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Anaphylactic shock after sensitization to gelatin

Editor—Allergic reactions are a known complication of general anaesthesia. A previous safe anaesthetic does not guarantee that subsequent anaesthesia with the same drugs will remain uneventful. We report here a case of anaphylactic shock to gelatin given 60 days after a previous uneventful administration.

A 64-yr-old woman was admitted with a fracture of L2 after a fall and a dorsal stabilization of Th11 to L3 was performed. General anaesthesia was given using propofol, sevoflurane, fentanyl, remifentanyl, and atracurium and the patient received crystalloid and gelatin without any complication. Her medical history included liver cirrhosis (Child A) and various allergies. Wound healing was delayed because of infection and negative pressure wound therapy was initiated. About 2 months after the initial operation, a secondary closure of the wound was planned and a general anaesthesia with propofol, fentanyl, and rocuronium was performed. Surgery was complicated by clinically relevant epidural bleeding and additionally to the volume substitution with crystalloids and infusion with gelatin was started. Shortly thereafter, the patient developed shock and the ECG showed ST-segment elevation. With a high degree of suspicion for an anaphylaxis, the infusion with gelatin was immediately stopped and therapy with oxygen, antihistamine, corticosteroids, catecholamines, and volume therapy with Ringer's lactate was started. As no improvement could be observed, external cardiac massage was started in the prone position. After 40 min of cardiopulmonary resuscitation, the patient showed return to a spontaneous circulation. Computer tomography excluded a pulmonary embolus, and the unstable patient was then transferred to the intensive care unit (ICU). Blood tests performed 60 min after the event showed a tryptase level of 144 E litre⁻¹ (normal <11.4 E litre⁻¹). The tracheal was extubated the following day on the ICU but she developed severe sepsis with *Pseudomonas aeruginosa* and died of multiorgan failure on post-operative day 4.

Post-mortem serum analysis from frozen probes established a high IgE titre in the plasma (7.19 kU litre⁻¹) confirming the allergic background. From the clinical situation, the

most likely diagnosis is IgE-mediated anaphylactic reaction to gelatin-based colloid.

During general anaesthesia, the incidence of anaphylaxis is in the range of 1 in 2000 to 1 in 20 000 anaesthetics.^{1–4} The most frequently responsible medications for anaphylaxis are the neuromuscular blocking agents (>60%) and latex (15%) followed by colloids, hypnotic, antibiotics, and opioids.^{5, 6} The particularity of this case is the fact that the patient received the identical gelatin during the first operation about 60 days before the event without any reaction.

The most important complication of gelatin-based colloids is the possible allergic reaction.² Gelatin-based substances are known to create a sensitization. Nakayama and colleagues⁷ observed that IgE antibodies to gelatin were detected in 93% of the patients with anaphylaxis during vaccination and that 98% of these patients had a previous vaccination with gelatin-containing vaccine. A cross-reaction between gelatin-containing vaccines and gelatin-based colloids is possible.⁸ The time necessary for a sensitization to occur is still not elucidated but a minimum of 10–15 days seems to be necessary for a major anaphylactic reaction.^{9, 10} The severity of the reaction is not predictable.

This case emphasizes the need to be alert when administering colloids as their use can lead to severe and possibly fatal complications. The previous administration of the same substance does not guarantee that it will be safely tolerated again since sensitization can always occur. The rate and risk of sensitization to gelatin or other possible allergens is still not precisely known and should be further analysed.

Conflict of interest

None declared.

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A case for routine tonometry to avoid postoperative visual loss

Editor—Visual loss due to central retinal artery occlusion (CRAO) is a rare but devastating complication after cardiac, spine, and orthopaedic procedures.¹ Despite CRAO occurring commonly secondary to external globe compression, spontaneous occurrence of CRAO has also been reported.² We encountered a case of CRAO during a low-risk abdominal procedure, without anaemia and not requiring blood transfusion.

An 81-yr-old man with a history of controlled open-angle glaucoma, macular degeneration, essential hypertension, and chronic renal disease underwent a laparoscopic nephrectomy and open ureterectomy for transitional cell carcinoma of the right ureter. During the procedure, the patient was positioned in the left semilateral decubitus position with kidney rest elevation and a 15° head-down tilt. Peak airway pressures were <35 cm of water during the entire procedure. P_{aCO_2} increased to 6.0–6.8 kPa during carbon dioxide insufflation. After 3 h, the surgeons elected to convert to an open procedure in the supine position. Mean arterial pressure was maintained within 20% of baseline. The blood loss was estimated to be 200 ml and the patient received 5000 ml of crystalloids. He underwent an uneventful recovery after extubation. At 23 h after operation, the patient noticed acute, painless loss of vision in the dependent eye. Preoperative haematocrit was 35% and on the morning after surgery was 29%. The ophthalmologist diagnosed CRAO based on an afferent pupillary defect and fundoscopic findings of attenuated arterioles, pale retina, and a cherry red spot. No emboli were seen in the retinal circulation. There were no obvious signs of external globe compression. Intraocular pressures (IOPs) were normal and equal in both eyes after operation. Topical travoprost drops and eyeball massage did not lead to improvement. Transthoracic echocardiogram, carotid duplex ultrasound, and temporal artery biopsy did not reveal an embolic or arteritic cause. Visual loss remained

permanent with perception of hand motion from a distance of 60 cm at 1 yr follow-up.

The risk of intraoperative CRAO is 1.54/10 000, slightly higher compared with the non-operative CRAO risk of 1/10 000.^{1–3} With a pre-existent glaucoma, our patient could have developed an intraoperative increase in IOP which may have led to this event. Factors which may have contributed to an increase in IOP in our patient include preoperative omission of IOP-lowering medication, the 15° head-down tilt, high airway pressure, the intraoperative hypercapnia, and the prolonged surgical time. In addition, an increase in intravascular fluid volume as in overzealous fluid replacements could also predispose to an increase in IOP. A common cause of CRAO is an embolus.³ Lack of demonstrable emboli during transoesophageal echocardiography (TOE) and Doppler studies does not rule out this as an aetiology.³

Despite not receiving transfusion, our patient's haematocrit did demonstrate a decrease of 6%.

In 70% of operative cases with CRAO, signs of trauma such as proptosis, corneal abrasion, hyphaema, and bruising have been reported.⁴ Our patient had a mild chemosis in the post-anaesthesia care unit (PACU). Involvement of the dependent eye might suggest a watershed-type lesion.

When the ocular perfusion pressure decreases, there is a compensatory increase in IOP or central venous pressure. Low ocular perfusion pressure is yet another reason for CRAO. IOP increase has been shown to occur with hypercapnia, laparoscopy, head down, and dependent eye position.^{5–7} Normal IOP in the PACU may not be of relevance in the prevention of CRAO. In our case, CRAO occurred after nephrectomy in the lateral decubitus position. Earlier detection may have been facilitated by routine intraoperative tonometry in selected patients who may be at risk for CRAO. What may need to be considered in the future is the role for routine intraoperative checking of IOP in selected patients.

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