Idiopathic facial paralysis following general anesthesia

Sir,

Idiopathic facial paralysis (IFP) or Bell’s palsy is a frequent neuropathy. The annual incidence is 15-30 patients per 100,000. The majority of patients have a complete recovery. Peripheral facial paralysis has been associated with several factors: trauma, tumor compression, infection (herpes simplex virus) and pontine lesions. Whenever no clear etiology is established, the peripheral facial paralysis is called IFP. Diagnosis is made on weakness of the muscles on one side of the face and inability to close eyelids totally. On attempting to close the eyelids, the eye rolls upward and outward (Bell’s phenomenon). Central nervous system lesions such as stroke or tumor can also cause facial nerve paralysis, but Bell’s phenomenon is absent. Postoperative facial paralysis due to the mechanical stress during general anesthesia (GA) has been described and is a rare complication attributed to direct compression or stretching of the nerve. Digital pressure behind the mandible or excessive pressure exerted by the facemask can cause a traumatic lesion of the facial nerve. Nevertheless, no case of IFP is reported after GA.

A 66-year-old man with chronic pancreatitis underwent a pancreatic duct stenting. The day before surgery, he presented a left retroauricular pain without fever. After preoxygenation, 3 mg/
kg of propofol and 0.2 mcg/kg of sufentanil were administered intravenously (IV) for induction of GA. The patient was ventilated twice by facemask and trachea immediately intubated. Anesthesia was maintained with nitrous oxide 50% in oxygen and sevoflurane. In postanesthesia care unit, a left peripheral facial paralysis was noted. An emergent brain computed tomography (CT) scan was normal. Oral prednisolone was introduced after neurologist’s assessment. The patient was discharged on day 2. One year after, hemifacial spasms and synkinesis were still observed.

A 47-year-old woman presented with a postcoital headache with loss of consciousness. A brain CT scan revealed a subarachnoid hemorrhage (ruptured basilar artery aneurysm). On emergency, an external ventricular drain was inserted and aneurysm was occluded by coiling. Patient’s trachea was extubated on day 10. Right labial herpes lesions appeared on day 21 and were treated by topical application of acyclovir. Five days after, a ventriculoperitoneal shunt was inserted because of a persistent hydrocephalus. Induction of GA was performed with propofol (2.5 mg/kg), sufentanil (0.3 mcg/kg) and cisatracurium (0.15 mg/kg) IV. The patient’s lungs were ventilated by facemask and trachea intubated without difficulty. Anesthesia was maintained with sevoflurane without nitrous oxide. After tracheal extubation, a right peripheral nerve palsy was noted. Intravenous acyclovir and methylprednisolone were administered for 10 days. Six months later, a severe facial palsy was still present.

These cases are highly suggestive of associations between IFP and GA. In demyelinating disorders, such as multiple sclerosis, GA has been implicated in causing disease exacerbation. However, no clear conclusion can be driven from these observations. In our cases, patients presented prodromic signs of IFP. The first patient had retroauricular pain and the second presented labial herpetic lesions. Nevertheless, even if both patients had prodromal symptoms that have been associated with IFP, it is just as likely that they might have gone on to develop IFP without GA. Facial paralysis has been associated with GA due to mask ventilation. We do not consider this a possible mechanism in these cases. Both patients were ventilated by mask for a few minutes at induction (and not during all the surgery), so this cannot be considered as a plausible mechanism. Moreover, it would be surprising that IFP appears in the same side as prodromic signs.

In patients with prodromic signs of IFP, GA may promote the apparition and increase the severity of IFP.

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