolites that bind to and alter cell proteins, triggering a T-cell-mediated cytotoxic reaction to drug antigens in keratinocytes. The pathogenesis of SJS/TEN (Toxic epidermal necrolysis) is not fully understood but is believed to be immune-mediated, as re-challenging an individual with the same drug can result in rapid recurrence of SJS/TEN. It is the most severe form of erythema multiforme known as erythemamultiforme major. It is a rare condition with a

reported incidence of one case per million people per year. Such a rare case of SJS in pregnancy is reported.

## CASE REPORT

A 30 year old G4P1L1A2, 8 weeks of gestation consulted a private practitioner 4 days back for high grade fever with common cold for which she was put on tablet containing Amoxicillin and clavulonic acid. Patient was a known case of epilepsy and on treatment with tablet levipril since 8 years. Fever subsided after treatment. After 4 days, she started having rashes and blisters all over the body. She presented to us in emergency with features suggestive of SJS:

- i) Oral cavity: Erosions on bilateral buccal mucosa, whitish coating on tongue.
- ii) Lips: Crusting over both lips.
- iii) Multiple erosions with crusting over face, trunk, bilateral lower limb.
- iv) Genitalia: Multiple erosions with crusting over pubic region.
- v) Peeling of skin over back.

On examination patient was febrile (temperature – 100 F), tachycardia was present (110/min), BP was normal, she was pale. There was no icterus lymphadenopathy or edema. Cardiorespiratory examination was normal. Internal examination revealed a 8 weeks size uterus

corresponding to USG findings. There was no history of any allergy to drugs and food products.

A diagnosis of Steven Johnson Syndrome was made and she was kept nil by mouth and intravenous fluids were started. Oral toileting was carried out every 2 hourly, topical steroids, analgesics and antacids were started. Investigations were within normal limits and normal kidney and liver function tests. Swabs from the mouth were sterile.

The potential precipitating factor could be antibiotics, which was aggravated in the presence of pregnancy. Patient was discharged after 7 days with topical steroids and mouth washes and asked to follow up. All the lesions were healed and there was hyperpigmentation over the skin. The antenatal period was uneventful till 34 weeks of gestation when she admitted with us with threatened preterm and diagnosed to have gestational diabetes and gestational hypertension. She was started with anti hypertensive drugs and diabetic diet. Patient was discharged after 10 days. Patient was re admitted at 36 weeks in labour and delivered with LSCS in view of previous LSCS with fetal distress. Mother and baby were well on discharge and followed up for one year both mother and baby were good and all lesions were subsided and only hyperpigmentation was left all over the skin.

## DISCUSSION

Cutaneous adverse drug reactions (CADRs) are very commonly encountered in the dermatology department. SJS or toxic epidermal necrolysis (TEN) is one of the dermatologic conditions that can be potentially fatal.<sup>1</sup> Although the exact etiology of SJS/TEN is not fully understood, it is believed to be an immune mediated hypersensitivity reaction in which cytotoxic T lymphocytes play a role in the pathogenesis.<sup>2</sup> Patel *et al* reported that penicillins are one of the antimicrobials frequently causing severe CADRs in the Indian population.<sup>3</sup> Till date, few cases of amoxicillin-induced SJS/TEN have been reported.<sup>4,5</sup> We came across the SJS caused by amoxicillin clavulanic acid. SJS is marked by the rapid attack of fever, skin lesions and sores on the mucous membranes of eyes, mouth, nasal passage, lips and genitals. Clusters last for about 2-4 weeks. The skin lesions may look like target lesions or bubble like. The diagnosis is often obvious by the appearance of lesions and rapid progression of symptoms. Histologic examination of sloughed skin shows necrotic epithelium, a distinguishing feature. The condition is characterised by severe constitutional disturbance and may result in death from pneumonia, septicaemia, myocarditis or renal failure. Erosive changes may occur in the genitalia. Severe scarring of the genital tract may also occur occasionally, however there is no mention of permanent

damage to the female genital tract.<sup>6</sup> There has been one case report of vaginal stenosis following SJS in pregnancy, which was discovered 6 weeks after cesarean section for breech presentation.<sup>7</sup> However, our patient delivered baby by LSCS and there was no genital problem on follow up. The management includes prompt withdrawal of all potential causative drugs, intravenous fluid replacement. Symptomatic treatment are careful and aseptic handling, maintenance of venous peripheral access distant from affected areas, initiation of oral nutrition by nasogastric tube anticoagulation, prevention of stress ulcer. Topical antiseptics (0.5% silver nitrate or 0.05% chlorhexidine) are used to paint, bathe or dress the patients. New dressings with Apligraf®, Biobrane®, TransCyte® etc are being tried. Corticosteroid use is highly debated.<sup>6</sup> Tegelberg used 400 or 200 mg prednisolone, gradually diminished over a 4 to 6 week period, and observed a single death among eight patients.<sup>8</sup> It's difficult to prevent an initial attack of Stevens-Johnson syndrome because what triggers it is not known. However, if Steven-Johnson syndrome occurred once, which was caused by medication, the drug is to be avoided to prevent another attack. A recurrence is usually more severe than the first episode and, may be fatal.<sup>6</sup> SSSS was one of the most important differential diagnoses in the past, but the incidence is currently very low with 0.09 and 0.13 cases per one million inhabitants per year.<sup>9</sup> HIV infected patients have a higher predisposition to condition because of decreased antioxidant levels owing to infection.<sup>10</sup> Attack of SJS developing in pregnancy can be fatal because immunity is compromised.<sup>6</sup> However, early diagnosis and prompt management saved the mother and the child.

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