

# **Original Research Paper**

# Anaesthesiology

## **AIRWAY MANAGEMENT IN EXTENSIVE FACIAL BURNS- A CASE REPORT**

Dr Rathna Paramaswamy*	Professor, Anaesthesiology, Saveetha Medical College, Chennai, *Corresponding Author
Dr Shakir R.T	MD (Anaesthesia) PG student , Saveetha Medical College, Chennai

Anesthetic management of burns of the head, neck, face and upper airway needs special precautions as establishing the airway provides challenges to the anesthesiologist. A 27 year old female presented with acute second degree thermal burns of the head, neck, face, shoulders and was posted for emergency escharotomy and dressing. Her scalp hair was singed, nasal mucosa was burnt and her whole face and neck were burnt with huge blisters and raw areas over the chest, shoulders and both upper limbs. Intravenous line was established through the right femoral vein and she was resuscitated with ringer lactate. Holding the face mask was impossible in view of her facial burns. Awake oro tracheal intubation was done by fiber optic bronchoscopy guidance and airway was established successfully. Anesthesia was maintained by titrated doses of opioids, non-depolarising muscle relaxants and inhalational agents. She recovered well and was extubated awake. Hence awake fiber optic bronchoscope guided intubation can be done to establish the airway in extensive head, neck, face and upper airway burns.

# **KEYWORDS**: Burns, Airway management, awake fiber-optic intubation

#### INTRODUCTION:

Out of the several life threatening traumas, burn injuries are the leading cause. The increased mortality and morbidity is associated with percentage of burns, any inhalation injuries, and surface area involved. Burns involving face, genitalia, or airway are always considered as major burns irrespective of degree of burns [1]. Thermal burns from fire or flame and hot water sources for 80% of all reported burns and can be classified on depth of burns [2, 3]. Burns can be classified on the basis of depth of tissue involved.

First-degree burns are considered mild compared to other burns. They result in pain and reddening of the epidermis (outer layer of the skin).

Second-degree burns (partial thickness burns) affect the epidermis and the dermis (lower layer of skin). They cause pain, redness, swelling, and blistering.

Third-degree burns (full thickness burns) go through the dermis and  $affect \, deeper \, tissues. They \, result \, in \, white \, or \, blackened, \, charred \, skin$ that may be numb. Burns involving Total surface area >20% can result in acute systemic response known as burn shock. [2, 3]. It is characterized by increased capillary permeability, increased hydrostatic pressure across the microvasculature, protein and fluid movement from the intravascular space into the interstitial space, increased systemic vascular resistance, reduced cardiac output, and hypovolemia requiring fluid resuscitation [4,5 6]. The tissue odema during first 8 hours after burn injury is faster as compared to later phase. Due the activation of the inflammatory response and release of inflammatory mediators, the normal physiology of the body organs is altered. Burn injury can alter the entire immune system resulting in decreased immune functioning and patients are more  $susceptible for in fection. The {\it systemic disorders observed in the first}$ few hours after a severe burn injury can have increased systemic capillary permeability with protein leakage and a tendency toward hypovolemic shock [2, 3, 4, 5, 6, 7].

Lipid protein complex (LPC) released after burn injury is found to be major cause of immune suppression. Abnormal levels of proinflammatory mediators, such as tumour necrosis factor alpha (TNF-), interleukin-1b (IL-1b), interleukin-6 (IL-6), interleukin-8 (IL-8), and interleukin-10 (IL-10) have been reported both systemically and locally in burn patients [4,5]. Early escorotomy helps in early healing and prevents damage by the inflammatory mediators. Anaesthetic management is a challenge in the initial and late face of burn injury. The most common cause of death in burn injury is inhalational

injury. The patients having burns injury overface, neck and shoulder will be challenging to anaesthesiologist because of their difficulty in mask ventilation, oedema in the airway, painfully restricted mouth opening and their limited neck mobility [5]. Awake fibre optic intubation is widely done for anticipated difficult airway. Topical lignocaine spray and gargle is used to reduce the discomfort and make the procedure painless.

### **CASE REPORT:**

Here we present a 27 year old female patient who was brought to the emergency room with extensive thermal burns over face, neck, shoulder with 30% total body surface area involvement. On examination she was conscious, oriented to time, place and person and was in severe pain. Her blood pressure was 100/60 mmhg in her right lower limb, heart rate was 100 beats per minute and oxygen saturation was 95 % in room air. Her haemoglobin was 10 gm %, platelet 2.05 lakhs/cmm, urea 14.2 mg/dl, creatinine 0.47mg/dl, sodium140mEq/L and potassium4.5mEq/L.

