

ERYSIPELAS AS A COMPLICATION OF MASTOID DISEASE.*

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In 1907, Dr. C. R. Holmes, of Cincinnati, published a most elaborate paper on the etiology of erysipelas and its relation to the nasal cavities. This paper was based on a comprehensive study of the literature of the infectious organisms present in the upper air tract, supplemented by a large number of clinical reports, both published and hitherto unpublished, showing the presumptive relation of facial erysipelas to infectious diseases of that portion of the mucous membrane of the upper air tract which has continuity of surface; namely, the nasal passages with their accessory cavities, Eustachian tubes, mastoid cells, the fronto-nasal ducts and conjunctivae. These studies show that streptococci are nearly always present somewhere in this region, their pathogenicity varying considerable; in some cases largely losing their naturally virulent character, but probably becoming more virulent when planted on more suitable soil. In other words, the individual becomes practically immune to them at the site of their original location but is not immune to the same streptococcus when transplanted to another place, and when the resisting power of the individual is for any cause reduced.

Dr. Holmes recorded 102 cases of facial erysipelas occurring in connection with disease of the nose and accessory sinuses or following upon acute and chronic mastoid disease. These cases occurred in the practice of 69 American surgeons. They do not include those cases of the same character gathered from the literature. His conclusions were that the identity of the streptococcus as the microbial cause of erysipelas seems now to be a definitely established fact. This streptococcus is of slow growth and of variable morphology and pathogenicity whenever its environment is changed. It is probably but rarely air-borne, requiring direct personal contact for its transference.

The streptococcus is of nearly constant presence in the normal nose and neighboring spaces. It may be latent for months or years without betraying its presence, but this quiet existence may

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be broken at any moment by some temporary or unknown cause, inducing virulence in the microorganism through some unfavorable condition supplying the environment necessary to incite the organism into virulence, with the attendant formation of toxins. The extreme frequency of facial erysipelas has been noted for many generations, and Dr. Holmes' studies showed an apparently causative relationship between facial erysipelas and suppuration in the regions studied. Even in those cases in which no bacteriological examination was made, there was sufficient presumptive evidence to assume that the erysipelas-producing streptococcus was probably present in the discharges of the disease which was under treatment at the time of the appearance of the erysipelas. These cases of erysipelas occurred in the hands of careful surgeons, were not due to neglect, occurred in hospitals some of which were new, in others where no cases of erysipelas had occurred for many months, and unless some such explanation as this satisfies, the cause must be regarded as unknown.

In most of the cases reported from the statistics gathered by Dr. Holmes, the erysipelas does not seem to have had any serious effect upon the progress of the original disease. There were 12 deaths, but only in 5 could it be said in fairness that the erysipelas was the cause of the fatal result.

Observations of substantially the same character as those of Dr. Holmes are also made by Welty in an article on "Acute and Chronic Suppuration of the Ear and Nose as the Direct Cause of Facial Erysipelas," presented before the Section on Laryngology and Otology of the American Medical Association in June, 1906.

In addition Welty thinks that epidemics of erysipelas are nothing more than direct wound infection carried by surgeons or nurses, and that the so-called idiopathic erysipelas is a misnomer. The fact that erysipelas does not occur in every case of streptococcus infection is due to the character of the streptococcus and the immunity of the patient. Assuming these observations to be correct, the practical conclusions would seem to be:

1. The occurrence of a case of facial erysipelas in a hospital ward is not of necessity any reflection on the surgeon or the hospital, as it arises in nearly every primary case as an auto-infection.
2. Erysipelas is probably much less contagious by direct contact than has been supposed and should therefore be less feared; nor is there much likelihood of an epidemic of erysipelas occurring from a single case of erysipelas, provided the infection is not transmitted by the attendants, instruments, dishes, etc.

3. As a routine measure in all operations on the mucous membrane of the upper air tract it would be advisable before operation to cleanse the nose as carefully as possible, though it is by no means asserted that thereby every streptococcus or pneumococcus germ can be destroyed.

The three cases now to be described I offer as a contribution to the clinical history of facial erysipelas and as substantiating the foregoing conclusions. This point of view of the general etiology of erysipelas does not seem to have been as yet sufficiently grasped by the general surgical profession. In a very recent article on erysipelas in a general medical encyclopedia, no mention is made of this theory of the causation of facial erysipelas.

Case I. M. J. D., 55 years of age, though looking much older: slight tendency to alcoholism; otherwise, general health good. First seen, April 5, 1908, complaining of otitic media; next day incised the drum; following day, ear discharging freely, some mastoid tenderness; noticed for first time erysipelas blush at the outer angle of the left nostril. This slowly involved the entire left side of the face, reaching the region of the mastoid last of all, extending from this point backward to the mastoid and the corresponding side of the neck, temperature going to $104\frac{1}{2}$ and becoming normal on April 15. The erysipelas lasted about 10 days, the blush disappearing in about a week.

