A CONTRIBUTION

TO THE

PATHOLOGICAL HISTOLOGY

OF

ACUTE PAROTITIS.

BY

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A CONTRIBUTION TO THE PATHOLOGICAL HISTOLOGY OF ACUTE PAROTITIS.

The minute anatomy of the salivary glands is comparatively well known. The physiological processes involved in their specific secretion are, in the main, fully understood. But our comprehension of pathological changes occurring in the glands of this group is singularly incommensurate with the knowledge we possess of their anatomo-physiological relations.

Some time ago I had occasion to observe an acute parotitis, as an accidental complication in a fatal case of meningitis. The opportunity for studying the morbid anatomy of parotid inflammation seemed a good one, and the glandular lesion was therefore carefully examined. My investigations resulted in the more accurate determination of several hitherto questionable points, which I believe to be of interest in this connection, even though they may be found devoid of any startling pathological novelty.

Briefly summarized, the essential circumstances attending the development of parotitis in this instance were as follows: In March, 1878, a patient, physically and mentally much debilitated, was admitted to the German Hospital, in New York, where I was then Resident Surgeon. The man's history, elicited from friends, and the symptoms he showed, both

pointed to a chronic meningitis as the cause of his troubles. In spite of all treatment the disorder gained ground, he sank rapidly, and died on the 16th of March, at 8 P. M. On the previous day he had had a chill, rapidly followed by elevation of temperature; the febrile movement continued up to the time of his death, the rectal temperature varying between 101° and 103° F. On the morning following the chill, a painful tumefaction of the right parotid region became noticeable; subsequently there also appeared a slight swelling on the left side. In the evening, as stated, he succumbed.

At the autopsy, made twelve hours after death, there were found the meningeal affection that had had been diagnosticated, incipient cerebral softening, and profound general atrophy of the various organs and tissues of the body. The apices of both lungs showed scattered caseous deposits and old indurations, but disseminated tuberculosis was not discoverable. The right testicle was larger and harder to the touch than the left one. The parotid and submaxillary glands were dissected out, and, together with both testicles, removed for examination.

Macroscopical Relations.—The right parotid gland, somewhat larger than a hen's egg, was enveloped in layers of œdematous areolar tissue. Its surface was lobulated, and showed grayish protuberances and elevations, with surrounding areas of a reddish tint. Two thick processes bulged out from the inner inferior margin, and these were paler than the remaining portions of the gland. They were also found to be harder to the touch than the rest of the organ. The cut surface pre-

FIG. 1.—Portion of Cut Surface of Right Parotid, showing round spots studding the glandular lobules—the latter inclosed in a reticulum of hyperæmic interstitial tissue. (Slightly magnified.)

sented a mottled appearance. Rounded dots, varying in size (the largest not much greater than a pin's head, the smallest scarcely perceptible to the naked eye), were scattered over the parenchyma. These spots were especially conspicuous toward the central portions of the gland. They had a pale, muddy look. The surrounding structure was composed of irregularly formed patches of a fleshy hue, contained in meshes of a still darker tinge. In the processes above mentioned these appearances were somewhat modified. Here the parenchyma was recognized to consist of a yellowish tissue, relieved by a network of reddish streaks, and containing faint specks, barely visible to the naked eye.

The submaxillary gland was of normal appearance. The inferred absence of structural modification was corroborated by the microscope. This gland may therefore be dismissed from further consideration.

The parotid of the left side was of abnormally firm consistence, but was only moderately enlarged. In all other respects it resembled the two processes of the right parotid.

Microscopical Appearances.—Teased preparations of both glands were examined in Müller's fluid and diluted chromic acid ($\frac{1}{2}\frac{1}{0}$ per cent.) immediately after removal from the body. Different portions of the organs were hardened, some in strong alcohol, some in neutral chromate of ammonium, others in Müller's fluid, or a $\frac{1}{8}$-per-cent. solution of chromic acid. Numerous sections from every available portion of the glands were then made. Some of these, properly stained, were examined in glycerine, others were mounted in Canada balsam. As staining-fluids I employed Beale's carmine, Fischer's eosine, picro-carmine, hæmatoxyline, and my own combinations of eosine and picric acid, or hæmatoxyline and eosine.*

