BULLOUS OTITIS EXTERNA

By J. V. CLARK

BULLOUS or hæmorrhagic otitis externa is characterized by a hæmorrhagic effusion into the subepithelial tissues of the osseous meatus and drum-head itself. Hæmorrhagic areas appear as bluish swellings of varying size, which are soft to the touch. The vesicles normally subside within a few days, but others may arise to take their place. In most textbooks they are stated to be limited to the inferior and posterior walls of the meatus, but it would seem they can occur anywhere in the deep meatus and outer surface of the drum-head, although the drum-head often remains normal throughout.

The degree of associated tissue reaction is variable. In severe cases, some degree of periosteal reaction is not uncommon, as evidenced by the presence of mastoid tenderness. Occasionally, a serious effusion may develop in the middle ear.

With an associated upper respiratory infection secondary invasion may take place up the eustachian tube, and set up a true inflammation in the middle-ear cleft, with possible mastoiditis.

Pain is the commonest symptom, and, though of varying degree, this can be very severe.

In one series of cases observed, bleeding from the ear was a feature, due to rupture of one of the bullae.

The hearing is not affected in the early stages, and deafness is only noted if the meatal lumen becomes occluded by the hæmorrhagic swellings, or if associated tissue reaction is severe enough for effusion to develop in the middle ear.

The degree of constitutional disturbance is often marked, but it may be negligible.

Clinically, the close relationship that bullous lesions of the external auditory meatus bear to influenza has long been recognized, but, while most cases seem to occur within a few days of the onset of a feverish illness of an influenzal type, others show no constitutional disturbance whatever.

While in charge of an E.N.T. Centre in India during the War, I recorded a short series of bullous otitis externa, amongst 525 cases of common types. These cases presented within two months of one another, and I concluded that this was a seasonal epidemic : but though I was in India for two years, I saw no more cases of this type.

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Features of interest were the sudden onset of agonizing pain, the early rupture of the bullae which resulted in such free bleeding that it ran down the cheek, and, finally, the absence of constitutional disturbance—these patients were *not* ill. In this country, I have noted bullous lesions in the absence of constitutional disturbance after swimming, and recently two cases on the tympano-meatal flap in the second week after a fenestration operation. In none of these cases did the patients suffer from symptoms of typical influenza.

Bacteriological investigation of 23 cases of bullous otitis externa observed have failed to reveal any specific organism—in fact, perhaps significantly, they have generally failed to reveal any accepted pathogenic organism at all. Cultures for examination were taken by pricking the bullae with a needle and dipping this straight into broth, plus a swab from the meatus itself.

Total and Differential White Counts have been within normal limits.

Mastoid X-rays in those cases with mastoid tenderness, previously mentioned as an indication of periosteal reaction, were normal.

This evidence would seem to point to the possibility that this type of tissue reaction is due to a virus infection.

The fact that a complicating otitis media or mastoiditis gives a culture of Streptococcus, Pneumococcus or Staphylococcus is probably evidence that this is the organism responsible for the secondary infection, and not for the primary.

As a result of the work of Laidlaw published in 1933, it has been established that influenza is due to a virus infection. Two types, A and B, are now recognized, and a third is suspected. More than this, variants of each type would appear to exist.

Symptoms of patients suffering from proven influenza can be extremely variable, even though the virus type is constant. The fact that negative evidence suggests that bullous otitis externa is of virus origin, and so many cases seen in this country occur during the course of influenza, makes one suspect the possibility that the causative virus in all cases of bullous lesions *is* that of influenza.

The suggestion that otitis media or mastoiditis being due to recognized pyogenic bacteria is probably secondary, is the exact opposite of the views expressed in most text-books. The most recent edition of Ballenger, for example, states that otitis externa hæmorrhagica is " most probably a *complication* of influenzal otitis media " and yet also follows by saying " the hearing would not be affected unless the canal is swollen shut ".

Normal treatment advocated is to incise the bullae and evacuate the content. Once bullae have developed, they do not seem to increase in size, and as their content in all my cases proved sterile, deliberate incision hardly seems justified, and may even increase the risk of secondary infection. Chemotherapy does not appear to help, and if the virus

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theory is correct, could not help, although in the presence of an acute upper respiratory infection it may be justified as a prophylactic measure designed to avoid secondary infection spreading into the middle-ear cleft. It would seem, therefore, that there is no treatment other than measures designed to avoid secondary infection.

It has not been possible for me to prove these suggestions, as normal hospital laboratories cannot undertake this work. The cases observed by me have been scattered, and recorded over several years, and opportunity has not offered to get help from a research unit.

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