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Postabortal Anterior Abdominal Wall Necrotizing Fascitis after Hysterotomy: A Case Report

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Abstract: Necrotizing Fasciitis (NF) is also known as the flesh eating disease. The incidence is rare. Mortality is a high as 40%. Very few cases have been reported in English literature. Researchers report a case of a previously healthy woman that developed necrotizing fasciitis after an uneventful hysterotomy that required antibiotics and debridment. The organisms causing were polymicrobial including *Staphylococcus aureus*, *Streptocoocus* sp. and *Klebsiella* sp.

Key words: Necrotizing Fasciitis (NF), flesh eating disease, English literature, healthy woman, Egypt

INTRODUCTION

Necrotizing fasciitis is a rare life threatening wound infection which requires prompt recognition and treatment. It is a polymicrobial infection that mainly involves the fascia and spreads very rapidly along the relatively ischemic fascial planes with wide spread undermining of surrounding tissue and extreme systemic toxicity. There is generalised thrombosis of the penetrating vessels causing devascularisation of the skin. The diagnosis should be suspected when a wound margin looks dusky and necrotic in a seriously ill patient (Moir-Busy *et al*., 1984; Farrell *et al*., 1980; Ott, 1981).

CASE REPORT

A 30 years old, G3P2 with history of two previous caesarean sections before was referred to the hospital from a general practitioner clinic with alleged missed abortion at gestational age of 20 weeks and severe vaginal bleeding after vaginal administration of misoprostol for termination of pregnancy. The patient was morbidly obese. She was complaining of labour-like pains and vaginal bleeding since 4 days after vaginal intake of misoprostol for termination of pregnancy. The patient was haemodynamically unstable. Blood transfusion was initiated. Ultrasound revealed a dead foetus of 20 weeks gestation with placenta completely covering the internal os. PV examination revealed severe vaginal bleeding and closed cervix. After correction of the general condition of the patient, the patient was transferred to the operating room for hysterotomy. A dead male foetus was delivered through pfennisteil incision. Surgery lasted for 2 h during which blood and plasma were transfused. After 5 days, the patient started complaining of abdominal pain, nausea, vomiting and fever. Her vital signs included a temperature of 38.5°C, a heart rate with sinus tachycardia of 120 min^{-1} and a blood pressure of 100/60 mmHg. On physical exam she had a firm, tender abdomen. There was erythema and swelling around the wound. Skin around the wound looked dusky red without any distinct margins. With a foul smelling, watery discharge from the incision. The wound was partially opened and probed there was no evidence of dehiscence. On removal of stitches copious amount of foul smelling seropurulent discharge came out which was sent for culture. Wound was bathed in hydrogen peroxide. Empiric antibiotic therapy was commenced. Relevant laboratory results showed the pus to contain a profuse growth of *Strept*., *Staph aureus* and *KlebsiellA* species. The patient's clinical condition had deteriorated and there was increasing abdominal wall induration and inflammation in the area between the umbilicus and the pfannenstiel scar which continued to drain a profuse quantity of pus. Haemorrhagic bullae and the colour of skin looked purplish. The diagnosis of necrotizing fasciitis was made. Her other admission laboratory tests were unremarkable except a white blood cell count of 25000 cell/mm^3.

Lower abdominal wall debridement of necrotic tissue anterior to the rectus abdominus muscle was performed under general anaesthesia. An abscess was found at the base of the previous incision. The infection had tracked up through the rectus sheath bilaterally. The necrotic fascia was much more extensive than skin involvement. During debridement the fascial tissues were relatively less vascular and fresh blood was transfused during the procedure (Fig. 1). Pathology later confirmed the

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Fig. 1: After debridment of necrotic areas at the incision scar and periumblical

Fig. 2: Healed incisions after 2 months

diagnosis of acute necrotizing fasciitis. Samples of necrotic tissue were sent for bacteriological analysis. The report of culture sensitivity ascertained the presence of *Strept.*, *Staph aureus* and *KlebsiellA* species. Post-surgical debridement, the patient improved dramatically and the wound started granulating. The wounds were left to heal by secondary intension as the patient did not accept secondary suture (Fig. 2).

DISCUSSION

The incidence of post-operative infection varies with the expertise of the surgeon, pre-operative preparations and the routines followed by the surgeon. Each extra hour of surgery doubles the risk of infection. Systemic factors like hypovolemic shock, complicated surgery, decreased immunity and diseases like diabetes mellitus are also important (Gibbs *et al*., 1983; Stage *et al*., 1977).

Necrotizing fasciitis is a clinical entity and not aspecific bacterial infection which is characterized by extensive necrosis of superficial fascia with wide spread undermining of surrounding tissue owing to infections/thrombosis of vessels. Though it is essential to avoid under estimating the severity of disease and confusing it with cellulitis, phlebitis or localized abscess, at the same time it is important not to confuse it with clostridial myositis and vascular gangrene and thereby over treat this condition. Early diagnosis can be confirmed by frozen section biopsy of wound (Stamenkovic and Lew, 1984). Necrotizing fasciitis has been reported on perineum at the location of episiotomy in the abdominal wall following caesarean section, abdominal hysterectomy, salpingectomy and laparoscopy (Henderson, 1977; Cederna *et al*., 1990; Sotrel *et al*., 1983).

