# Acute bilateral submandibular gland swelling during rigid oesophagoscopy under anaesthesia

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EDITOR:

Acute salivary gland enlargement is a rare complication of upper gastrointestinal endoscopy [1] and oesophageal stricture dilatation [2]. It is also seen occasionally during endotracheal [3] and laryngeal mask airway anaesthesia [4]. We would like to report a case of acute bilateral submandibular gland swelling during rigid oesophagoscopy under general anaesthesia.

A 23-yr-old male presented to the Department of Otorhinolaryngology with complete dysphagia following ingestion of alkali 6 months ago. A feeding jejunostomy had been carried out under general endotracheal anaesthesia 4 months earlier, which was uneventful. There was no history of any concomitant illness. The patient was thin built, weighing 50 kg, and the clinical examination did not reveal any abnormality. An oesophagoscopy was planned to evaluate the extent of the injury in order to plan the treatment.

This patient was scheduled for rigid oesophagoscopy under general anaesthesia on a day care basis. The patient was fasting overnight and did not receive any premedication. In the operating room, an intravenous (i.v.) access was established and monitoring was instituted. Anaesthesia was induced with fentanyl $2\,\mu g\,kg^{-1}$ and propofol $2\,mg\,kg^{-1}$ and muscle relaxation was achieved with succinylcholine $1.5\,mg\,kg^{-1}$. Forty-five seconds after administration of succinylcholine, the patient's trachea was intubated with 7.5 mm cuffed tube at the first attempt. Anaesthesia was maintained with 33% oxygen in nitrous oxide with isoflurane with manually assisted positive pressure ventilation. The patient was then positioned and the surgeon inserted a Macintosh laryngoscope and passed the rigid endoscope into the oesophagus. Three minutes later, bilateral submandibular gland swellings were noted. The swellings were firm and non-crepitant in nature. The oesophagoscopy revealed a stricture in the mid-oesophagus and was subsequently dilatated with gum elastic bougies. The procedure took about 7–10 min and, after removal of the laryngoscope, the swelling reduced to half its size. The patient's trachea was extubated after he resumed adequate spontaneous respiration and the patient remained asymptomatic with no signs of airway obstruction. The patient was observed in the recovery room and submandibular gland swellings disappeared within the hour.

Salivary gland enlargement during anaesthesia, although rare, seems to be associated more commonly with instrumentation of the trachea or use of laryngeal mask airway [3,4]. This phenomenon can also be seen with upper gastrointestinal endoscopy and stricture dilatation performed without anaesthesia [2]. Various mechanisms have been proposed to be involved in the acute enlargement of the salivary glands. Coughing and straining resulting in the venous congestion and enlargement of the glands, reflex stimulation of the parasympathetic system following airway manipulation leading to glandular hyperaemia and retention of saliva due to anticholinergic agents which results in inspissation of secretions are a few mechanisms described in the literature [5].

Our patient had no episode of coughing or straining and did not receive any anticholinergic agents. On the other hand, intense stimulation of the parasympathetic nerve supply can cause profuse secretion of watery saliva along with pronounced vasodilatation in the gland. The pharynx and the gastric end of the oesophagus has a rich nerve supply of vagal afferent nerve fibres, which is stimulated during oesophagoscopy [6]. Also, succinylcholine, which is parasympathomimetic in nature, could have had an additional effect. Since the salivary secretion is under neuronal control, it can lead to an acute increase in the production of saliva up to as high as $4\,mL\,min^{-1}$ in the submandibular gland [6]. Further, the application of constant pressure of the suspension laryngoscope may have resulted in the distortion of the anatomy of the submandibular gland [7] or led to the blockade of Wharton's duct which opens under the tongue. Acute increase in the production of saliva coupled with an obstruction to its outflow could have been responsible for the swelling of the submandibular salivary glands. This is probably also why the swelling was restricted to the submandibular glands and did not involve the parotid glands.

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Acute salivary gland swelling may occur following oesophagoscopy and stricture dilatation under general anaesthesia, which can be alarming for both the surgeon and the anaesthesiologist. Although this swelling is usually harmless and resolves without any residual complications, the possibility of its occurrence should be kept in mind.

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# Tourniquet-associated cardiac ischaemia in a healthy patient undergoing trauma hand surgery

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EDITOR:
Surgical tourniquet use is commonplace in orthopaedic, plastic and trauma theatres. Complications are well recognized. Here we describe an unusual and previously undescribed complication during hand surgery on a fit, healthy young male.

## Case report

A 25-yr-old male smoker (10 cigarettes per day) with no previous medical history and muscular body habitus underwent tendon and nerve repairs of his fourth and fifth right-hand digits following an accident with a knife. The patient was fasted and anaesthesia was induced with propofol, midazolam and fentanyl. A size 4 disposable laryngeal mask was inserted and anaesthesia maintained with isoflurane and nitrous oxide. Intravenous (i.v.) morphine was given (total of 10 mg over 4 h) along with diclofenac (75 mg) and paracetamol (1 g). The patient received 2 L of Hartmann's solution during the first 3 h in theatre.

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The tourniquet was deflated after 2 h at 250 mmHg. This coincided with a rise in end-tidal $CO_2$ and then, around 20 min following tourniquet deflation, ST segment depression was clearly noted on the ECG tracing in leads II and III. This progressed over the final hour of the operation such that gross ST segment depression was noted (up to 7.5 mm) in leads I, II and III and was associated with relative hypotension (mean arterial pressure (MAP) falling from around 70 to 55 mmHg). This was refractory to a further 1 L of Hartmann's solution and 1.5 L of Gelofusine. Ventilation was continued with oxygen and isoflurane at the onset of ST depression.

The patient was catheterized and a temperature probe was inserted (patient was normothermic throughout). On chest auscultation, air entry was equal bilaterally and breath sounds vesicular. I.V. nitrates were commenced cautiously (approximately 0.3 $\mu g\,kg^{-1}\,min^{-1}$) with no further drop in MAP and slight improvement in the electrocardiogram (ECG). Blood samples were sent for full blood count, urea and electrolytes. The patient was taken to theatre recovery for 12 lead ECG and further management.

In recovery, a 12 lead ECG confirmed global ST segment depression, and a cardiological opinion was sought. An echocardiogram was unremarkable and showed good left ventricular systolic function. The patient awoke not complaining of any symptoms

 