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Case Report

Management of patient with necrotizing fasciitis: a challenge for anaesthesiologist

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ABSTRACT

Necrotising fasciitis, a highly lethal infection of deep seated subcutaneous tissue and fascia, is associated with high mortality and long term morbidity. A five year old child of necrotizing fasciitis with poor general condition, deranged investigations and unstable vitals was posted for debridement. After initial resuscitation, TIVA was given, intraoperative period was uneventful and post operatively patient was shifted to recovery room as fully conscious with O_2 by face mask. After 1½ hours, patient became drowsy, hypotensive with bradycardia and urine output was nil. Immediate resuscitation started and vasopressors added. Despite all aggressive interventions, the patient died due to sepsis induced multiorgan failure. Blood samples and wound aspirate culture showed group A beta hemolytic Streptococcous. In this case report we discuss the best possible management of such patients and tried to minimize several barriers like lack of early recognition of severe sepsis and septic shock, treatment delay, lack of several investigations and drugs, shortage of health care providers, absence of locally written protocol, remote area and tried to give the massage that adherence to published guidelines for the management of severe sepsis patients lowers mortality.

Keywords: Sepsis, Anaesthesia, Necrotizing fasciitis

INTRODUCTION

Necrotising fasciitis is a highly lethal infection of subcutaneous tissue and fascia. Initial clinical characteristics include pain, swelling, erythema and as disease progresses skin discoloration, bullae, and necrosis are often observed. Mortality rate ranges from 30%-50%.[1] Necrotising fasciitis often results in serious complications such as acute respiratory distress syndrome, acute renal failure, cardiac failure and concomitant nosocomial infections.[1]

CASE REPORT

A 5 year old male child weighing 15 kg came to emergency department with history of swelling, blackening and blisters over right anterior chest wall, back, abdominal wall and right shoulder. Patient's relatives noted a small erythematous patch over right chest wall 6 days back which rapidly increased and spread to large area. On physical examination, general condition was poor, child was conscious but lethargic with Heart Rate (HR) 127/min, Blood Pressure (BP) 86/42 mmHg, SPO_2 92% on room air, respiratory rate 32/min and temperature 36 degree centigrade. Skin discoloration and bullae were observed over the chest wall. Lab investigations showed hemoglobin 7.2 gm%, Total Leucocyte Count (TLC) 37000/cmm, polymorphs 68, and platelets were 90000 with blood urea 86 mg/dl, creatinine 1.5 mg/dl, serum sodium 122 meq/l and potassium 5.1 meq/l with normal chest X-ray. Arterial Blood Gas Analysis (ABGA), Sr. lactate, CT scan and pediatric central venous line were not available as our institute is a developing tertiary centre in remote area.

Patient was posted for urgent surgical debridement within three hours of admission under ASA grade IV E. After preloading with 250 ml ringer lactate and 150 ml of whole blood, HR settled down to 92/min, BP 104/56 and mental status improved. Foley's catheterization was done and initial urine output was 25 ml. Intravenous (I.V.) antibiotic amoxicillin clavulanic acid 350 mg bd was administered. After preoxygenation, intravenous induction of anaesthesia was done with inj. glycopyrollate 0.2 mg, inj. ondensterone 2 mg and inj. ketamine 20 mg initially with 5 mg intermittently till total 45 mg and spontaneous/assisted mask ventilation maintained with oxygen. Operative findings revealed diffuse necrosis of skin, fascia and muscles and nearly 250 ml pus was drained. Intraoperatively total Ringer lactate 600 ml and whole blood 250 ml was given. Surgery lasted for 15 minutes and intraoperative period was uneventful. Chest and abdominal dressing in the form of strapping was done and patient was shifted to post op recovery room with monitor as fully conscious with HR 102/min, BP 94/58 mmHg SPO2 100% with O2 with adequate tidal volume by facemask. After 1½ hours, suddenly patient became drowsy, hypotensive with bradycardia. HR was 32-35/min, saturation was unrecordable. Immediately inj. atropine 0.3 mg i/v given, patient intubated, ventilatory support provided with 100% O_2 and dopamine@ 15 mic/kg/min was started. Inj. hydrocortisone 60 mg was given. CPCR was started and dobutamine infusion added. Despite all aggressive medical and surgical interventions, the patient died due to sepsis induced multi organ failure.

DISCUSSION

Necrotising fasciitis occurs commonly in patients with diabetes, alcoholism, vascular disease, intravenous drug abuse, obesity, malnourishment and/or immunosuppression.[1] It may also occur due to local tissue trauma in the form of contusion, ingrown nail or small burn. In the present case, the patient was 5 year old malnourished child from poor economic status with six days old history and presented late in emergency department only when family members noted drowsiness. Early diagnosis, aggressive fluid resuscitation, early emergency surgical debridement, and appropriate antibiotic therapy decrease both morbidity and mortality. Here anaesthesiologist play a central role as immediate surgery or within few hours of presentation and critical care support is life-saving.[2] Otherwise this could probably end in mortality due to overwhelming sepsis syndrome and/or multi organ failure. Most commonly found organism in necrotizing fasciitis is group A streptococcus but polymicrobial infections with gram positive and negative aerobic and anaerobic organisms may be involved. Clinical symptoms are variable but usually present with pain and redness progressing rapidly. Fever, chills, myalgia, diarrhea, cellulitis, edema, blisters, subcutaneous crepitus may also be present. Within 72-96 hours significant signs of dehydration, altered mental status, and cardiovascular instability are evident in the absence of treatment and later on multisystem failure may occur.[1]

