After premedication with midazolam and fentanyl, thiopentone and succinylcholine were administered. The trachea was intubated atraumatically with a size 7.5 mm cuffed tracheal tube (Sheridan Catheter Corporation). A No. 2 Miller blade previously cleaned and sterilized in two per cent glutaraldehyde solution[1,2] was used for intubation. Anaesthetic maintenance was with isoflurane in nitrous oxide/oxygen (50/50).

After several hours of uneventful surgery and anaesthesia the trachea was extubated and the patient was taken to the recovery room. He was given oxymorphone for pain relief. Fifteen minutes later he complained of swelling of his tongue. On examination, the tongue was twice its normal thickness; however, his airway and ability to swallow were intact. Diphenhydramine 50 mg IV and methylprednisolone 300 mg IV were administered. Over the next ten minutes the swelling of his tongue rapidly progressed to fill the entire oral cavity and force his mouth wide open. The patient complained of inability to clear secretions and his speech was obviously impaired. With no topical or regional anaesthesia, a fibreoptic nasal intubation was performed with a 7 mm tracheal tube. The epiglottis and vocal cords appeared normal. The patient was taken to the intensive care unit, breathing spontaneously without difficulty. Four hours after his arrival in the intensive care unit, the patient's tongue had returned to normal size. The tracheal tube was removed and the subsequent postoperative course was uneventful.

Discussion

The cause of this life-threatening complication is unclear. Three mechanisms for swelling of the tongue and oropharynx can be postulated: (1) mechanical trauma or obstruction of venous or lymphatic flow, (2) hypersensitivity response to intravenous medication, or (3) tissue reaction to a substance applied locally.

It seems unlikely that mechanical trauma would cause this symmetrical, profound, isolated swelling of the tongue – the intubation was atraumatic. Also, it is unlikely that the reaction was due to a systemic hypersensitivity response because it was confined to the tongue. The remainder of the mucous membranes in the oropharynx were normal, as were the epiglottis, vocal cords, and tracheal lining. A skin test for previous sensitization to thiopentone was negative. The most likely explanation for this patient's problem is a local reaction to an applied substance. The lidocaine jelly had been used with each previous occasion at this institution without complication. The tracheal tubes used are implantation-tested and carry the Z-79 designation. The most likely explanation for this problem may be the glutaraldehyde solution.

A skin test was performed with 0.02 per cent glutaraldehyde solution. The patient did not complain of burning or irritation in response to the intradermal injection.[3] However, there was a wheal-and-flare response that was interpreted by the allergist as indicating sensitization. While we cannot be certain that the result of this skin test was not due to local irritation by the glutaraldehyde, the response is strongly suggestive of prior sensitization.

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Acute pulmonary oedema after tourniquet release

To the Editor:

We would like to describe the unusual case of a fit adult man undergoing arthroscopy for septic arthritis who developed acute pulmonary oedema shortly after deflation of a limb tourniquet.

Case report

A fit 20-yr-old man, diagnosed as having septic arthritis of the knee, was scheduled for emergency arthroscopy and irrigation. Preanaesthetic assessment revealed: temperature 39° C; pulse rate 120 per minute; cardiorespiratory assessment was otherwise normal. All investigations were normal except for a leucocytosis of $19.5 \times 10^9 \cdot L^{-1}$. The patient was adequately fasted. Anaesthesia was induced with thiopentone 5 $mg \cdot kg^{-1}$ and maintained with

33 per cent oxygen, 66 per cent nitrous oxide and two per cent halothane breathing spontaneously via a face mask. Surgery and anaesthesia were unremarkable until the tourniquet was deflated after 50 min of occlusion. Within five minutes increasing tachypnoea and laboured respirations were noted. In the recovery room the patient exhibited clinical and radiological features of acute pulmonary oedema. The PaO_2 was 57 mmHg (7.6 kPa) with an inspired oxygen of 40 per cent. A provisional diagnosis of pulmonary aspiration was made and the patient was transferred to the intensive care unit, where he was re-anaesthetized, the trachea was instituted and the lungs were ventilated mechanically with positive end–expiratory pressure. Tracheal aspiration revealed frothy pink pulmonary oedema fluid with no evidence of aspiration of gastric contents. Pulmonary artery catheterization revealed PA pressure of 25/16 mmHg, an occlusion pressure of 8 mmHg but pulmonary vascular resistance was not determined. Neisseria meningitidis was isolated from the joint aspirate and one blood culture. High-dose benzylpenicillin was commenced. Eight hours after admission to the ICU a dramatic improvement in clinical signs had occurred and chest radiology was normal. The trachea was extubated and arterial blood gas analysis breathing 28 per cent inspired oxygen was satisfactory. Thereafter, he made an uneventful recovery.

There are many causes of acute pulmonary oedema and it was difficult to define the aetiology in this case. The wedge pressure excluded a cardiogenic cause. The absence of systemic arterial hypotension excluded a hypotensive insult to the lung, and upper airway obstruction did not occur at any time. The absence of associated features makes anaphylaxis unlikely. The onset of tachypnoea shortly after tourniquet deflation is consistent with a causal relationship, but in such circumstances the systemic effects of tourniquet deflation are mild and cardiopulmonary compromise is unusual.[1] It is proposed that tourniquet deflation was associated with a bacteraemia which caused acute non-cardiogenic pulmonary oedema due to increased microvascular permeability, and this mechanism is consistent with the rapid clinical recovery. Sepsis can cause pulmonary oedema[2] by increasing microvascular permeability[3] and endotoxin infusions have been used in animal models of acute lung injury.[4] Primary meningococcal arthritis is very rare,[5] but arthroscopy is known to precipitate bacteraemia in meningococcal arthritis,[6] and meningococcaemia has caused acute pulmonary oedema in children.[7]

The contribution of sepsis to the aetiology of this patient's pulmonary oedema is not proven, but it suggests that septicaemia may be associated with severe respiratory complications which may be precipitated by deflation of a limb tourniquet if used in the presence of a septic focus.

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A new syringe holder

To the Editor:

In most operating rooms the anaesthetist has only a small workspace which is often cluttered with a variety of equipment and a collection of syringes. Some of the syringes are clean, others are in use. Some are intended for the next case, and some are prepared in case of emergency. Some may have exposed needles. There is a considerable risk of syringe-swap errors and of needle-stick injuries, and it is often difficult to find the appropriate drug in a hurry.

I have made a device which helps solves these problems (Figure). It keeps the workspace tidy, and increases the amount of usable space. It permits one-handed re-sheathing of needles, thereby decreasing the risk of