Surgical emphysema and pneumomediastinum in a child following minor blunt injury to the neck

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Summary

Laryngotracheal and pharyngoesophageal tears following minor blunt trauma to the neck are uncommon. A child with such an injury is reported and the modes of diagnosis and management are discussed. Patients may initially present with minimal signs and symptoms, but their condition may deteriorate rapidly or insidiously. In the absence of respiratory compromise, conservative management is appropriate, but all patients with significant blunt neck trauma should undergo early direct laryngoscopy under a general anaesthetic.

Keywords: blunt injury; neck; emphysema; pneumomediastinum

A 4-year-old girl fell forwards onto the backrest of a wooden chair, causing minor pain to the neck and upper chest. Although initially well, 4 hours later she was taken to Mayday Hospital with neck swelling and hoarseness. On examination there was marked surgical emphysema over the neck and chest extending to the sixth rib (figure 1). There was no bruising or stridor and the trachea was central. The larynx was

normal on palpation and there was no dysphagia. Oxygen saturations remained normal on air. A chest X-ray demonstrated a pneumomediastinum (figure 2).

The child was transferred to St George's Hospital where flexible fiberoptic laryngoscopy revealed moderate swelling of the supraglottis without pooling of saliva. A thoracic opinion was obtained and the clinical findings were suggested to be compatible with barotrauma to the oesophagus resulting in a pinhole perforation. The child was monitored on the paediatric ITU overnight and kept nil by mouth. The following day she remained well and afebrile. There was some resolution of the surgical emphysema and she was commenced on clear fluids and later a soft diet. She was subsequently discharged without complication.

Discussion

Minor blunt trauma to the neck and upper chest in children is common, but significant injury to the pharynx, larynx, trachea or oesophagus is unusual. However, cases of laryngotracheal and pharyngoesophageal tears from relatively minor blunt trauma have been documented in the literature.

Children have a proportionally large mandible and short neck in comparison with adults. In addition, a child's soft and pliable laryngeal and tracheal cartilages can probably sustain greater temporary force than the adolescent or adult airway, a child's non-calcified larynx being more likely to recoil to its original position without fracturing. Despite protective mechanisms, however, with the neck extended, a midline blow can exert excessive force on the larynx and trachea, crushing the trachea

Figure 1 Lateral soft-tissue X-ray of the neck showing surgical emphysema

Figure 2 Chest X-ray showing pneumomediastinum

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against the posterior vertebral column, with tearing of the posterior membranous wall of the trachea, resulting in air being forced into the retropharynx and mediastinum.¹

Cervical oesophageal perforation secondary to minor blunt trauma is extremely rare. Barotrauma resulting from a compressive thoracic injury is the proposed hypothesis for this injury. The rapid rise in oesophageal pressure can exceed the tensile strength of the oesophagus leading to perforation. The mechanism for barometric perforation of the distended, hollow viscus of the oesophagus has been well documented.²⁻⁴ An anatomic weakness at the hypopharyngeal-oesophageal junction (Killian's dehiscence) predisposes this area to perforation. With a blow or fall against the neck and upper chest, the upper airway may be sealed off at the hyoid level. Simultaneously the thoracic cavity is emptied of its inspired air. If these barometric forces exceed the bursting pressure of the hypopharyngeal-oesophageal junction, perforation results, with the compressed air from the lungs being forced through the ruptured pharynx into the fascial planes of the neck. The literature shows that most oesophageal perforations secondary to blunt trauma occur at this level.^{2-5,6}

The most common cause of acute laryngeal trauma in adults is blunt injury, especially from road traffic accidents, followed by sports injuries, falls and assaults, with victims of road traffic accidents usually presenting with the most severe trauma.^{7,8} In children, falls from bicycles and playground equipment-related accidents are the most common cause of blunt neck injuries.⁹ These generally insignificant injuries in children rarely result in serious harm and parents and healthcare workers sometimes discount early warning signs. Patients may present with minimal signs and symptoms, but their condition may deteriorate rapidly or insidiously. External signs of injury are usually minimal and local bruising or swelling may be absent. The child is assessed for airway compromise but often the only sign of significant upper aerodigestive tract injury will be the presence of surgical emphysema. A tear of the upper aerodigestive tract, somewhere in the larynx, trachea, hypopharynx or oesophagus, must be assumed in the presence of surgical emphysema.¹⁰ It should be noted that the site and extent of cervical surgical emphysema do not necessarily indicate the anatomical site of injury.

