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Case Report

Septicaemia in pregnancy: a case report

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ABSTRACT

Necrotizing fasciitis (NF) is a rare, life-threatening surgical infection in pregnancy with high rates of morbidity and mortality. It is a severe, potentially fatal infectious disease which rapidly extends from the subcutaneous tissue along the superficial and deep fascia causing vascular occlusion, ischemia, and necrosis of tissues. A 30-year-old gravida 2 para 1 living 1 woman, at 32 weeks of gestation with previous caesarean section and recently diagnosed diabetes, hypertension was admitted to our hospital with signs and symptoms of severe sepsis with pruritic black lesions over abdomen and perineum. Patient was in a morbid state in our hospital. During clinical examination, fetal heart sound was not localised suggestive of intrauterine fetal demise (IUID), with ulcerative lesions over abdomen and vulva. Patient was immediately taken for surgical intervention and was suggestive of ruptured uterus with extrusion of fetus in abdominal cavity with cellulitis of abdominal and vulva. During initial laboratory examinations, diabetes mellitus was diagnosed. Patient was kept on ventilatory support and was vitals were stabilised. Multidisciplinary therapy with immediate aggressive surgical debridement of necrotic tissues, multiple antibiotics, and intensive care monitoring was performed successfully. The patient's postoperative course was uncomplicated and skin defect healed by second intention of healing. The following case emphasized the potential immunosuppressive role of pregnancy state in conjunction with diabetes mellitus in the development of severe necrotizing soft tissue infections.

Keywords: Septicaemia, IUID, Diabetes, NF

INTRODUCTION

Necrotizing fasciitis (NF) and associated septicemia is a life-threatening invasive soft tissue infection which is characterized by widespread necrosis of subcutaneous tissue, superficial fascia, and other adjacent tissue.¹ It is a surgical emergency with high mortality rate among patients and primarily involves the subcutaneous tissue and rapidly extends along superficial fascia planes.² Management of NF is aggressive surgical debridement of necrotic tissues, broad spectrum antibiotics, and intensive supportive care.³ NF occurs mainly in patients with predisposing factors such as diabetes mellitus, obesity, peripheral vascular disease, and immune system impairment.⁴

Sepsis, is a life-threatening organ dysfunction resulting from a dysregulated host response to infection, remains a major cause for the admission of pregnant women to the intensive care unit and is one of the leading causes of maternal morbidity and mortality.⁵ The obstetric causes include uterine infection, septic abortion, and wound infection.⁶ The non-obstetric causes include pyelonephritis and pneumonia.⁷ Maternal sepsis may also be from obstetrical critical illness, such as obstetric severe hemorrhage, obstetric (amniotic fluid/pulmonary) embolism, acute fatty liver of pregnancy, and congestive heart failure, cardiopulmonary arrest, and major trauma.² The most commonly reported pathogens in maternal sepsis include *Escherichia coli*, *Streptococcus*, *Staphylococcus*, and other gram-negative bacteria.⁸ Maternal sepsis may cause intrauterine infection, which results in (1) preterm

premature rupture of membranes or preterm labor or birth, (2) cerebral white matter damage or cerebral palsy or neurodevelopmental delay, (3) stillbirth, (4) early- or late-onset sepsis, and (5) perinatal death.⁹

Pregnancy is responsible for an immunosuppressive state, which may contribute to the development of severe necrotic soft tissue infections.¹⁰ NF in pregnancy is rare and usually is characterized by acute onset and rapid clinical progression involving the vulva, perineum, lower extremities, and abdominal wall of the pregnant or postpartum women.¹¹ Hereby we are reporting a rare case of 32 weeks pregnancy with widespread NF who presented with IUD and ruptured uterus. Patient was managed by obstetric hysterectomy with a good clinical outcome.

CASE REPORT

A 30-year-old polio afflicted female patient who has been married for 7 years, presenting an obstetric history of gravida 2 para 1 living 1, was referred to our department of obstetrics and gynaecology, grant government medical college and JJ group of hospitals, Mumbai. She had undergone a previous lower segment caesarean section (LSCS) at a primary health care centre at Dhule and was registered for her second pregnancy as well. She was at 8 months of gestational age when she presented with complaints of altered sensorium present for 1 day, fever spikes for 3 days and pain and swelling over abdominal wall for 15 days. She also had a history of blackish discoloration of vulval skin associated with itching since a month. The private hospital at Dhule thereby her to our tertiary care centre in view of cellulitis with septicaemia and acute kidney injury (AKI) with hypertension.

Menstrual history

Her last menstrual period (LMP) date was unclear, BD was therefore indistinct. BS was 32 weeks (by scan of 14 weeks)

Obstetric history

Gravida 2 para 1 living 1, G1-3 years/F/LSCS done i/v/o Cephalopelvic disproportion. Patient had past history of pre-eclampsia in previous pregnancy, P/H-patient has paralytic left leg since childhood secondary to poliomyelitis.

On general examination patient was drowsy with poor GCS-responding to deep stimuli, afebrile, pulse-112/min, BP of 140/90 mmHg, SpO₂ of 99% on 4 litres of O₂, HGT-215, with pallor, generalised oedema, urine albumin-traces, DTR-absent, urine output 200 ml, clear. Patient did not have any petechiae or purpura.