In our patient the clinical deterioration was fast and he landed up in multiorgan failure. The lab risk indicator for necrotizing fascitis (LRINEC) is based on C reactive protein, Hb, TLC, electrolytes, creatinine, and blood sugar and has high specificity. Patients with LRINEC score >6 have high mortality rate.[3]

Laboratory tests include complete blood count, coagulation status, electrolytes, typing and cross match, liver function tests, renal function tests, arterial blood gas to assess pulmonary status, lactate level to assess tissue perfusion. Additional laboratory tests include blood, urine, CSF and sputum cultures, and X-ray films according to symptoms.[2] C-reactive protein levels rise in sepsis. Procalcitonin is useful in discriminating septic shock from nonseptic shock.[4] Broad spectrum coverage involving a third-generation cephalosporin or imipenem plus an aminoglycoside is considered as the standard of care.[5] Early goal-directed resuscitation (Table 1) within the first 6 hours of patient arrival in the emergency department helps in reduction in the mortality of the patients with sepsis and septic shock.[6] Blood transfusion should be avoided unless Hemoglobin is less than 7 gm/dl.[7]

Ideally[6,8] (Table 1) invasive monitoring should be considered in patients with comorbid conditions. Central venous pressure should be restored to 8-12 mmHg; mean arterial pressure should reach above 60 mmHg; and central venous oxygen saturation should be greater than 70% but unfortunately these were lacking at our part. Inadequate perfusion parameters should be corrected with administration of normal saline or lactated Ringer solution. Colloids with pentastarch are associated with higher rates of acute renal failure than Ringer's lactate.[1,9] Albumin should be used in fluid resuscitation when patient requires substantial amounts of crystalloids.[10] Lack of response to IV fluids necessitates use of vasopressors. Surviving sepsis guidelines 2012 recommend norepinephrine (NE) as first choice vasopressors[10] followed by dopamine and dobutamine. If these agents prove inadequate, norepinephrine or vasopressin 0.03 units/minute can be added. Levosimendan may be a useful adjunct to inotropic therapy in refractory cases.[1] Once pharmacologic agents become necessary to combat hypoperfusion, the risk of death climbs to 80%.[11] We used dopamine as it was readily available. Among alternate measures, human recombinant activated protein C (drotrecogin alpha) works via anti-inflammatory and anticoagulation effects and used to treat patients who had multiorgan dysfunction and a high likelihood of mortality. A major risk of this treatment is serious bleeding.[12] Adult patients with severe sepsis and low risk of death (typically, APACHE II <20 or one organ failure) should not receive rhAPC.[13] I.V. hydrocortisone and tight control of blood glucose has demonstrated a significant reduction in mortality.[14]

Table 1: Early goal directed therapy.

Infection	Goals to be achieved	Achieve by
Cardio vascular system	MAP ≥65 mmHg	i.v. fluids ↓ (If no response) Dopamine first line drug ↓ (If no response) Add dobutamine ↓ (If no response) Start norepinephrine ↓ Last resort epinephrine
	PCWP 14-18 mmHg	
	CVP 8-12 mmHg	
	CvO_2 saturation ≥70 mmHg	
	Hematocrit ≥30%	Red blood cell transfusion
Renal	Urine output ≥0.5 ml/kg/hour normalization of creatinine	i.v. fluids, vasopressor, inotropes
Pulmonary	O_2 saturation >88% Minimize A-a gradient	Oxygen supplementation Mechanical ventilation
Others	Remove source of infection Start antibiotic as early as possible	

Anaesthesia management

Here we discuss some key points for anesthesia management. In necrotizing fasciitis patients, the combined effects of sepsis, anaesthesia, intravascular volume loss, bleeding, and surgical stress results in unstable cardiovascular status and patient may present in septic shock.[1] Such patients need highest level of surgical priority. Sufficient preoperative systemic blood pressure is not indicative of adequate volume status, and therefore, heart rate, urine output, and mental status should all be considered[2] and vigorous fluid resuscitation and correction of hemoglobin and clotting deficiencies should be done and antibiotic should be continued. Risk for aspiration is high since ileus is often associated with sepsis.[2] A modified rapid sequence induction, using rocuronium rather than succinylcholine to facilitate tracheal intubation may be required. Options for the induction technique are- ketamine, etomidate and propofol- and should be done using small doses of i.v. anaesthetic agents, titrated to clinical response. We preferred ketamine in this patient due to its good hemodynamics properties, preservation of respiration and better availability. Among inhalational agents, sevoflurane is the agent of choice and the MAC is reduced in severe sepsis.[15] We avoided inhalational agent due to its tendency of decreasing tidal volume. Severe sepsis may lead to Multiple Organ Failure (MOF) so use of arterial and central venous catheters, capnography and monitoring of core temperature is necessary.[2] Among other treatment modalities, hyperbaric oxygen-therapy is administered at 2.5 to 3.0 atmospheres for 90 min twice daily following surgical debridement until no ongoing necrosis is evident.[16] Peripheral nerve blocks and neuraxial block (spinal and epidural anaesthesia) should be undertaken with caution, as their haemodynamic effects in sepsis-induced cardiovascular compromise patients may be difficult to reverse.[17,18] Recent blood tests confirming normal coagulation are essential.[1]

Postoperatively mechanical ventilation is often continued in persistent hemodynamic instability[2,8] or according to arterial blood gases obtained preoperatively.