I will begin by recording the morbid appearances found in the left parotid, where, as became manifest by comparison, the disease was still in its incipient stage. A general survey of a number of thin sections of the hardened gland showed clearly enough that the most prominent textural changes had

* See Wendt, "Ueber die Harder'sche Drüse der Säugethiere," Strassburg, 1877, pp. 27, 28.

concerned the blood-vessels and the inter-acinous spaces. All the arterioles were greatly distended and thickly packed with blood-corpuscles; the capillaries were in a similar condition

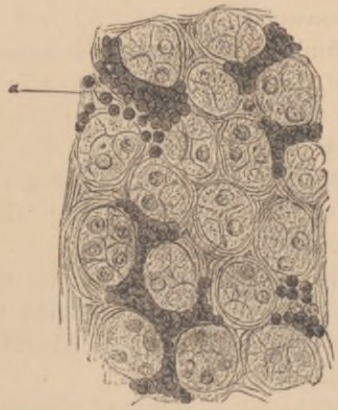

Fig. 2.—Section of Gland of Left Side (Hartnack, oc. 3, obj. 7), arterioles choked with blood-globules; a ruptured capillary at *a*; hypertrophy and proliferation of secreting epithelia. (The leucocytes crowding the inter-acinous conjunctive tissue have purposely been omitted from the drawing.)

of active hyperæmia—here and there, indeed, one had become ruptured, and free hæmorrhage into the surrounding tissue had resulted. The interstices of the parenchyma proper showed serous imbibition, dense infiltration of leucocytes, and scattered red blood-corpuscles. The circumglandular connective tissue exhibited similar morbid alterations, but in a less degree, the changes being as yet chiefly intravascular. On examining very thin sections with a higher power (Hartnack, oc. 3, obj. 7), the secreting elements were also found to have already undergone important alterations. Upon comparison with normal glands it became evident, not only that the epithelial components of the lobules were considerably enlarged, but that their protoplasm was far less granular, more homogeneous, and paler, and the cell-boundaries withal less marked. In addition, the nucleus was often dimmed by seemingly morbid cell-contents, though the nucleoli, frequently two in number, were distinctly visible, as bright specks in a vaguely spheroidal mass, indicating the nucleus. Hence these cells were engaged in active proliferation. There was, then, indi-

vidual epithelial hypertrophy and concomitant cellular hyperplasia. The remaining gland-elements, the salivary ducts,

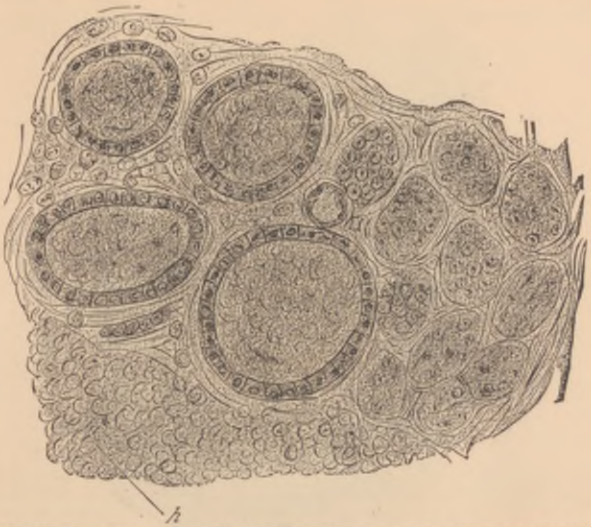

FIG. 3.—Section from Gland of Right Side, showing portion adjoining periphery. Transverse section of four distended salivary ducts; hæmorrhagic collection at *h*. (Oc. 4, obj. 7.)

were distended with an opaque amorphous substance, in which lay imbedded occasional bodies resembling leucocytes. The lining epithelium of these canals was somewhat flattened and abnormally dark, otherwise unaltered.