Systemic examination: Cardiovascular/ respiratory system-Normal, per abdomen (On obstetric examination)-uterus-gravid state corresponded to 30 weeks of gestation,

FHS was not localised, scar of previous caesarean section was present.

Local examination: A 10×10 CM BLACKISH patch was present over right lower abdomen with local rise of temperature, foul smelling purulent discharge and tenderness (Figure 1).



Figure 1: Abdominal wall ulcer.

Per speculum: no leak demonstrable, per vaginum OS-8 cm dilated and 50-60% effaced, membranes absent station high up and uterine rupture.

Deep ulcerative lesion of 15×10×10 cm was seen over right labia majora reaching up to ischial rectal fossa and right groin with irregular edge with foul smelling purulent discharge, oedema, redness and ulceration. Reddish patches were present over bilateral thighs (Figure 2).



Figure 2: Valvular ulcer.

Investigations

Immediate ultrasonography was suggestive of single intrauterine fetal death of MGA 30.6 weeks with anhydramnios, the upper uterine segment was empty and the fetus was seen in the lower uterine segment and appeared clumped. USG local was suggestive of multiple small patchy hypoechoic areas with adjacent fat stranding

suggestive of anterior abdominal wall cellulitis. Routine blood investigations were done (Table 1).

Table 1: Blood investigation.

| Parameters | Values |
|------------------|-----------------------|
| Hb | 10.9 g% |
| TLC | 22.11 mm ³ |
| Platelets | 46000 mm ³ |
| T Bili | 0.6 mg% |
| SGOT/SGPT | 40.5/22.8 U/l |
| Serum creatinine | 1.8 |
| Urea | 186 |
| Na+ | 127 |
| K+ | 4.42 |
| PT | 21.7 |
| INR | 2.14 |
| Hbsag, HCV, HIV | Non-reactive |

Treatment

In view of poor GCS, patient was put on ventilator support and started on injection meropenem. Antihypertensives were given. 4-pint RDPs and 2-pint FFP were administered. Patient was thereafter taken for emergency laparotomy in view of rupture uterus. 1-pint whole blood and 3-pint FFP were given intraoperatively (Figure 3).

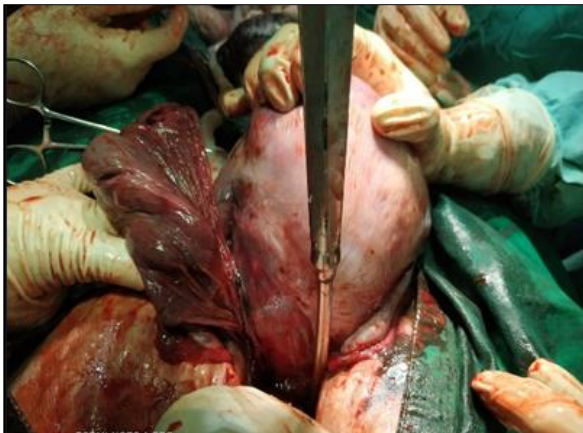


Figure 3: Rupture of uterus, placenta extruding through uterus.

Intraoperative findings

The 10x8 cm of subcutaneous blackish lesion with multiple discharging sinus with purulent discharge. *In situ*- findings- uterus empty; fetus in left broad ligament with rupture on uterine left lateral wall (Figure 3 and 4) male-MSB of 1.9 kg removed with placenta of 200 gm. Intra-operative decision for obstetric hysterectomy was made. Bilateral internal iliac artery ligation was done. Surgical team was called for debridement of ulcer. Abdominal wall ulcer was debrided and pus pockets were removed till healthy tissue was seen. Similarly perineal ulcer debridement was done, involving right sided labia majora,

perianal region and upper aspect of right thigh. Right perianal ulcer was noted communicating with abdominal ulcer along right labia majora. (Figure 5 and 6) debridement was done and pus pockets were drained (Figure 7). Abdominal wall closure done (Figure 8).



Figure 4: Rupture of uterus on left lateral wall.



Figure 5: Perineal ulcer communicating with abdominal wall ulcer.

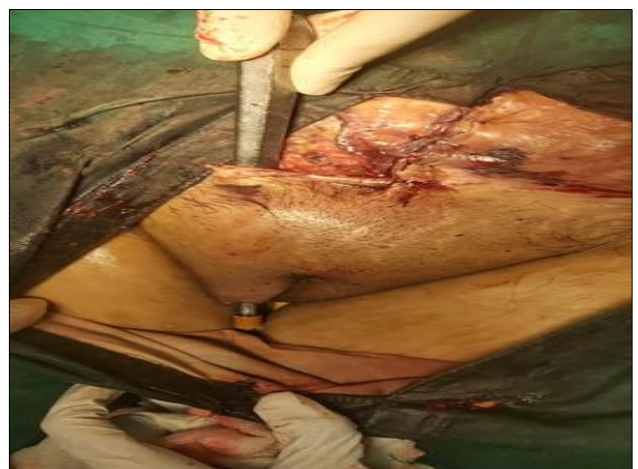


Figure 6: Communication with abdominal wall and vulval ulcer shown with the help of suction cannula.



