

Management Of Pulmonary Oedema & ARDS following suicidal hanging

T Garg

Citation

T Garg. *Management Of Pulmonary Oedema & ARDS following suicidal hanging*. The Internet Journal of Anesthesiology. 2007 Volume 17 Number 1.

Abstract

This article sums up the successful management of a patient following non-lethal hanging. Airway obstruction is the main cause of morbidity, though pulmonary oedema and ARDS may develop subsequently. Pulmonary oedema following suicidal hanging has been reported very infrequently, probably due to poor survival rate in such cases. Though in most cases it is a reversible process if recognized and treated properly.

CASE REPORT

A 19 year old female patient was brought to our ICU with alleged history of suicidal hanging. On arrival she was in respiratory distress, with peripheral cyanosis. Her pulse rate was 172/min, SpO₂ -30%, Blood pressure 60mmhg systolic. She was exuding copious pink frothy secretion from oral cavity and was in delirious state.

The patient was immediately intubated with 7 no. cuffed portex endotracheal tube and was ventilated with AMBU bag on 100%O₂, saturation improved to 80% within few seconds. The patient was put on a ventilator with following settings: Mode-Assist contro l(ACMV), FiO₂-1, PEEP-10cm of H₂O, Tidal volume -400 ml, Respiratory rate 18/min, to achieve I:E ratio of 1:2. (weight of the patient-60 kg). Peak airway pressure varied between 30-35 cm of H₂O with these settings. Dopamine infusion was started at the rate of 3-5 ug/kg/min to maintain systolic blood pressure above 90 mm hg.

She was sedated with 5mg diazepam every 2hour, for next 12 hours. After stabilization of blood pressure, 10 mg lasix iv was given which resulted in brisk diuresis of 800 ml. Oxygen saturation improved to 95% and pink frothy secretions subsided. Chest-Xray done in ICU revealed bilateral non homogenous opacities. Blood gas analysis could not be done due to non-availability of this facility. Coarse crepitation were auscultated bilaterally.

Repeat chest-Xray next day revealed bilateral fluffy shadows (ARDS picture). The patient was kept sedated to increase ETT tolerance. Dopamine was continued and BP was

maintained 120-130/70-80mmhg. Antibiotics were started to prevent infection. PEEP was increased to 15 cm of H₂O, FiO₂ was decreased to 0.7, maintaining Spo₂ between 90-95%. Hemogram and all biochemical parameters were within normal limits. ECG showed sinus tachycardia. Nebulisation was done with Asthalin and Budecort. For the next two days the patient's condition remained the same.

On the fifth day the patient was conscious, was tolerating ETT well. On auscultation the chest was clear. Dopamine was tapered and stopped after BP stabilized at 100-110 mm hg systolic, pulse rate was 120-130 /min. PEEP was reduced to 10 cm of H₂O. Enteral nutrition was established using nasogastric feed with prokinetics.

On the sixth day of hospitalization weaning from artificial ventilation was done using SIMV mode with decreasing rate of ventilation, followed by spontaneous breathing trial on T-piece for two hours. Post extubation BP was 110/70 mm hg, pulse rate was 92/min, SpO₂-99%, Xray-chest showed considerable improvement. Chest physiotherapy was instituted and the patient was encouraged to cough out secretions. She was discharged on the 12th day of hospitalization.

DISCUSSION

Post obstructive pulmonary oedema (POPE) is a rare form of fulminant non-cardiogenic pulmonary oedema which becomes apparent within seconds to minutes after relief of upper airway obstruction. If it is associated with diffuse pulmonary parenchymal injury resulting in severe respiratory distress and hypoxemic respiratory failure, then it

is known as Acute respiratory distress syndrome (ARDS). There are two recognized types of POPE. Type I follows a sudden, severe episode of upper airway obstruction such as postextubation laryngospasm, epiglottitis, choking, and is seen in strangulation and hanging. Type II POPE develops after surgical relief of chronic upper airway obstruction reported after removal of upper airway tumors. If untreated it results in a sequelae of multiple organ failure and death.

The presence of agitation, tachypnea, tachycardia, pink frothy pulmonary secretions, rales and progressive oxygen desaturation suggest diagnosis of POPE in appropriate clinical settings. Most cases respond promptly to treatment which consists of supplemental oxygen, ventilatory support and PEEP.

In patients who develop ARDS, aim of ventilation is to improve oxygenation without causing further damage to the lungs. Difficulties arise as some alveoli are normal and open whilst other alveoli are stiff and collapsed. It is therefore necessary to try to open the collapsed alveoli without damaging the normal areas.

Management consists of-

- Treating the underlying condition, providing support for failing systems and early invasive ventilation.
- Limiting the FiO₂ may help to prevent further lung damage.
- Limiting tidal volumes to 6-8 ml.kg⁻¹ has been shown to reduce mortality.
- Positive end-expiratory pressure.
- Drug induced diuresis.
- Inotropic support of cardiac function.
- Removal of secretions.
- Control of infections.
- Nutritional support.- Enteral nutrition should be established quickly, using nasogastric feed with prokinetics (such as metoclopramide) or nasojejunal feeding. TPN can be considered if all attempts at enteral feeding fail

To conclude, with proper management and care mortality in patient with non-lethal strangulation can be reduced.

CORRESPONDENCE TO

Dr Twinkle Garg, Gurunank hospital, Palwal, India. Email: gargtwinkle76@rediffmail.com

References

1. Lang SA, Duncan PG, Shephard DA, Ha HC. Pulmonary oedema associated with airway obstruction. *Can J Anaesth* 1990;37:210-8.
2. Oswalt CE, Gates GA, Holmstrom FM. Pulmonary edema as a complication of acute airway obstruction. *Rev Surg* 1977;34:364-7.
3. Luce JM. Acute lung injury and the acute respiratory distress syndrome. *Crit Care Med* 1998; 26(2): 369-76.
4. A Kaki, ET Crosby and AC Lui. Airway and respiratory management following non-lethal hanging. *Can J Anaesth* Vol 44, 445-450.
5. Galvis AG. Pulmonary edema complicating relief of upper airway obstruction. *Am J Emerg Med* 1987; 5:294-7
6. Adolph MD, Oliver AM, Dejak T. Death from adult respiratory distress syndrome and multiorgan failure following acute upper airway obstruction. *Ear Nose Throat J* 1994;73:324-7.
7. Soldano SL, Place LB, Edwards FH, Cohen AJ. Post-extubation non-cardiogenic pulmonary edema. *Mil Med* 1993;158:278-80

Author Information

Twinkle Garg

Consultant Anaesthesiologist, Gurunank Hospital