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A CASE OF ORBITAL CELLULITIS AND PRIMARY MASTOIDITIS INTERNA COMPLICATING INFLUENZA; OPENING OF MASTOID PROCESS; RECOVERY.

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IN the following I beg leave to communicate an ocular and an aural complication of influenza, which, even if by too great a skepticism their connection with the latter should be doubted, are sufficiently interesting in themselves on account of their rather rare occurrence.

History: The patient, a girl of ten years, moderately well nourished, had as an infant blennorrhœa neonatorum. In December, 1888, I saw her first on account of adenoid vegetations in her naso-pharynx, which I removed with Gottstein's knife, and subsequent cauterizations with chromic acid, according to Hering's[1] method, with lasting good result. On March 28, 1891, I was called in consultation with Dr. L. Reinhard to see her at her residence for an inflammation of her left eye. Her family physician stated that she fell sick two days previously with the symptoms of influenza, especially severe headache and general nervous prostration.

Status Præsens: She is lying in bed. The left upper lid, in a state of ptosis, is hanging over the eye and cannot be raised to its normal position. It is slightly red and swollen. The palpebral conjunctiva shows only an inconsiderable increase of redness, no swelling, looks as if affected with a commencing catarrhal inflammation, and is partly covered with some threads of mucous discharge, which however is very scanty. The ocular conjunctiva is not chemotic, only somewhat hyperæmic. Cornea clear.

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Pupil reacting to light perfectly well and equally with that of the other eye. But there is a moderate exophthalmus with displacement of the left eye downwards and towards the temple. The movements of the eye are impaired, especially inwards and upwards. When looking upwards and inwards, crossed diplopia sets in. Tension the same as in the healthy eye. Pressure on the globe is painful, but the finger cannot feel any hardness between the walls of the orbit and the bulbus. Especially at the upper temporal region no circumscribed swelling, so that an affection of the lachrymal gland can be excluded. V and field of V normal. The ophthalmoscopic examination reveals a normal disc, but a marked tortuosity of the retinal veins, which might have been taken for a sign of retinal congestion, had not the other eye exhibited the same appearance, which, as it has not changed until now—at the same time causing not the least functional trouble,—seems to be an individual peculiarity of vascular arrangement. The tongue is thickly coated and has a strawberry-like aspect, as seen in scarlet fever. Both tonsils are very much swollen and painful, but are not covered with pseudo-membranes. The skin of the whole body shows an erythema of a scarlatinous redness. No œdema. No albuminuria. The fever, which at first has been very high, is now moderate. I made the diagnosis *incipient orbital cellulitis*, and ordered iced applications, to be applied day and night. The next morning I found about the same condition. Under this rigorous antiphlogosis, which was carried out most faithfully, the inflammation gradually subsided, so that, when I was called again on April 3d, the swelling of the upper lid was diminished and the child could partly open her eye. There was hardly any discharge. Exophthalmus and displacement downwards and towards the temple, divergent strabismus in looking to the right, and difficulty and limitation in movement upwards and inwards, and diplopia persisted. The swelling of tonsils had decreased also. But another ailment had set in, a violent pain in left mastoid process, which was increased by the touch with the finger. The external meatus plugged by epidermic scales, so that *Mt* could not be seen. Ord. ice-bag on mastoid process, instillation of natr. carbon. into the meatus, and calomel internally.

April 4th.—Some of the impacted epidermis removed by syringing. Walls of the external meatus swollen, preventing a view at the *Mt.* The child could stand the ice-bag very well, which

relieved her greatly. The sensitiveness of the mastoid considerably diminished. She slept well. No headache.

