HERPES LARYNGIS

By D. A. DRAFFIN (Nottingham)

T.C., male, 46 years, was admitted to the General Hospital, Nottingham, in *February, 1950*, complaining of pain in the region of the larynx and upper left side of neck, dysphonia, complete dysphagia and general malaise.

History

1st day. On this morning he went to work feeling quite fit; but a few hours later he was seized by a severe pain, gripping in character, related to the larynx mainly, but with twinges shooting to the left ear and left upper neck. It reached its peak of intensity in about half an hour's time and was somewhat easier when admitted 24 hours later. From his story it appeared almost as if a sharp foreign body had lodged somewhere at the back of the throat.

2nd day after admission. *On examination* the epiglottis was acutely inflamed and grossly enlarged, being easily visible on depression of the tongue with a spatula. The mucosa of the mouth, tongue, fauces, palate and upper pharynx was quite normal. On indirect laryngoscopy only the mucosa on the posterior surfaces of the arytenoids could not be visualized in view of the swelling of the epiglottis. Saliva pooled in the hypopharynx, and since the patient was unable to expectorate satisfactorily no worthy opinion could be given of the state of affairs below the epiglottis.

T. 100° F. P. 90. No diagnosis as yet made.

Respiration was not sufficiently embarrassed to require tracheostomy but in view of the apparent infection and obvious œdema penicillin 100,000 units 3-hourly was given.

3rd day from onset. Pain somewhat less but dysphagia still complete. Epiglottis more swollen still. Airway still sufficient.

T. 100·4. P. 95.

4th day. Pain much less, dysphagia not so marked, speech easier. Epiglottis less swollen.

5th day. All symptoms less marked.

On direct examination the epiglottis appears pear-shaped with base pointing to the left. It is much smaller and a good view could be obtained of the larynx for the first time. There is a superficial ulcer 4×4 mm. in diameter with a yellow base situated on the left laryngeal surface of the epiglottis towards the tip. The mucosa over the left arytenoid is swollen as is the ary-epiglottic fold, the false vocal fold, and the mucosa in pyriform fossa.

The true vocal fold remained sublimely indifferent to these local changes, neither colour nor mobility being affected in any way.

The diagnosis of herpes was now made. No contact admitted with case of chicken-pox.

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6th day. A few vesicles are now clearly visible on the lower laryngeal surface of epiglottis but nowhere crossing the middle line. A few small ulcers with same distribution. No vesiculation or ulceration was at any time seen elsewhere on the larynx.

7th day from onset. Slight pain only on swallowing now.

T. and P. normal.

All ulcers on epiglottis healing rapidly.

Mucosal swelling diminishing.

Penicillin still continued.

No vesicles or congestion appeared at any time in region of the auricle. No facial paresis. No tinnitus or vertigo. Audiogram normal. Posterior surface of soft palate and post-nasal space and nose normal at all times. Palatal arches symmetrical. No paralysis. No loss of sensation to pin prick or touch detected on the buccal surfaces or tongue, or on the tonsils, faucial pillars or pharyngeal wall. Sterno-mastoid and trapezius no paresis. W.R. negative. Kahn negative.

11th day. Swallowing well. Slight residual swelling of affected mucosa. Home.

One month and three months from beginning of illness, no symptoms, larynx normal.

Discussion

The annoying feature about this case is that I failed to make a diagnosis until the fourth day after admission when the swollen epiglottis subsided and a small ulcer was seen. The appearance of a few vesicles on the same surface the following day made the diagnosis more certain. The unilateral distribution of the mucosal changes completes the picture. Daily laryngeal examination showed how rapidly vesicles could rupture and disappear entirely; this was probably due to the prevention of secondary infection by penicillin.

The pain in the ear was assumed to be referred through the auricular branch of the vagus as no congestion or vesiculation at any time appeared.

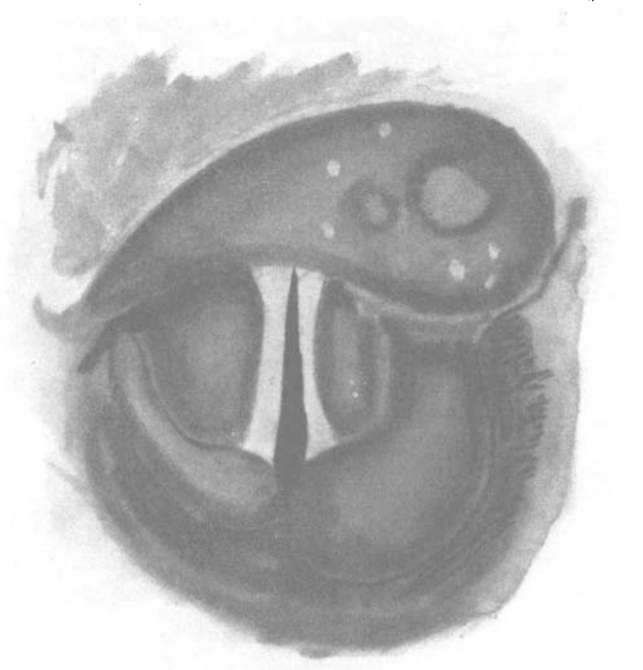
Before attempting to localize the lesion to the inferior ganglion of the vagus, it will repay to examine the evidence that such localization can occur to vagal ganglia. The agent, probably a variant of the chicken-pox virus, usually picks out more than one ganglion, and vesicles in due course are commonly placed in a convenient position for all to see. Sometimes it picks out a sensory cell station here and there, and sometimes a motor group, e.g. the VIth cranial.

The auricular branch of the vagus has a variable area of distribution on both surfaces of the auricle and extending back into the mastoid process. The first three cases described by Hunt in the *Archives of Internal Medicine*, 1910, had a vesicular eruption limited to these areas but without facial paralysis (or paresis). He accordingly placed the site of invasion on the superior ganglion of the vagus.

In other cases where facial paralysis supervened, the eruption was more anteriorly in the concha and external auditory meatus, suggesting geniculate involvement.

Can one place an acute inflammatory lesion in the inferior ganglion of the vagus and avoid a recurrent paralysis? Two points are worthy of consideration

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in this respect. The first that the recurrent fibres on their way from nucleus ambiguus travel in the cerebral portion of the XIth cranial nerve (Wedell quoted by Negus), and thus they will not be greatly exposed to pressure if they do not enter the vagus until just below the inferior ganglion (Chevalier Jackson). No doubt slight variations do occur in the level of entry.

The second point, motor paralysis associated with herpetic phenomena is often short-lived where the motor cells are not themselves affected (Dan McKenzie and others) and in the case under review a transient motor paralysis could not be excluded in view of delay in visualizing the larynx.

Finally, the failure of the mucosa over the true vocal fold to share in the neighbouring congestion, either casts doubt on its true nerve supply or suggests ability to resist the full vigour of an herpetic onslaught.

Dan McKenzie, Negus and Crabtree, Bateman and Mawson all point out the rarity of references in the literature to lesions localized to the vagus. Out of the 76 cases reviewed by Hunt in 1910 three other cases in addition to those already mentioned of Sheff, Spephanow, and Boulai seem to have been a close parallel to the ones here described and to Bateman and Mawson's case, but in mine the epiglottis stole the limelight.

Summary

A case of unilateral Herpes Laryngis affecting the vagus nerve only without observed vocal cord paralysis or vesiculation on the auricle.

The difficulty in its early diagnosis and the rarity of references in the literature to such localized lesions stimulated are recorded.

I extend my thanks to Mr. Gilroy Glass for his permission to publish this record.

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