Figure 7: Perineal ulcer after debridement.



Figure 10: Healthy granulation tissue seen after 26 days post-op.



Figure 8: Ulcer after debridement of abdominal wall.



Figure 11: Healing on day of discharge (40 days post-op).

Post-operative care

Patient was shifted to CCU in post operative period. Patient was started on higher antibiotics (injection meropenem, amikacin, piptaz) 4 pints of blood, 4 RDPs and 19 FFPs were given in post-operative period-during the entire hospital stay.

Patient was extubated 4 days later, was started on injection insulin, OHAs in view of newly diagnosed diabetes and antihypertensives for chronic hypertension. Daily dressing of ulcer done till healthy granulation tissue 26. (Figure 9 and 10). The patient showed an outstanding improvement and overcame her condition after intensive care support. She was shifted toward after 15 days and discharged from hospital after 40 days (Figure 11).



Figure 9: Healing after 20 days post-op.

DISCUSSION

NF is a life-threatening surgical emergency.^{1,12} It is a severe, potentially fatal infectious disease which rapidly extends from the subcutaneous tissue along the superficial and deep fascia causing vascular occlusion, ischemia, and necrosis of tissues.¹² Bacterial endotoxins with the release of cytokines are mediators of rapid tissue destruction and have a crucial role in progression of the disease.^{1,13}

Predisposing factors for the development of NF are well documented, diabetes mellitus and pregnancy represent two main poor prognostic factors.¹⁰ In our case, diabetes mellitus was diagnosed incidentally at the time of hospital admission. Pregnancy itself represents an additional risk factor due to suppression of immune system during the second and third trimester and in postpartum period.¹⁴

The clinical presentation of NF is often characteristic, including high fever with chills, signs of systemic toxicity, and severe pain. Without prompt and urgent therapeutic intervention, it may rapidly lead to septic shock syndrome with cyanosis, hypotension and tachycardia, altered level of consciousness, multiorgan failure, and death.¹³ The inflamed skin appears erythematous with edema and blistering but its involvement is smaller than the extent of necrosis of the underlying subcutaneous tissue and fascia, making the clinical distinction between simple cellulitis and NF extremely difficult.¹²⁻¹⁴ On admission, our patient

had local inflammatory skin changes, clinical signs of sepsis and an abdominal ulcer communicating with a perineal ulcer.

The definitive diagnosis of NF is made after surgical debridement with microbiologic and histological examination of infected tissues.^{1,4,13,15} The pathophysiology core issue is a multifaceted host response to an infecting pathogen that may be significantly amplified by endogenous factors. Sepsis is now recognized to involve early activation of both pro- and anti-inflammatory responses, along with major modifications in pathways such as cardiovascular, neuronal, autonomic, hormonal, bioenergetic, metabolic, and coagulation, all of which have prognostic significance.¹⁶ Patients with sepsis can have clinical presentations in various systems, including respiratory, cardiovascular, hepatic, and gastrointestinal, renal, hematological, endocrinological, and central nervous systems.¹⁶

To recognize maternal sepsis early, many early warning scores have been developed. The sensitivity and specificity of sepsis screening tools with the highest to lowest sensitivity were systemic inflammatory response syndrome, maternal early warning, and quick sequential organ failure assessment (qSOFA) criteria, and the highest to lowest specificity were qSOFA, maternal early warning, and systemic inflammatory response syndrome.¹⁷

Risk factors for sepsis identified from the women who died included factors like obesity, impaired glucose tolerance/diabetes, impaired immunity/immunosuppressant medication, anaemia, vaginal discharge, history of pelvic infection, history of group B *Streptococcal* infection, amniocentesis and other invasive procedures like cervical cerclage, prolonged spontaneous rupture of membranes, GAS infection in close contacts/family members and urinary tract infection and chorioamnionitis.¹⁸

Prognosis of patients with NF depends on early diagnosis and aggressive multidisciplinary management such as deep surgical debridement of all necrotic tissues, intravenous antibiotics, fluids and electrolytes management, appropriate analgesia, and intensive care support. Daily evaluation of the open wound with irrigation and aggressive surgical debridement until infection is halted was notably associated with reduced mortality.^{11,13,19} Broad-spectrum intravenous antibiotics should be empirically administered before the results of cultures to cover gram-positive cocci, gram-negative enteric rods, and anaerobic flora.²⁰ Most often, a broad-spectrum carbapenem (e.g., meropenem, imipenem/cilastatin, or doripenem) or extended-range penicillin/β-lactamase inhibitor combination (e.g., piperacillin/tazobactam or ticarcillin/clavulanate) is used.²⁰ Several third or higher generation cephalosporins can also be used, especially as part of a multidrug regimen.²⁰

CONCLUSION

On conclusion, our case emphasized the potential immunosuppressive role of pregnancy state in conjunction with diabetes mellitus to development of severe necrotizing soft tissue infections. Early recognition, determination of the extent of necrosis, appropriate aggressive therapy, and intensive care support are the most important predictors of survival in patients with NF.

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