April 5th.—The patient feels very much better. Mastoid process hardly painful any more upon pressure. Epidermic plates in meatus still, but no pus. Eye affection the same, though less in degree. Tonsils still swollen, the left side of the soft palate cannot be raised so well as the right side (Loewenstein [11] observed the same). Iced applications on eye and ice-bag on mastoid continued, and calomel. I did not visit the child any more until April 16th, since the pain had disappeared, and she felt so well that she could leave the bed. On April 16th I was sent for again, the pain in left mastoid process having begun anew the night before last. The day before she felt better, was out of bed, and played. But last night the pain became exceedingly severe, radiating over the neck, head, and left shoulder. Pressure on the mastoid is very painful. The walls of meatus swollen, but not red. *Mt* cannot be seen on account of the swelling and some remaining epidermic scales, which resisted all efforts of removing, the child being very nervous. But the auditory canal is perfectly dry, does not contain the least discharge. Exophthalmus gone, but still slight ptosis and crossed double images, when looking to the right and upwards. Pupils acting normally. Fundus, especially optic disc in both eyes normal, the retinal veins exhibiting the above-mentioned tortuosity. V not affected. Temperature slightly raised. Pulse rapid and small. Child is put to bed. Mastoid and neck brushed with tinct. iod., and ice-bag applied to mastoid. Internally calomel 0.30 dos. iii. Temperature at 5 P.M., 38.4° C.; at 8 P.M., 38.4° C.; *April 17th*, Temp. at 6 A.M., 36.10° C. She slept pretty well, but could not stand any more the pressure of the ice-bag. I ordered warm-water dressings and tinct. iodi applied twice a day. The integuments of mastoid presented not the least swelling or redness, so that there was no sign of a commencing periostitis externa. But the severe localized *pain*, the *fever*, and the *sickly appearance* of the patient, who looked rather pale, led me to believe, that there was an *acute mastoiditis interna* going on, and that the inflammation of the lining membrane of the mastoid cells might have turned into suppuration already, or at least was threatening to do so. Therefore I prepared the parents for an eventual opening of the mastoid process on the next day. Temp. at 8 P.M., 38.° C. (in axilla).

April 18th.—The condition of the patient being the same as yesterday (temp. 38,° at 6 A.M.), I thought it unsafe to wait any longer with the operation, for which I had everything ready. In chloroform-narcosis and under strictest aseptic and antiseptic precautions I made a large incision down to the bone. No ligatures were required. The periosteum and the external surface of the bone were healthy. The bone was opened with chisel and mallet. Corticalis pretty thick. When the antrum was reached the bone was chiselled off in such an extension, that an opening of a little more than 1 *cm* in diameter was obtained. The mastoid cells were found to be completely filled with red spongy fungoid granulation tissue, which was scraped off with Volkmann's sharp spoon. After thorough scraping and irrigation with sublimate solution 1 : 2000 a tampon of iodoform gauze with plenty of iodoform powder was introduced into the cavity and tightly packed. The external wound was not stitched. The meatus was also filled with iodoform gauze, and then a thick layer of iodoform gauze and carbolated absorbent cotton was fastened by sterilized bandages, encircling the head only, leaving the lower jaw free. The operation was performed at 8 A.M. Temperature at 6 P.M. normal. No pain. The child feels easy, sitting up in bed. The pain never returned after the operation, and the highest temperature was 37.5 ° C. The first change of dressing was made April 27th, on the 9th day after the operation. There was very little secretion in the iodoform gauze. No pain at all in the wound. Walls of the external meatus still swollen but not painful, and no discharge in its gauze. The swelling was gone at the next change of dressing, a week later. The dressing was renewed once a week until July 6th, when the very small tampon was left off. July 17th the wound was perfectly healed, exhibiting a depressed funnel-shaped scar. Hearing normal. *Mt* of normal appearance. Since the operation the child's general condition had improved wonderfully.

When I first saw the patient I had the impression that her disease was scarlet fever. The scarlatinous exanthema, the severe angina tonsillaris, the strawberry tongue, and the fever formed the well-known aspect of this disease. But the sudden attack of the previously healthy child, the rapid development of these symptoms with nervous prostration, and the subsidence of the exanthema after two