The first glance at the other gland at once displayed numerous and extensive degenerations. I shall try to analyze these, step by step, in order to avoid possible confusion. Certain gland-territories were still almost unchanged in structure, others resembled the portion already described, and still others had become so altered as to be no longer recognizable as glandular tissue. The most intense modification had occurred in the interior of the gland; the outer borders had scarcely been reached by the process of degeneration. Turning our attention for the moment to the former, we found, instead of sacs containing secreting epithelia, only rounded meshes of connective tissue, filled with accumulated débris, the product of cellular disintegration. Scattered in this mass we were still able to discover free nuclei, fat-granules of various sizes, and pus-

corpuscles imbedded in a finely molecular substance. The periphery of these meshes sometimes showed the remnants of

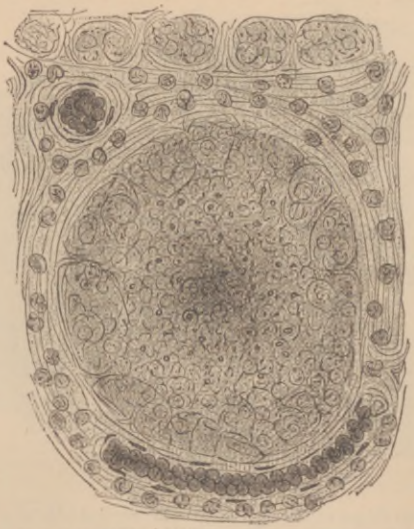

Fig. 4.—Central Portion of Right Gland, showing a round mesh containing internal débris; peripheral remnants of acini; the surrounding connective tissue moderately infiltrated with leucocytes. (Oc. 4, obj. 7.)

acini with barely recognizable epithelial elements containing altered nuclei. These collections represented the most advanced

Fig. 5.—Sero-purulent Infiltration and Globular Extravasation of Interstitial Connective Tissue. (Oc. 4, obj. 7.)

stage of the disease, were not very numerous, and may be regarded as small pseudo-abscesses. They were surrounded

by blood-vessels less distended than in the portions already referred to, and by interstitial tissue, the seat of sero-purulent infiltration and globular extravasation. Then followed gland-tissue where the distribution of cellular elements still retained

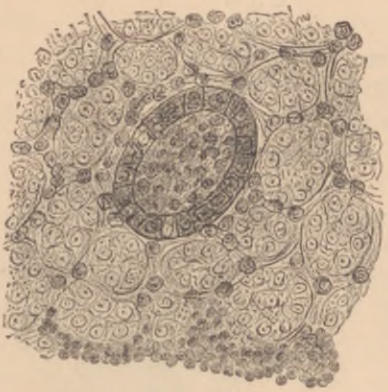

FIG. 6.—From the Gland of the Right Side. Toward the center, a salivary duct, replete with puriform matter, its lining epithelia flattened and abnormally dark; the acinous epithelia contain distinct nuclei; the interstitial tissue still infiltrated; at the lower right-hand corner free hæmorrhage has occurred. (Oc. 3, obj. 7.)

the typical appearance of secreting structure. But the epithelia were very much swollen, filled with fat-granules, the nucleus either absent or invisible, and their interconnection quite loosened. In a word, they represented a condition of granulo-fatty degeneration, just short of final disintegration. Further on, i. e., in an eccentric direction, we noticed the circumlobular tissue less infiltrated, the epithelia possessing characteristic nuclei, some, indeed, inclosing a double nucleus. But, instead of the normal granular protoplasm, the cell-contents consisted of a homogeneous substance, with occasional fat-molecules. In this region the salivary ducts were crammed with puriform elements, their lining epithelia flattened, darker, but less granular than normal. Many of these duct-cells included two nuclei, but there was an occasional gap in their continuity, as if an epithelium had dropped out of rank and its place had been left vacant. Still advancing, we came upon groups of epithelia so arranged as to accurately resemble normal acini. On comparison, however, it appeared that, apart from this general similarity of configuration, a wide

diversity obtained. The individual cells had encroached on the intraäcinous lumen to such an extent as to make that