As in the case, a minor penetrating injury or a surgical incision is usually involved with postoperative cases accounting for approximately 20% of the total (Thompson *et al*., 1993). While group A Streptococcus is the most common monomicrobial isolate, polymicrobial infections with a variety of Gram-positive, Gram-negative, aerobic and anaerobic isolates can also occur. The polymicrobial combination of the patient's organisms is unique from previously described cases. The aetiology of necrotizing fasciitis is not fully understood. Major risk factors include diabetes mellitus as well as age >50 years and appear to be associated with a higher mortality and morbidity (Francis *et al*., 1993). NSAIDs have been linked to necrotizing fasciitis in a temporal fashion. It is controversial if the NSAID effect is to simply mask the signs and symptoms to delay diagnosis of the necrotizing fasciitis (Aronoff and Bloch, 2003) or if inhibition of granulocyte chemotaxis, phagocytosis and bactericidal activity and depression of lymphocyte transformation documented to occur *in vivo* is occurring in these patients (Rown and North, 1995).

Most patients present with signs of inflammation such as erythema, swelling and pain at the infected site. Severe pain disproportionate to local findings and associated with systemic toxicity should definitely raise the suspicion of necrotizing fasciitis.

Radiologic plain film imaging reveals gas in the muscles and superficial fat in only approximately 35% of cases. CT scan may help distinguish cellulitis from necrotizing fasciitis and may help guide management

(Wall *et al*., 2000). The therapy is based on suitable combination of antibiotics, surgical removal of necrotic tissue, rinsing with hydrogen peroxide and longitudinal facial incisions in order to lower tension in the muscle.

The histologic findings indicating necrotizing fasciitis include necrosis of superficial fascia, fibrinous thrombi of the blood vessels penetrating the fascia, presence of polymorphonuclear leukocytes in the fascia and nomuscle involvement. It is important to note that serial debridement may be necessary and that closure of the fascia after the first debridement is not advisable to facilitate further operations. Serial debridements may also limit the size of the final defect needing to be reconstructed.

CONCLUSION

Necrotizing fasciitis in the postpartum patient remains a rare challenge with a high mortality. This patient's rapid deterioration with septic shock and multisystem organ failure could have resulted in maternal death. Clinical suspicion must remain high despite the rarity of the disorder as early diagnosis is critical and is most commonly made without confirmatory radiological imaging. Early aggressive debridement of any and all necrotic tissue, forms the cornerstone of therapy with additional serial debridements offering the best chance of survival.

REFERENCES

Aronoff, D.M. and K.C. Bloch, 2003. Assessing the relationship between the use of nonsteroidal anti-inflammatory drugs and necriziting fasciitis caused by group A streptococcus. Medicine, 82: 225-235.

Cederna, J.P., B.W. Davies, S.A. Farkas, J.A. Sonta and T. Sworniowski, 1990. Necrotizing fasciitis of the total abdominal wall after sterilization by partial salpingectomy case report and review of literature. Am. J. Obstet. Gynecol., 163: 138-139.

Farrell, S.J., H.F. Andersen and B.A. Work Jr., 1980. Cesarean section: Indications and postoperative morbidity. Obstet. Gynecol., 56: 696-700.

Francis, K.R., H.R. Lamaute, J.M. Davis and W.F. Pizzi, 1993. Implications of risk factors in necrotizing fasciitis. Am. Surgeon, 59: 304-308.

Gibbs, R.S., J.D. Bianco and P.J. St Clair, 1983. A case-control study of wound abscess after cesarean delivery. Obstet. Gynaecol., 62: 498-501.

Henderson, W.H., 1977. Synergistic bacterial gangrene following abdominal hysterectomy. Obstet. Gynecol., 49: 24-27.

Moir-Busy, R.M., R.M. Hutton and J.R. Thompson, 1984. Wound infection after caesarean section. Hospital Infect., 5: 359-370.

Ott, W.J., 1981. Primary cesarean section: Factors related to postpartum infection. Obstet. Gynecol., 57: 171-176.

Rown, J.A. and R.A. North, 1995. Necrotizing fasciitis in the puerrprium. Am. J. Obstet. Gynecol., 173: 241-242.

Sotrel, G., E. Hirsch and K.C. Edelin, 1983. Necrotizing fasciitis following diagnostic laparoscopy. Obstet. Gynecol., 62: 67S-69S.

Stage, A.H., H. Long, R. Silberman, D.P. Moradiellos and C.M. Greene, 1977. Wound infection following cesarean section. Surg. Gynecol. Obstet., 145: 882-884.

Stamenkovic, I. and P.D. Lew, 1984. Early recognition of potentially fatal necrotizing fasciitis: The use of frozen-section biopsy. New England J. Med., 310: 1689-1693.

Thompson, C.D., A.L. Brekken and W.H. Kutteh, 1993. Necrotizing fasciitis: A review of management guidelines in a large obstetrics and gynecology teaching hospital. Infect. Dis. Obstet. Gynecol., 1: 16-22.

Wall, D.B., S.R. Klein, S. Black and C. de Virgilio, 2000. A simple model to help distinguish necrotizing fasciitis from nonnecrotizing soft tissue infection. J. Am. Coll. Surgeons, 191: 227-231.