On examination her scalp hair was singed, nasal mucosa was burnt and she had huge blisters over both shoulders, upper limbs and chest. Intravenous access was established in the right femoral vein and fluids were given according to Parkland formula. Holding face mask was impossible in this patient due to her facial burn injury. She had a restricted mouth opening, restricted neck movements and oedema over her oral mucosa. After shifting the patient to operation theatre she was connected to a multipara monitor and ECG, SPO2,ETCO2, core temperature and NIBP was monitored.

Her oral cavity was anaesthetised by gargling with 2% lignocaine. Awake oro tracheal intubation was done with fibre optic bronchoscopy guidance with spray as you go technique with 2% lignocaine. Bronchoscopy revealed extensive oral mucosal oedema up to the vocal cord. A 6.5 mm cuffed armoured latex tube was inserted and tube position was confirmed with capnographic trace and air entry on auscultation. Induction of anaesthesia was done with IV propofol 2mg/kg and fentanyl 2mcg/kg, Anaesthesia was maintained with a mixture of air and oxygen (50:50) with sevoflurane and titrated dose of atracurium was used with controlled ventilation. Her hemodynamic parameters were stable in the intra operative period. At the end of the procedure, residual neuromuscular blockade was reversed with 2.5 mg of neostigmine and 0.4 mg glycopyrrolate. She was extubated when fully awake with protective airway reflexes, and the neuromuscular transmission recovered to a train of four (TOF) ratio of 0.9 and was shifted to the PACU for the further management.



Figure 1- Picture of the patient prior to induction.



Figure 2- Picture of the patient after intubation.



Figure 3- Bronchoscopy showing congested airway.

## **DISCUSSION**

Burn injuries are associated with intense pain, inflammation mediated hyperalgesia, severe haemodynamic changes and psychological trauma. Careful assessment of the risk factors (area involved, inhalational injury, and difficult airway) and decision making is extremely important. The pharmacokinetics and pharmacodynamics of anaesthetic drugs are altered in burn

patients [6, 8 9, 10]. Judicious use of sedatives, opioids and non-depolarising muscle relaxants has to be considered. In our case the patient had airway oedema, inhalational injuries, face and neck burns with limited airway neck mobility. Awake fibre optic intubation may be the safest option for securing the airway in such a scenario where even mask holding is technically impossible.

### CONCLUSION

Anaesthesia must not be delayed in acute burns because early escharotomy and dressing are critical for patient's survival. Anaesthetic management includes adequate analgesia, careful consideration of the haemodynamic sequence due to massive fluid shifts, inflammatory response and pathophysiological changes from extensive tissue destruction. Airway management in extensive burns of head, neck and face is a potential challenge to the anaesthesiologist.

### REFERENCES

- Taylor SL, Lawless M, Curri T, Sen S, Greenhalgh DG, Palmieri TL.Burns. 2014 Sep;40(6):1106-15.
- Hettiaratchy S, Dziewulski P (2004) ABC of burns: pathophysiology and types of burns. BMJ 328:1427-1429.
- Yeung, JK, Leung, LT, Papp, A A survey of current practices in the diagnosis of and interventions for inhalational injuries in Canadian burn centres.. Can J Plast Surg. (2013).21221–5
- Accardo Palumbo, G. I. Forte, D. Pileri et al., "Analysis of IL-6, IL-10 and IL-17 genetic polymorphisms as risk factors for sepsis development in burned patients," Burns, vol. 38, no. 2, pp. 208–213, 2012
- Wilmore, DW, Goodwin, CW, Aulick, LH, Powanda, MC, Mason, ADJr, Pruitt, BAJr Effect of injury and infection on visceral metabolism and circulation.. Ann Surg. (1980). 192 491–504
- Predicting mortality from burns: the need for age-group specific models.Taylor SL, Lawless M, Curri T, Sen S, Greenhalgh DG, Palmieri TL.Burns. 2014 Sep;40(6):1106-15.
- Mackie, DP, Spoelder, EJ, Paauw, RJ, Knape, P, Boer, C Mechanical ventilation and fluid retention in burn patients. J Trauma. (2009). 67 1233–8;
- Pham TN, Cancio LC, Gibran NS, American Burn Association. J Burn Care Res. 2008 Jan-Feb; 29(1):257-66.
- Suzuki N, Niiyama Y, Tokinaga Y, Yamakage M. [Anesthetic management in a patient with head and neck burn by asphalt]. Masui. 2013 Oct;62(10):1250-2. Japanese.
- Edward A. Bittner, M.D., Ph.D., F.C.C.M., Erik Shank, M.D., Lee Woodson, M.D., Ph.D., J. A. Jeevendra Martyn, M.D., F.R.C.A., F.C.C.M. Acute and Perioperative Care of the Burninjured Patient, Anesthesiology 2015; 122:448-64