ADULT RESPIRATORY DISTRESS SYNDROME AFTER ATTEMPTED STRANGULATION

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SUMMARY

We describe a case of severe acute lung injury after attempted strangulation. The patient presented initially with cerebral irritability and florid, non-cardiogenic pulmonary oedema which were followed by a prolonged period of the adult respiratory distress syndrome, severe sepsis and multiple system organ failure, although the patient eventually survived. The pulmonary injury following strangulation is proposed to be a result of the generation of marked subatmospheric pressures within the lungs during vigorous inspiration against an obstructed airway, although the processes involved in the so-called neurogenic pulmonary oedema are difficult to exclude. (Br. J. Anaesth. 1993; 70: 583-586)

KEY WORDS

Lung: adult respiratory distress syndrome.

Many factors, chemical, physical and biological, have been associated with the evolution of the adult respiratory distress syndrome (ARDS) [1,2]. The expanded definition of ARDS [3] requires exposure to a process or pathology recognized to be associated with the condition. Mechanical asphyxiation has not been specifically identified as a cause of ARDS. In this report, we describe the evolution of ARDS and multiple organ failure in a patient asphyxiated by manual strangulation.

CASE REPORT

A 30-yr-old Caucasian female was admitted to our Accident and Emergency Department after an anonymous telephone call to the emergency services. She was restless, opisthotonic and unresponsive; the admission Glasgow Coma Scale (GCS) score was 7 (E^Mi). She was normotensive (systolic/diastolic pressure 120/90 mm Hg), tachycardic (heart rate 150 beat min⁻¹) and reasonably well oxygenated, breathing oxygen-enriched air via a simple face mask (SpO₂ 95%, Pao₂ 13.0 kPa). She was tachypnoeic, however, with a marked stridor and fine inspiratory râles in both lung bases. Ecchymoses and petechial haemorrhages were noted on the neck, cheeks, forehead and sclera, the soft palate was congested and bilateral papilloedema noted. Fresh bruising and linear scratches, but no ligature marks, were seen on the neck. A diagnosis of attempted manual strangulation was made, and in view of the evident significant diffuse cerebral insult and the possibility of upper airway injury, the trachea was intubated and the lungs ventilated. After a rapid sequence induction using fentanyl 250 µg, etomidate 20 mg and suxamethonium 100 mg, the trachea was intubated easily with an 8.0-mm cuffed tracheal tube, although haemorrhagic supraglottic oedema was noted. Profuse, blood-stained pulmonary oedema was observed soon after intubation, and the patient was transferred to our Intensive Care Unit for further management. The subsequent clinical events have been divided into three epochs, and described semi-quantitatively using the APACHE II [4] and acute lung injury scoring [3] systems (fig. 1).

Days 1–3. The prominent features of this initial period were acute respiratory failure and cardiovascular instability. Florid pulmonary oedema was associated with diffuse patchy opacification and bilateral pneumothoraces visible on the chest x-ray (fig. 2A). Satisfactory oxygenation was only achieved by positive pressure ventilation with inspired oxygen fractions of up to 0.8, and positive end-expiratory pressure of 5-10 cm H₂O. More refractory to treatment was the systemic hypotension, which failed to respond to inotropic agents (dobutamine 20 µg kg⁻¹ min⁻¹) and which improved only after infusion of 2500 ml of colloid. The site for such apparent volume loss was not clear: there were no long bone or pelvic fractures, abdominal lavage was negative and an ultrasound failed to demonstrate any hepatic, renal or splenic injury, although significant and unexpected ascites was demonstrated (fig. 2B). The considerable pulmonary oedema (which, together with the ascites, appeared responsible for the initial hypovolaemia) was persistently associated with a pulmonary artery wedge pressure less than 15 mm Hg, suggesting that it was non-cardiogenic in origin.
ventilation (the acute lung injury (ALI) score remaining greater than 2.5—the score at which lung injury is said to be severe [3]).

Days 4–22. In this second phase, persisting severe lung injury (ALI score > 2.5) was associated with non-pulmonary organ failure. Repeated episodes of severe sepsis suggestive of Gram negative septicaemia (pyrexia > 38.5 °C, cardiac index 5–6 litre min⁻¹ m⁻², systemic vascular resistance index 450–700 dyn s m⁻² cm⁻⁶) were observed, but always without any microbiological identification of the responsible organism. Despite minimal sedation, conscious level deteriorated, so that the APACHE II scores, including a GCS component, were particularly increased. Persistent ileus prevented successful enteral nutrition; total parenteral nutrition was therefore required. A striking thrombocytopenia developed (< 50 x 10¹² litre⁻¹), with increased concentrations of bilirubin (> 100 µmol litre⁻¹) and aspartate aminotransferase (70–272 iu litre⁻¹ (normal range 10–40 iu litre⁻¹)), and increased prothrombin ratio (1.4–1.9, days 2–12) suggesting modest liver disturbance. Renal function remained satisfactory throughout.