Learning points

- minor blunt cervical trauma can lead to significant laryngotracheal and pharyngoesophageal injury, although this is uncommon
- patients need to be properly assessed and monitored for airway compromise as their condition may deteriorate after presenting with a seemingly insignificant injury
- hoarseness, stridor, dyspnoea, dysphagia and odynophagia are important symptoms to look for
- surgical emphysema is often the only presenting sign
- patients presenting with a minor blunt cervical trauma should have a lateral soft-tissue neck X-ray and chest X-ray. This will disclose the presence of surgical emphysema, pneumomediastinum or pneumothorax
- conservative management is appropriate in the absence of respiratory compromise, but endoscopic assessment under a general anaesthetic needs to be undertaken if there is any suspicion of more serious injury

The most common symptoms of a laryngotracheal tear at presentation are hoarseness (including dysphonia and/or aphonia), stridor and surgical emphysema⁹ and of a pharyngoesophageal tear, dysphagia, odynophagia, dyspnoea, hoarseness and surgical emphysema.¹¹ It is important to exercise a high index of suspicion and consider a laryngotracheal or pharyngoesophageal tear in any child presenting with these symptoms following a seemingly minor blunt trauma to the neck or chest. A lateral soft-tissue neck X-ray and chest X-ray will disclose the presence of free retropharyngeal and subcutaneous air, pneumomediastinum or pneumothorax. A computed tomographic scan of the neck and chest and a contrast swallow are investigations which should also be considered to further define the extent of the injury. All patients with significant blunt neck trauma should undergo early and meticulous direct laryngoscopy under a general anaesthetic. Rigid oesophagoscopy and rigid bronchoscopy should also be considered. In the absence of respiratory compromise, conservative management with close nursing observation, preferably on a paediatric intensive care unit, is appropriate.

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Extrinsic cerebral venous sinus obstruction resulting in intracranial hypertension

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Summary

We report the case of a 70-year-old man reporting with headache and visual disturbances who was being treated for prostate cancer. Investigations showed him to have intracranial hypertension caused by venous sinus obstruction. Patients with metastatic disease and raised intracranial pressure in the absence of focal signs should be considered as possible cases of venous outflow obstruction.

Keywords: intracranial hypertension; venous sinus thrombosis; malignancy

A 70-year-old man presented with a 1-month history of progressive generalised headache, worse in the morning and on straining, and a 1-week history of visual obscurations. He was otherwise well, although he was taking goserelin for carcinoma of the prostate, diagnosed in 1994 on the basis of a raised prostate-specific antigen and malignant-feeling prostate. On examination visual acuity was decreased to 6/24 in the right eye and 6/36 in the left eye. Both fundi were severely papilloedematous. The remainder of the neurological and general examination was normal.

Baseline haematology and biochemistry, including calcium levels, were normal. Erythrocyte sedimentation rate was 12 mm/h. Serum

immunoglobulins and electrophoresis were normal. Cranial computed tomography showed midline expansion and sclerosis of the occipital bone. Magnetic resonance imaging (MRI) confirmed midline occipital enlargement with loss of normal diploic marrow signal (figure 1). Axial images showed extrinsic compression of the superior sagittal sinus superior to the torcula (figure 2) which was confirmed on venous magnetic resonance angiography (MRA) (figure 3). A bone scan showed several metastases including the lesion in the occiput. He proceeded to have a lumbar puncture to confirm the clinical diagnosis of intracranial hypertension, to treat it by removal of cerebrospinal fluid (CSF), and to look for an underlying cause, such as malignant meningitis. The opening CSF pressure was 37 cm. The CSF was acellular with a protein content of 0.23 g/dl. He was treated with high-dose dexamethasone and repeated lumbar punctures prior to radiotherapy to his occiput. His visual acuity returned to 6/6 with resolution of his headaches. He subsequently developed further headaches and underwent surgery. The malignant deposit was peeled off the dura with visible re-expansion of the underlying venous sinus. Histology was very suggestive, but not diagnostic of a prostatic metastasis. The patient's headaches promptly resolved.

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Figure 1 Sagittal short TE/short TR (T1 weighted) MRI showing midline expansion of the occipital bone (arrows). The normal high signal intensity marrow within the diploic space has been replaced by low signal intensity tissue

Figure 2 Transaxial long TE/long TR (T2 weighted) MRI at the level of the occipital lobes. There is extrinsic compression of the superior sagittal sinus flow void (arrow) by the midline occipital bony expansion