CONCLUSION

Patients with severe sepsis often require surgery for source of infection control. The anaesthetist has a crucially important role in patient survival outcome. In this case the patient presented with signs and symptoms of severe sepsis. Ideally measures should include 6 hour resuscitation bundle with lactate monitoring, early cultures and antibiotic and early goal directed therapy (intensive fluid resuscitation and catecholamine administration). The standard intensive measures also include ventilation support, activated protein c administration, tight glucose control, steroids and renal replacement therapy. Alternative measures such as hyperbaric oxygen, immunoglobulin or granulocyte colony stimulating factor may also be effective. Although adherence to published guidelines for the management of severe sepsis patients is known to lower mortality, actual adherence to these recommendations is low. There are several barriers like lack of early recognition of severe sepsis and septic shock, treatment delay, difficulties in obtaining central venous oxygen saturation and pressures, shortage of health care provider, absence of locally written protocol, remote area, non-availability of many investigations and drugs etc. In the above case despite of our best efforts we lost the patient. We believe that uniform adherence to current recommendations and achievement of the goals may heighten the chances of survival.

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REFERENCES

1. Eissa D, Carton EG, Buggy DJ. Anaesthetic management of patients with severe sepsis. Br J Anaesth. 2010;105(6):734-43.
2. Baluch A, Janoo A, Lam K, Hoover J, Kaye A. Septic shock: review and anaesthetic considerations. MEJ Anesth. 2007;19(1):71-86.
3. Wong CH, Khin LW, Heng KS, Tan KC, Low CO. The LRINEC score: a tool for distinguishing necrotizing fasciitis from other soft tissue infections. Crit Care Med. 2004;32(7):1535-41.
4. Luzzani A, Polati E, Dorizzi R, Rungatscher A, Pavan R, Merlini A. Comparison of procalcitonin and C-reactive protein as markers of sepsis. Crit Care Med. 2003;31(6):1737-41.
5. Roach AC. Antibiotic therapy in septic shock. Crit Care Nurse Clin North Am. 1990;2(2):179-86.
6. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med. 2001;345:1368-77.
7. He´bert PC, Wells G, Blajchman MA, Marshall J, Martin C, Pagliarello G, et al. A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care. Transfusion Requirements in Critical Care Investigators, Canadian Critical Care Trials Group. N Engl J Med. 1999;340:409-17.
8. Stoelting R, Dierdorf S. Early goal-directed resuscitation. In: Stoelting R, Dierdorf S, eds. Anaesthesia and Co-existing Disease. 4th ed. London: Churchill Livingstone; 2002: 571-584.
9. Brunkhorst FM, Engel C, Bloos F, Meier-Hellmann A, Ragaller M, Weiler N, et al. Intensive insulin therapy and pentastarch resuscitation in severe sepsis. N Engl J Med. 2008;358:125-39.
10. Surviving Sepsis Campaign. Guidelines, 2012. Available at: www.survivingsepsis.org/guidelines.
11. Edwards JD. Practical application of oxygen transport principles. Crit Care Med. 1990;18:S45-8.
12. Bernard GR, Vincent JL, Laterre PF, LaRosa SP, Dhainaut JF, Lopez-Rodriguez A, et al. Efficacy and safety of recombinant human activated protein C for severe sepsis. N Engl J Med. 2001;344:699-709.
13. Laterre PF, Levy H, Clermont G, Ball DE, Garg R, Nelson DR, et al. Hospital mortality and resource use in subgroups of the Recombinant Human Activated Protein C Worldwide Evaluation in Severe Sepsis (PROWESS) trial. Crit Care Med. 2004;32:2207-18.
14. van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, et al. Intensive insulin therapy in the critically ill patients. N Engl J Med. 2001;345:1359-67.
15. Allaouchiche B, Duflo F, Tournadre JP, Debon R, Chassard D. Influence of sepsis on sevoflurane minimum alveolar concentration in a porcine model. Br J Anaesth. 2001;86:832-6.
16. Taviloglu K, Yanar H. Necrotizing fascitis: strategies for diagnosis and management. World J Emerg Surg. 2007;2:19.
17. Guay J. Benefits of adding epidural analgesia to general anesthesia: a meta-analysis. Br J Anaesth. 2006;20:335-40.
18. Schuz-Stubner S, Pottinger JM, Coffin SA, Herwaldt LA. Nosocomial infections and infection control in regional anaesthesia. Acta Anaesthesiol Scand. 2008;52:1144-57.

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