days, without desquamation later on, corroborated in my mind the diagnosis of the well-experienced family physician who had seen the sickness from the first day. *Exanthemata* in influenza, especially scarlatinous erythema, are described by a number of observers. Fraentzel[2] relates two cases of influenza in two children, which he at first took for scarlet fever, on account of the erythema, but found this diagnosis erroneous from the short duration of the redness and the absence of subsequent desquamation. Leyden,[3] Ewald,[4] P. Guttmann,[5] Renvers,[6] Riess,[7] Schwimmer,[8] Duflocq,[9] report similar cases, and Antonin[10] distinguishes a special form of influenza, which commences with an exanthema. *Pharyngitis* and *angina tonsillaris* are considered also as very often associated with influenza by Leyden, Renvers, Fraenkel,[11] Glower.[12] Loewenstein[13] observed it in all his 78 cases of influenza from the very commencement. Since, from these considerations, there could be no doubt any more that we had a case of influenza, the next question arose, whether the affection of the left *eye* was in connection with the general disease. There being no local cause for the *orbital cellulitis*, I thought it to be of metastatic nature, induced by the general infection. It seemed to be analogous to orbital cellulitis or orbital phlegmon observed in other infectious diseases, as in glanders, anthrax, typhoid fever, variola, and scarlatina (Berlin,[14] Schweigger[15]). In the last two years several observations of similar eye affections due to influenza have been published, wavering, however, between the diagnosis *orbital cellulitis* and *tenonitis*, so that it seems to me worth while to give this question some attention. *Tenonitis* was first described by O'Ferrall[16] under the name "inflammatio tunicæ vaginalis oculi," not based, however, on anatomical researches. He laid the chief stress on one symptom, the limitation of the inflammatory œdema to the tarsal portion of the upper lid. Berlin[17] contends the pathognomonic value of this symptom, and is of opinion that the affection of Tenon's capsule is only a complication in some forms of phlegmon of the retrobulbar tissue, being only a portion of the latter. Schweigger[18] thinks it to be very unlikely that an inflam-

matory process of well-marked symptoms should be confined exclusively to Tenon's capsule, without involving the orbital tissue. Linhart [19] considers tenonitis only as a "supposed possibility." In opposition to that Mooren [20] characterizes tenonitis by the following symptoms: impaired mobility of the globe with slight protrusion of the eye and transparent chemosis. Hock [21] describes a case of genuine tenonitis, based upon redness and chemosis of the ocular conjunctiva, lack of mobility in all directions, tearing pain, especially in movements, but the eye not protruded. He considers the latter symptom as the most important in retrobulbar cellulitis. The clinical features of *orbital cellulitis* are: dull pain in forehead or orbit, inflammatory swelling of lids, especially the upper lid, swelling of the ocular conjunctiva, deficiency of mobility, either general or partial, exophthalmus, and diplopia. The lack of mobility and exophthalmus are caused either mechanically by the inflammatory effusion acting as a displacing foreign body, or by paralyzing the nerves and muscles through pressure. By some authors inflammatory and degenerative changes in the ocular muscles were observed (Schmidt-Rimpler,[22] Leyden,[23] myopathia propagata, Friedberg [24]). The superior rectus and the levator palpebræ superioris seem to show a predilection for the affection (according to Pagenstecher [25]), causing ptosis and preventing rotation upwards. In the milder forms of orbital cellulitis authors concur in the *absence* of *chemosis*. If we consider now the recent observations of tenonitis and orbital cellulitis in consequence of influenza, so far as I have perused the incident literature, they come under the following headings: *Purulent Tenonitis:* one case of Fuchs [26] and one of Schapringer.[27] *Serous Tenonitis:* three cases of Fuchs and one of Greef.[28] *Orbital Cellulitis:* two cases of Pflüger,[29] one of Stoewer,[30] one of Valude,[31] *with orbital abscess:* one case of Borthen,[32] and one of Socor.[33] Fuchs as well as Greef, in their cases of serous tenonitis suspected at first incipient phlegmon of the orbital tissue, but corrected this assumption into the diagnosis tenonitis on account of the three following points: 1. The intense œdema of the conjunctiva (chemosis); 2.