By April 29, the erysipelas had completely cleared up, the ear was discharging, there was considerable loss of tissue of the membrana tympani. During all this time there had been mastoid tenderness. On May 7, a thorough operation for acute mastoiditis was done. The mastoid was very large and very cellular, and there was a purulent debris in every cell. Recovery slowly took place, the last filling-in not being completed until June, owing to the presence of a small bit of carious bone tissue corresponding to the roof of the antrum cavity. This was finally curetted away, uncovering the dura for a short space, after which the cavity promptly filled in.

Now as to the relation of cause and effect. At the time of the advent of the erysipelas no one connected with the case had had anything to do with a case of erysipelas for months. The patient was at his own home, so there could be no hospital infection. It seems to me probable that the patient with his own fingers carried the infection from the ear to the corner of the nose, probably with the finger nail. This is the more likely as he was inclined to feel

around his ear with his fingers, and at first the ear was only wicked and not bandaged. I do not think the erysipelas caused the mastoid, as I was suspicious of beginning mastoid before the erysipelas began. The patient and his family physician were both loth to have the mastoid operation done, and he was out and about and came to my office after the subsidence of the erysipelas; the discharge and the mastoid tenderness continuing until the operation was finally done. The erysipelas was due to direct infection from the purulent discharge of the middle ear.

No bacteriological examination of the discharge was made, but on account of the extensive destruction of the mastoid and the subsequent slow recovery, the presumption is all in favor of the streptococcus infection.

Case 2. T. P. S., a dentist, aged 33, was operated on at St. Anne's Hospital for an acute mastoid on February 23, 1908. The operation had been delayed, owing to failure of the patient to give his consent, and the mastoid, which was large, was very much broken down, every cell in every direction being involved. After he had begun to leave the hospital and come to my office for treatment, but while the posterior wound, especially in the attic region, was still open, he developed about April 1, a facial erysipelas, invading the entire left side of the face, front and back, and going nearly to the vertex. It ran the usual course of an erysipelas, seeming in no way to interfere with the healing process of the mastoid. There was no other case of erysipelas in the hospital, nor was there any reason to suspect that either the surgeon or any attendant had carried the disease to the patient. Unfortunately, no bacteriological examination of the ear secretions were made; but from the intense degree of infection and tedious convalescence, it is probable that the case was one of streptococcus infection from the ear secretions. Recovery from the erysipelas took place after the usual time and the mastoid wound healed perfectly.

Case 3. Captain H., aged 65, retired officer of the United States revenue cutter service, had suffered for many years from an empyema of the right antrum. On January 22, 1910, an operation was done through the naso-antral wall, a portion of which was cut away so as to give permanent drainage. The after-treatment of this was uneventful for the first few days, and the patient was up and about, coming to the office for treatment. On January 31, nine days later, he developed facial erysipelas in the same side from an abrasion at the angle of the nose. The erysipelas was at

first on this one side, then involved the whole face. This ran the usual course and in the convalescent stage he seemed to acquire a general streptococcus infection and was quite sick for a number of days. No case of facial erysipelas had been recently seen, either by his family physician or myself. The amount of pus in the antrum was small, but there is little doubt but that the infection came from the nasal or antral secretions.

A coincidence, and perhaps in the nature of cause and effect though I am by no means certain of it, is the fact that my office assistant, who cleaned up the instruments after an examination of the nose at the time that this case of erysipelas was in the early active stage, herself developed erysipelas of the face 9 days later. The physician who attended her, however, did not believe that she got the infection from this source, as he had under treatment two or three cases of sporadic erysipelas at the same time. She left work complaining of fever and rheumatism, the erysipelas developing two or three days later. The disease ran the usual course and she was not very ill.

These cases are offered as a contribution to the subject of the etiology of facial erysipelas. The possibility of its occurrence after operations or infection of the upper air tract and adjacent communicating regions is to be borne in mind, though its occurrence under such conditions may be unavoidable.

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Scab Formation in the Nose. W. P. PORCHER, *Jour. A. M. A.*, Aug. 13, 1910.

Porcher recommends the use of potassium iodide in atrophic rhinitis, as it opens the accessory sinuses, gives free outlet to all inflammatory exudates, and produces lacrimation. It must be given in increasing doses, varying according to the individuals. Some patients absorbed 600 to 900 grains a day before the nasal secretions remained fluid, yet no painful effects were noted from these large doses.—Ed.