THE MANAGEMENT OF RESPIRATORY OBSTRUCTION SECONDARY TO ODONTOGENIC INFECTION – CASE REPORT

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SPREADING INFECTIONS within the fascial planes of the neck, with the ever present danger of laryngeal oedema, were frequently encountered in the pre-antibiotic era. The more severe classical cases of Ludwig's Angina are now rarely seen, due to improved dental care and widespread use of antibiotics. The clinician must, nevertheless, remain alert to the dangers of extension of odontogenic infection within the neck. The teeth most frequently involved are the mandibular second and third molars, the apices of which are in close proximity to the lingual plate of alveolar bone in the body of the mandible beneath the mylo-hyoid ridge. Infection originating at the apices of these teeth usually spreads in a lingual direction into the sub-mandibular space between the mylo-hyoid muscle and the deep investing cervical fascia. A virulent infection may not be contained there and the fascial planes will direct the spread in the line of least resistance, towards the lateral pharyngeal space where it may further extend upwards to the base of the skull, or down towards the larynx or mediastinum. Dependent drainage remains the basic surgical treatment for any collection of pus within the neck, and recognition of the presence and extent of respiratory obstruction is essential for the safe management of general anaesthesia. The following case report illustrates potential pitfalls in clinical evaluation, and the importance of radiological evidence of upper respiratory tract obstruction.

CASE REPORT

A 24-year-old male casual out-patient presented at the Dental Clinic complaining of increasing pain and swelling beneath the left lower jaw, beginning with toothache in this region three days previously. He experienced progressive difficulty in opening his mouth and swallowing. He had not eaten for 2 days, and on the morning of admission was unable to swallow liquids. There was no complaint of dyspnoea or stridor.

On examination he was pale and toxic, but alert, and held his head slightly forward and rotated to the right. Accessory muscles of respiration were not active, and exaggerated filling and emptying of neck veins was not seen. There was a tender, non-fluctuant oedematous swelling, increasing in firmness towards its centre, extending from the left angle of the mandible, beneath the jaw to the midline. The left submandibular lymph nodes were enlarged and tender. Trismus was present but not extreme; there was an opening of 2.5 cm in the incisor region. The floor of the mouth on the left side was tender, tense and raised. Although the teeth were difficult to examine closely, the left mandibular second molar tooth

was carious and quite mobile. During the dental examination it was noticed that the patient was very uncomfortable whilst reclining in the dental chair. He continually moved his head forward from the head rest. Upon inquiry it was found that he experienced difficulty in breathing with his head extended. The temperature was 40° C, the pulse 132 per minute, the respiration 28 per minute, and blood pressure 170/120 mm Hg.

Investigations included complete blood count, urinalysis, lateral and oblique radiographs of the mandible, A.P. and lateral radiographs of the neck. An urgent consultation with the Anaesthetist resulted in a request for laryngeal tomograms. During exposure of the lateral films, with the head extended, the patient again experienced dyspnoea, and the radiologist then first heard stridor.

The films of the mandible show considerable soft tissue swelling extending backwards to the region of the epiglottis, with spotty gas formation. The lateral films of the neck (Figure 1) show prevertebral soft tissue swelling extending from

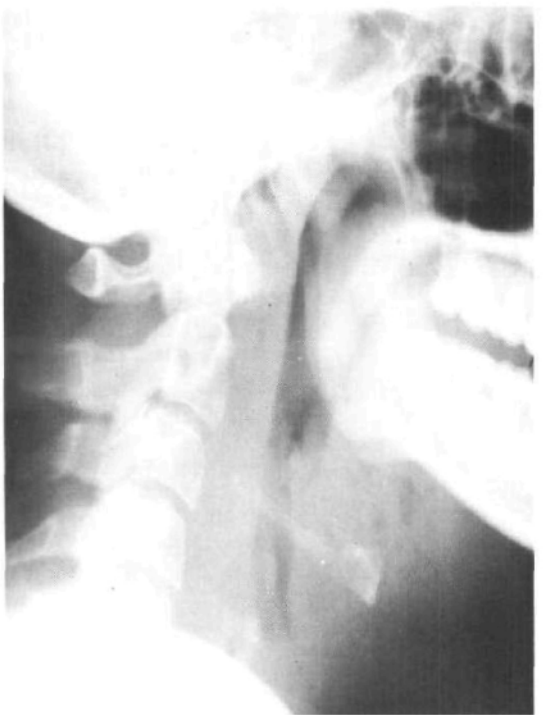

FIGURE 1

the base of the tongue to below the larynx, and pre-pharyngeal soft tissue swelling with displacement of the epiglottis and considerable narrowing of the entrance to the larynx. Tomograms of the neck, with particular attention to the larynx and trachea, show marked soft tissue swelling of the true and false vocal cords with obliteration of the valleculae. In none of the tomograms was the space between the swollen vocal cords more than 2 or 3 mm (Figure 2).

