Medical Memoranda

Fracture of Thyroid Cartilage During a Sneezing Attack

Fractures of the laryngeal cartilages are not particularly rare, but the following case seems worth recording because of its aetiology.

CASE REPORT

A man aged 44 was seen in May, 1948, complaining of pain and swelling in the front of his neck, pain on swallowing, and a hoarse voice. Three days previously he had suffered a violent sneezing attack during which something clicked in his throat and was immediately followed by pain in the front of the neck. His voice became slightly hoarse two days after the sneezing attack.

On examination a swelling was noted over the thyroid cartilage with tenderness on palpation. A definite vertical fracture could be felt in the thyroid cartilage about $\frac{1}{2}$ in. (1.25 cm.) to the right of the midline, and the two edges moved across each other with a fine click. The movement was very slight and at least one layer of perichondrium was presumed to be intact. On indirect laryngoscopy the ventricular folds were seen to be reddened and very slightly swollen, but there was a good airway and the vocal cords moved normally. The mucosa was not torn and there was no distortion. The fracture line did not show up on an x-ray film and the laryngeal cartilages were not excessively calcified for a man of his age.

On admission to hospital strapping was applied across the front of his neck, from the hyoid bone above to $\frac{1}{2}$ in. inferior to the cricoid cartilage below. A tracheotomy set was ready at his bedside, but as the fracture was three days old and there was no marked oedema it was considered safe to try to avoid a tracheotomy, especially as qualified surgical assistance was constantly available on the premises. He was ordered not to use his voice. His diet was confined to fluids and semi-solids for the first three days; his voice was then normal and he found that he could chew and swallow solid food with very little discomfort. From then on he had a normal hospital diet, but voice-rest was continued.

After a week there was still a little tenderness over the thyroid cartilage, but no movement or click could be elicited. Six weeks after the sneezing attack there was very slight tenderness on firm pressure.

At the end of June, 1948, he had a further attack of sneezing. Once again his neck was swollen and tender, and the clicking and mobility could be felt as before. His voice remained normal. An x-ray film again failed to show up the fracture. The same treatment as before was followed by uninterrupted recovery.

Since then he has been perfectly comfortable, but admits to a little tenderness on firm pressure over the larynx. There is no mobility, and an x-ray film taken in June, 1949, again showed no fracture line.

COMMENT

I have been unable to find a report of any other case of fracture of the thyroid cartilage during a sneezing attack. How the fracture occurred has given rise to considerable surmise without any definite solution. The patient is sure that there was no direct trauma to the neck. He did shake his head violently backwards and forwards when sneezing. He was not wearing a collar, and his shirt was open at the neck without any front stud. The thyroid cartilage did not appear excessively calcified on the x-ray films.

The possibility of a congenital dehiscence in the thyroid cartilage has been considered—the cartilage developed in

the lateral mesodermal plate not completely uniting with that developed in the dense ventral mesoderm and therefore fracturing easily. However, in these circumstances I would expect to see a gap at some point in the cartilage showing up on the x-ray film, but I may be wrong.

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Mumps Meningo-Encephalitis

In view of the article by Macrae and Campbell (1949) we are prompted to record the following case.

CASE REPORT

The patient, a schoolboy aged 12, had a definite history of contact with mumps 18 days previously. Apart from his being very excitable and subject to periodic attacks of headache and vomiting during the past three years, there was nothing of significance in his medical history.

On March 4, 1949, he had malaise, vomiting, headache, and an unproductive cough. Next day he became pyrexial and photophobia was manifest. Twenty-four hours later all symptoms were intensified and trismus and dysphagia developed. Swelling of the left parotid gland first appeared on March 7 accompanied by mental confusion, delirium, and screaming attacks. He rapidly deteriorated to a state of semicomatose, and was admitted to hospital.

On examination his temperature was 102° F. (38.9° C.), pulse 96, and respirations 22. He was comatose. Enlargement of the left parotid gland, with trismus, made throat examination difficult. There was no evidence of exudate or membrane on the fauces, and no neck rigidity. Acetonuria was present. All tendon reflexes were increased, abdominal reflexes were absent, plantar reflexes were extensor both sides, and Kernig's sign was negative. Cranial nerve involvement was not noted. The abdomen was markedly scaphoid and a pronounced tache cérébrale was present.

A lumbar puncture was made under difficulties, as interference made the patient wildly delirious and resistive. The results of the cerebrospinal fluid examinations are shown in the table. Penicillin, 200,000 units, was given twice daily for four days.

Twenty-four hours after admission the right parotid became involved. The patient was now semicomatose and was able to open his mouth freely and to take fluids moderately well. Next day the improvement was maintained and he was able to carry out very simple instructions. There was, however, considerable slowing of reaction time and a marked slurring of speech. Papilloedema was not present in either eye and the retinæ were normal. By the next day his temperature, pulse, and respirations had returned to normal and consciousness was fully regained. There was, however, a continued delay in reaction time and the speech remained slurred. The condition of the parotids and the nervous system remained unchanged.

By March 11 the general and mental conditions were much improved and the parotid swelling had to a great extent subsided. There was evidence of a commencing left facial paralysis of an upper motor neurone type characterized by loss of the left naso-labial fold with deviation of the mouth to the right on grinning. Next day the left facial paralysis was well marked, but for the first time since admission both plantar reflexes gave a flexor response.

By March 14 the patient showed no symptoms or signs of a lesion of the nervous system, the only residual abnormality being slight induration of the left parotid below the ear. There was no clinical evidence of pancreatitis or orchitis. The