The unusual limitation of mobility of the globe; 3. The moderate exophthalmus. Pflüger, however, thinks that the three cases of Fuchs, as well as his own two, are to be taken for cases of inflammatory œdema of the orbital structures, since in all the globe was displaced only in one direction, namely, forward and downward. He apparently thinks, and Stoewer and Borthen are of the same opinion, that in tenonitis the displacement is only forward, and the lack of mobility is general, not partial. He further says that the inflammatory œdema of the lids is a sign of orbital cellulitis, whereas Stoewer claims the absence of this phenomenon as confirming his diagnosis of retrobulbar effusion behind Tenon's capsule, excluding tenonitis. If we remember the anatomical relations of Tenon's capsule to the eyeball, nothing seems to be more natural but that one of the first symptoms of tenonitis, *i. e.*, an effusion between its parietal and visceral portions, must be chemosis of the ocular conjunctiva. The resistance there is much less—being only the loose, pliable, and easily movable conjunctiva—than at the posterior segment of the globe, where the whole eyeball would have to be pushed forward. The equal restraint of mobility in all directions, and the pain experienced in movement, are easily understood from the fact that all the ocular muscles are ensheathed by Tenon's capsule, and must naturally suffer from accumulations of inflammatory products in the capsule. In those cases described as tenonitis, in which the exophthalmus was more conspicuous, it seems to me more natural to assume a simultaneous inflammation of the retrobulbar tissue, which cannot expand except by protruding the eyeball, and therefore explaining, if circumscribed, the displacement not in the direction of the orbital axis, but forward and downward. In our case, however, as well as in those of Pflüger and Stoewer, the orbital cellulitis was not universal, but only confined to the upper portion of the orbit, affecting the upper branch of the oculomotor nerve by compression, and thus creating paresis of the levator and superior rectus, and the branch supplying the internal rectus. It was very mild, not implicating Tenon's capsule,

and therefore not producing chemosis, so that Tenon's capsule acted, as it were, as a barrier, preventing the effusion in the orbital tissue from finding its way into the capsule itself, and then beneath the ocular conjunctiva. It is not even necessary to assume an effusion; it might have been simply a hyperæmic condition of the retrobulbar tissues, as observed in other organs, being one of the characteristic effects of influenza.

The ear is the organ which has shown particularly well the tendency of influenza to lead to hyperæmia. Michael[34] has formulated the character of ear diseases in influenza in the following résumé: "The ear symptoms in influenza are objectively and subjectively the expression of an intense hyperæmia of the mucous membrane of the hearing organ." "Middle ear and mastoid process are enormously painful, because the swollen mucous membrane suffers from pressure. The products of inflammation, however, are absent, and with these the usual doughy swelling." "The hyperæmia is the indirect cause of the observed complications, leading to ruptures of blood-vessels, whose walls may be altered, and consequently to hemorrhages, and rendering the mucous membranes more susceptible for the invasion of infectious matter." Mostly all observers described as the most common ear complication in influenza, otitis media of great intensity and great pain, with tendency to hemorrhages. According to Schwabach,[35] the external ear participated more frequently than usual with intense inflammatory infiltration of the walls of meatus and extensive scaling of epidermis. Szenes[36] observed several times eczema of external meatus. In regard to affections of the mastoid, the observers are divided. Stimmel[37] never had to open the mastoid in his 100 cases of otitis media. Dreyfuss[38] found seldom the tendency to mastoiditis. Hoffmann[39] never observed it in 35 cases; Eitelberg[40] 1; Schwabach[41] 2 in 100 cases of otitis media, of which only one was opened. On the other hand, Politzer,[42] Gruber,[43] Ménière,[44] Szenes,[45] Chatelier,[46] noticed frequently the implication of the mastoid. Ludewig[47] saw repeatedly acute caries. Of Jansen's[48] 100 cases, the mastoid was affected in