The diagnosis was made of acute submandibular abscess with inflammatory

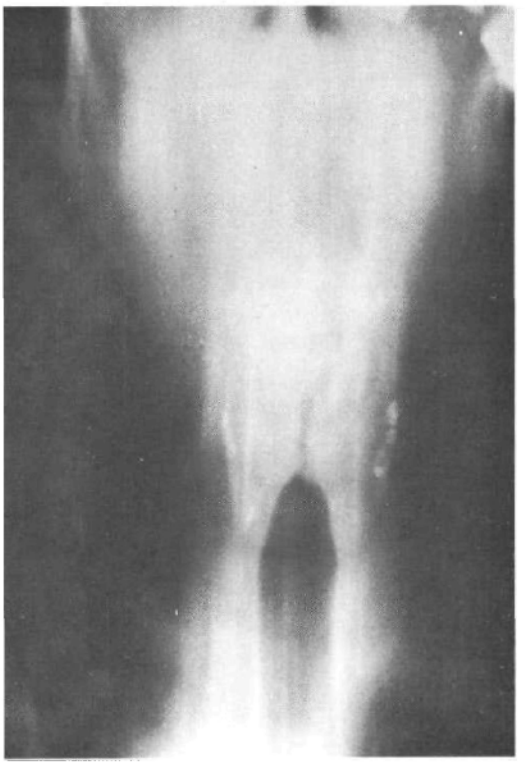

FIGURE 2

oedema of the larynx and respiratory obstruction. He was given Benzyl Penicillin 600 mgm by intra-muscular injection and prepared for incision and drainage of submandibular abscess and extraction of the infected tooth under general anaesthesia. After tracheostomy, performed under local anaesthesia, and insertion of a No. 9 cuffed James Tracheostomy tube, anaesthesia was induced with thiopentone and maintained with nitrous oxide, oxygen and halothane with spontaneous ventilation. The abscess was incised and drained, the infected tooth extracted, and recovery was uneventful.

Radiographic evaluation of the upper airway was repeated eight months later (Figures 3, 4, 5), showing the return to normal, a comparison between the effects of phonation and full inspiration on laryngeal aperture, and slight tracheal indentation at the healed tracheostome.

DISCUSSION

In patients with inflammatory conditions of the floor of the mouth, the possibility of airway obstruction must always be suspected. The fundamental consideration in the conduct of general anaesthesia in the presence of respiratory obstruction, is the establishment of a secure airway prior to induction.

This case illustrates that, in the adult, severe obstruction and distortion of the

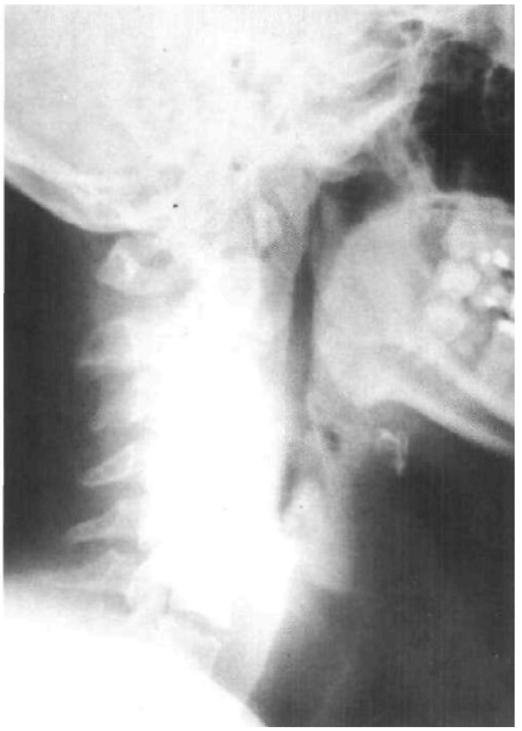

FIGURE 3

airway may be present without very obvious symptoms and signs. Dyspnoea, associated with increased work of breathing, and stridor, caused by soft tissue vibration, became apparent only with neck extension, when further slight narrowing of the airway caused previously predominantly laminar air flow to become turbulent.

With laminar flow in smooth tubes, the pressure gradient required to produce a given flow rate, is directly proportional to the flow rate. The tendency to turbulent flow is increased by geometric factors such as sharp angles and sudden changes in diameter, and is directly proportional to linear velocity of air flow, so that at a critical velocity, flow becomes entirely turbulent. When turbulent flow is established, the pressure gradient necessary to produce flow becomes proportional to the square of the flow rate. Considerably increased resistance to air flow is then associated with increased work of breathing and dyspnoea.

The respiratory pattern chosen by the patient represented a compromise between his ventilatory volume requirements, minimized at rest, and his need to maintain air flow below the critical rate. So long as the head was held in the most comfortable position, and an optimum respiratory pattern maintained, air flow remained below the critical flow rate at which turbulence occurs. Reduction of volume and rate of flow, associated with respiratory obstruction, is demonstrable by spirometry which, as accurate instruments become more portable, becomes more practical as a diagnostic tool.

