

LETTER

Acute stridor due to an upper tracheal membrane following endotracheal intubation

To the editor:

A 30-yr-old woman developed a placental abruption at 33 weeks gestation which was complicated by disseminated intravascular coagulation associated with low plasma fibrinogen ($0.76 \text{ g}\cdot\text{L}^{-1}$; normal $1.5\text{--}4.0 \text{ g}\cdot\text{L}^{-1}$) and a prolonged prothrombin time (18.5 s; normal 11.5–15 s). She was resuscitated with 1 L of colloid gelatin, 6 units each of red cell concentrate and fresh frozen plasma, 10 units of platelets and 8 g of fibrinogen. She subsequently developed noncardiogenic pulmonary oedema with respiratory failure requiring endotracheal intubation and assisted ventilation. A 7.5 gauge cuffed endotracheal tube was used and the intubation was reported to be routine and uncomplicated and did not require bougie insertion. She was sedated with 200 mg Propofol and given 100 mg Suxemethonium as a muscle relaxant. She subsequently improved and was extubated after 48 h but 3 days later developed acute stridor which varied in intensity. Fibreoptic bronchoscopy revealed an annular membrane (fig. 1) which was adherent to the anterior tracheal wall and was collapsing inwards intermittently to almost completely obstruct the airway. This was removed by rigid bronchoscopy

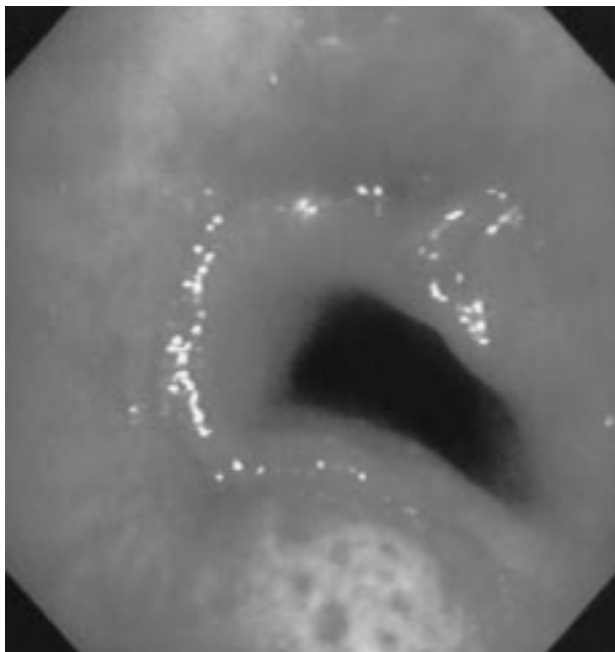


Fig. 1. – Membrane *in situ* as seen through a fibreoptic bronchoscope.

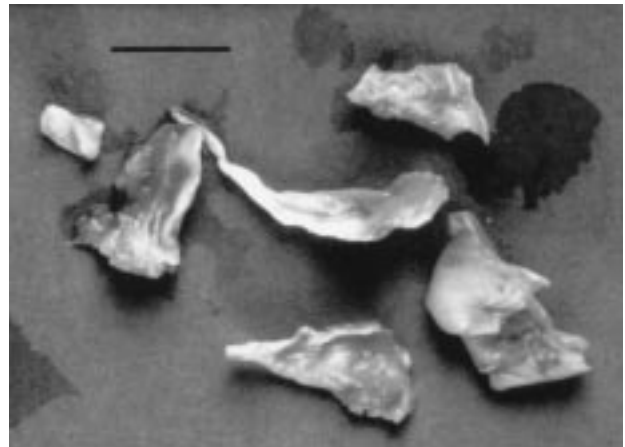


Fig. 2. – Membrane following removal. (Internal scale bar=1 cm.)

under general anaesthesia (fig. 2) and found to be composed mainly of fibrin on histological examination with few inflammatory cells. Repeat bronchoscopy 3 days and again 3 months later showed no recurrence of the lesion or significant tracheal stenosis.

This report describes a very rare, and potentially life-threatening, complication of endotracheal intubation which has been described only once before in two patients after tracheostomy [1] and adds to the already extensive list of potential complications of this procedure [2]. The mechanism of development of this fibrinoid membrane cannot be determined from the present report but it could be speculated that the fibrinogen therapy given during resuscitation together with the hyperfibrinogenaemia, which is a normal feature of late pregnancy and the early post-partum period [3], may have interacted with local tracheal trauma from the endotracheal tube to produce the lesion.

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