57, in 25 chiselling, in 12 subdural abscess, with 2 deaths. Moeser[49] describes a case of *primary periostitis* of the mastoid due to influenza. In all these cases the mastoiditis was *secondary* to otitis media. Our case, however, is a case of *acute primary mastoiditis interna.* The accumulation of epidermic scales had nothing to do with the inflammation, and the swelling of the walls of the meatus was only a secondary affection to the inflammation of the mastoid. There was never any discharge from the ear, and from the very beginning the pain was confined to the mastoid spontaneously, and on pressure, without redness or swelling of the integuments. *Primary mastoiditis interna, i. e.*, the primary inflammation of the lining membrane of the mastoid cells, without manifestations on the external surface of the bone or the periosteum, and without preceding otitis media, is a very rare disease (Schwartze,[50] Politzer,[51] Fulton,[52]). According to Politzer, it is due to the effects of colds, injury or syphilis. The chief symptom, on which the diagnosis rests, is the constant and *persistent pain* in the mastoid, radiating over the head, face, neck and shoulder, as very precisely described by Knapp.[53] If left to itself, there will be a very late appearance of inflammatory symptoms, as swelling and redness in the external parts. In our case the *pain* was very characteristic with *absence* of *external symptoms*, the *fever* and the influence on the *general condition* of the patient supporting the diagnosis very effectively. The intermission of pain of about ten days may be explained by the abatement of the hyperæmia (under the very rigorous antiphlogosis), which, from the violent symptoms from the very beginning, must have set in very rapidly and very intensely. It returned when the inflammatory products, the fungoid vegetations of the degenerated mucous membrane had developed to such a degree that they filled the mastoid cells, obstructing the access to the tympanic cavity, and thus preventing a secondary inflammation of the latter. Or it may be interpreted by a re-invasion of the pathogenic poison of influenza, as some observers commented upon the relapses during the course of influenza. In otitis media due to influenza, the following micro-organisms have been found:

diplococcus pneumoniæ (Fränkel–Weichselbaum), streptococcus pyogenes, staphylococcus pyogenes albus and strepto. pyog. alb. (Zaufal,[54] Finkler,[55] Scheibe,[56] Gradenigo [57]). Fuchs found the diplococcus pneumoniæ in purulent tenonitis, Socor diplo-, strepto-, staphylo-cocci in phlegmon of the retrobulbar tissue. This leads us to the question, whether the ear complications in influenza are a propagation of the inflammation of the pharynx and tonsils, or whether they are a localization of the influenza itself in the ear. In our case the latter seems to be the most probable, inasmuch as a transportation of the phlogogenic matter from the pharynx would have followed the anatomical paths and first affected the tympanic cavity instead of leaving it intact, invading only the mastoid cells. Besides that, the character of the hyperæmic condition, as found at the operation, would be in favor of a direct localization.

In regard to the treatment leeches (Michael, *l. c.*) are most recommended as best adapted to relieve such hyperæmic affections. The general condition of our patient, however, did not admit of such weakening measures, especially as in case of failure opening of the mastoid would have been resorted to. The affliction being an acute one, the pain alone would not have been a sufficient indication, as, according to Knapp (*l. c.*, p. 368), "the vast majority of cases of acute mastoiditis get well without an operation, when rest in bed and rigorous hygienic deportment are enforced." "Yet when the symptoms are alarming, and when in spite of strict treatment the headache does not abate, I think the opening of the mastoid indicated." Schwartze (*l. c.*) proposes the operation, if after eight days the antiphlogosis has not produced a decided improvement. In our case, the revival of the symptoms after an intermission of ten days, especially the fever and the impoverished condition of the child were sufficient proof that a further delay of the operation would have been deleterious to the patient. The possibility was that, our waiting for the development of empyema of the mastoid with more striking symptoms, might have exposed the patient to the dangers of pyæmia or meningitis, or to a secondary purulent otitis

media with perforation of *Mt*, and lingering course or, what in our case was not very likely, that by passing into a chronic mastoiditis interna it might have led to sclerosis of mastoid (as observed by Knapp, *l. c.*), causing great suffering to the patient, and necessitating, perhaps, a later operation under much more unfavorable circumstances. The condition of the mastoid cells found at the operation and the result proved, that in this case the operation was the only right proceeding. Our case, therefore, is a new addition to the series of those aurists who, under similar conditions, advocate an *early* operation. In regard to the technique of the operation and the treatment in this as in other cases, the strictest aseptic and antiseptic measures, with iodoform-gauze tamponade as drainage (no tubes, no plug of lead), have given excellent results.

To sum up, we have a case of influenza—its diagnosis based upon the sudden onset of symptoms with nervous prostration, exanthema, angina tonsillaris, pharyngitis—with orbital cellulitis and acute primary mastoiditis interna as a result of the tendency of influenza to cause hyperæmic, and further on inflammatory, conditions in different organs.

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