Although arterial blood gas analysis was not performed, hypertension and tachy-

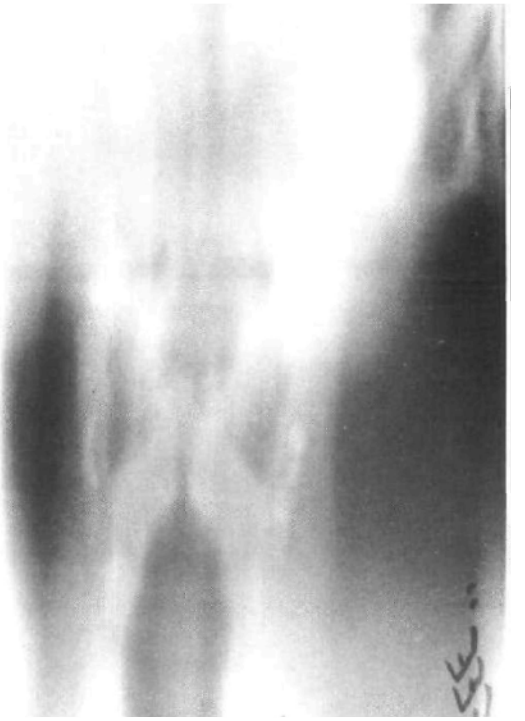

FIGURE 4

cardia suggest that there was respiratory acidosis, and that ventilation was not adequate. As ventilatory insufficiency increases in the presence of severe respiratory obstruction, hypoxic chemoreceptor drive becomes more important in maintaining respiration. Oxygen-helium mixtures are advocated in the treatment of severe respiratory obstruction, since resistance to turbulent gas flow is inversely proportional to density. One may speculate that with sudden improvement in oxygenation produced by such a mixture, ventilation may be further depressed, with exacerbation of respiratory acidosis and the danger of severe arrhythmias.

Dyspnoea and stridor on neck extension provided the first evidence of severe airway obstruction. Laryngeal tomography immediately revealed the extent of airway narrowing and distortion, and demonstrated the urgent need to establish a safe airway by tracheostomy under local anaesthesia, rather than by a risky attempt at blind awake nasotracheal intubation. A rational approach to the assessment of upper airway obstruction associated with inflammatory conditions of the floor of the mouth should include the following steps:

Clinical Assessment of Upper Airway Obstruction:

1. Vital signs: Temperature, Pulse, Blood Pressure, Respiration.
2. History: dysphagia, dysphonia, trismus, dysphoea on exertion.
3. Observation of the effects of flexion and extension of the head.
4. Observation of the effects of forced maximal inspiration and expiration.
5. Auscultation of the neck.

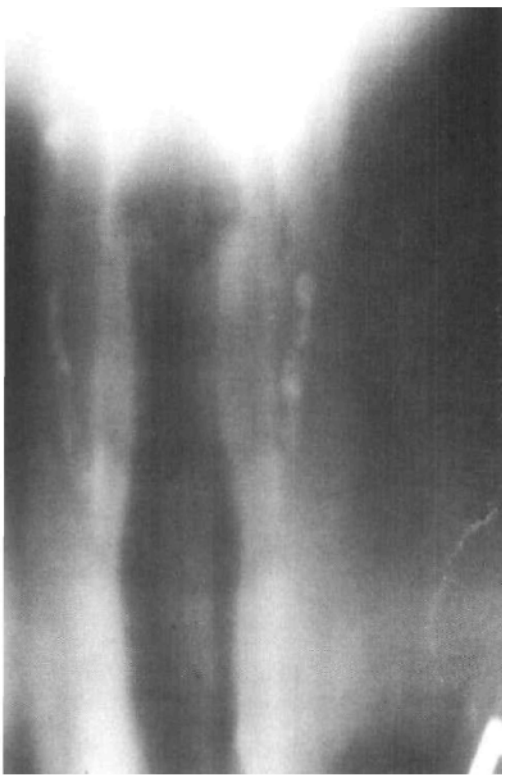

FIGURE 5

6. Dyspnoea, stridor, accessory muscle activity, and cyanosis are, in the adult, late signs indicating very severe obstruction.

Investigations in Upper Airway Obstruction:

1. Arterial blood gas analysis.
2. Spirometry.
3. Lateral radiographs of the neck and laryngeal tomography.

SUMMARY

A case of submandibular abscess with respiratory obstruction due to inflammatory oedema is presented, in which clinical evidence of respiratory obstruction was less than obvious, and in which radiographic evidence was important both in diagnosis and the correct choice of airway management.

RÉSUMÉ

Nous présentons un cas d'abcès sous-maxillaire avec obstruction respiratoire attribuable à l'œdème inflammatoire; l'évidence clinique d'obstruction respiratoire était moins que patente; l'évidence radiologique était importante aussi bien pour le diagnostic que pour le choix judicieux de la conduite à tenir pour libérer les voies respiratoires.