By day 11, the ALI score reached its peak value of 3.5. Bilateral pulmonary air leaks persisted with conventional positive pressure ventilation, but satisfactory oxygenation could not be maintained using high frequency jet ventilation. Core temperature (measured with the pulmonary artery flotation catheter) reached 41.6 °C and again a severe septicemic-like haemodynamic profile was noted without any further evidence of Gram negative infection. In view of the severity of the patient’s condition, it was felt necessary to treat several possibilities empirically. Thus the patient received methylprednisolone 2 g, a combination of broad spectrum antibiotics together with Centoxin 100 mg (monoclonal antibody to bacterial lipopolysaccharide, Centocor). As shown by the trend in the APACHE II scores,

Fig. 1. APACHE II and acute lung injury (ALI) scores. The clinical course has been divided into three epochs: I = acute lung injury; II = ARDS and multiple organ failure; III = recovery (see text for details). APACHE II data are presented with (●) and without (▲) the contribution of the Glasgow Coma Score. Severe pulmonary injury is defined as an ALI score > 2.5. The deterioration in lung function on days 32–38 reflected a second period of pulmonary oedema, on this occasion as a result of fluid overload. ↓ = Administration of methylprednisolone and Centoxin; *coagulase-negative staphylococcal septicaemia.

Although the patient was awake and orientated the day after the assault, gas exchange was still sufficiently impaired to prevent weaning from artificial ventilation (the acute lung injury (ALI) score remaining greater than 2.5—the score at which lung injury is said to be severe [3]).
from this point onwards the general trend was one of improvement, twice interrupted by episodes of coagulase-negative staphylococcal sepsicaemia (asterisks in figure 1) successfully treated with vancomycin. Conscious level improved also, as shown by the convergence of the APACHE II scores with and without the GCS score. High frequency jet ventilation was introduced successfully on day 17, and allowed the bronchopleural fistulae to heal.

Days 22-44. This third period was one of recovery. The patient was fully orientated and without any focal neurological defect. Enteral feeding was introduced successfully and the patient gradually weaned from artificial ventilation (although a continued susceptibility of the lungs to fluid overload was responsible for the deterioration in ALI score in days 31-35). The patient was transferred to a rehabilitation ward 44 days after admission to our Intensive Care Unit, and was discharged home after a total of 127 days in hospital.

DISCUSSION

The forensic features of fatal manual strangulation depend principally upon whether death occurs rapidly from vagally mediated cardiac arrest (as a result of intense carotid body/sinus stimulation), or more slowly from the combined effects of hypoxia (caused by upper airway obstruction) and intracranial hypertension (which develops when the venous drainage, but not the arterial supply, of the head is obstructed) [5]. The patient described in this case report displayed many of the features of the so-called hypoxic form of manual strangulation. The bruises and linear scratch marks around the neck relate to the assailant's attack itself, whilst the various manifestations of venous congestion in the head and neck (including supraglottic oedema and papilloedema) suggested significant jugular venous hypertension. It is not clear, however, if the acute brain injury seen at presentation could be explained by venous hypertension alone, or if cerebral hypoxia or ischaemia contributed.

The pulmonary consequences of fatal mechanical asphyxiation include acute focal emphysema and florid haemorrhagic pulmonary oedema. These features suggest an increase in the permeability of the alveolar–capillary barrier, and have been used to identify the cause of death in a series of homicides in which there was no external evidence of asphyxiation [6]. The florid pulmonary oedema with which this patient presented was associated with a normal pulmonary artery wedge pressure, suggesting that it was non-cardiogenic in origin. Some workers have proposed that the disruption of the alveolar–capillary barrier after asphyxiation is a consequence of the marked subatmospheric pressures developed within the lungs during forced inspiration against an obstructed upper airway [5, 6]. In this regard, this form of lung injury is similar to the well described phenomenon of pulmonary oedema after relief of upper airway obstruction associated with anaesthesia, laryngospasm, tumours, foreign bodies and goitre [7]. Other workers have shown that (experimental ligature) strangulation is associated with an intense, neurohumorally-mediated vasoconstriction which they propose may damage the pulmonary, renal and splanchnic microcirculations [8-10]. Intense pulmonary hypertension is also a frequent feature of neurogenic pulmonary oedema, although in this condition an increase in the permeability of the pulmonary microcirculation is thought to be a more fundamental pathophysiological feature, with increased pulmonary capillary pressure serving only to exacerbate alveolar flooding [11]. Either (or both) mechanisms could explain the florid pulmonary oedema seen in the early stages of this case, although the significant ascites with which this was associated suggests a process not restricted to the pulmonary microcirculation alone.

The progression to a protracted period of severe sepsis and multiple organ failure, (dominated by severe ARDS but accompanied by brain failure, persistent ileus, hepatic and bone marrow dysfunction), was unexpected and not described in a previous case report of pulmonary oedema after attempted strangulation [12]. It is tempting to attribute this to the release of toxins and microorganisms into the systemic circulation from a "leaky" gastrointestinal tract. Experimental ligature strangulation is known to disrupt the splanchnic circulation [10], and the marked ascites seen in this patient points to a similar disruption. In addition, sustained improvement was associated with the administration of methylprednisolone and a monoclonal antibody to bacterial lipopolysaccharide, both of which might interfere with various stages of the inflammatory pathways of severe sepsis [13].

The expanded definition of ARDS views acute lung injury as part of a spectrum of multiple system organ failure [3]. It requires that, in addition to identification of severe lung injury, there should also be evidence of non-pulmonary organ failure, with a history of clinical disorder or insult recognized to be associated with the evolution of the condition. It is not clear if, given these criteria, the term ARDS could be applied to the patient's initial condition, although there is no doubt that the lung injury was severe. In contrast, the later stage of established severe lung injury and multiple organ failure clearly qualified as ARDS, although strangulation is not a recognized cause. We propose that strangulation (and possibly other forms of mechanical asphyxiation) should be considered as physical insults capable of causing severe acute lung injury and ARDS.

REFERENCES