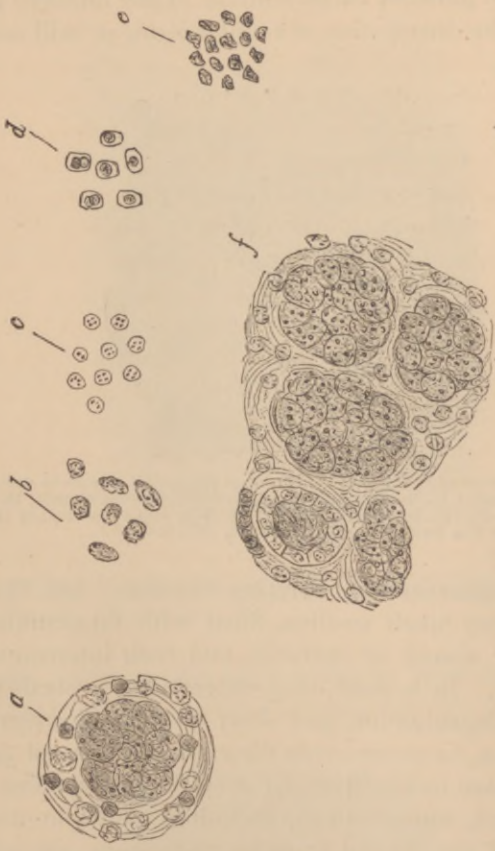

FIG. 7.—At *a*, an acinus, surrounded by connective tissue; the epithelia in a condition of cloudy swelling; the connective tissue infiltrated with leucocytes and containing red blood-globules. (Examined fresh in Müller's fluid—oc. 4, obj. 7.)
At *b*, *c*, *d*, and *e*, different forms of cellular elements floating in the liquid; *b*, ovoid and spheroidal leucocytes; *c*, nuclei of secreting epithelia; *d*, elements derived from the salivary ducts; *e*, shrunken corpuscles of various origin; *f*, cloudy swelling, with commencing fatty degeneration. (Oc. 3, obj. 7.)

opening seem to have entirely disappeared, and they were themselves in a condition of cloudy swelling. Here the circumglandular tissue was again replete with colorless blood-corpuscles. It formed wide meshes, in the interspaces of which lay the degenerated epithelia. Still other portions, those nearest the exterior rim of almost normal parenchyma, displayed only those changes already described as existing in the other gland. Modifications of a character similar to the latter were encountered in the two processes previously mentioned.

Estimation of Pathological Significance.—Having thus far considered the subject chiefly in its morphological bearings, I now take up the interpretation of the pathological processes involved in the disease. This may enable us to satisfactorily determine the interrelation of microscopical appearances and clinical characters, which should form, in part at least, the object of every investigation of this kind. I must premise, however, that I am not prepared to state whether the lesions in the parotid gland were only the indirect expression of some toxic principle circulating in the blood, or whether they were induced by some indeterminate and indeterminable topical irritation proceeding perhaps from the buccal cavity. Indeed, whether merely a coincident complication or a spontaneously developed so-called idiopathic affection, I find the main interest of the case to be centered in the fact of the unquestionably acute character of the extensive degenerations. The local signs in this instance, it will be remembered, were developed quite suddenly after a chill on the previous day, and some twenty hours before death. It might be argued that the disease began long before it became clinically perceptible; but when we consider the initial chill, preceding all local manifestation, such reasoning seems futile, and may be dismissed without further notice. Had the man lived and the morbid process been continued, there might have been further resolution, and finally re-absorption of the molecular detritus mentioned; for, along with the process of rapid destruction, we have observed a constructive hyperplasia of secreting epithelia, so that newly-formed elements were alread yon hand, waiting, as it were, to replace the vanishing older generation. This new formation of typical gland-tissue went on simultaneously with the retrogressive metamorphosis of other secreting structure. Had all the accumulated débris suffered rapid re-absorption (a process which is by no means improbable, considering the finely molecular and fatty nature of its ingredients), we should not have hesitated to call the disease mumps (parotitis polymorpha). On the other hand, had extensive suppuration or sloughing supervened, we should have pronounced the case an example of metastatic purulent inflammation. In the absence of either consummation, it would appear idle to discuss the

greater or less probability of one or the other termination. Nor does this seem to me to affect the interest of the case. The question therefore remains an open one, whether we were dealing with mumps, *sensû strictiori*, or with secondary parotitis. That it was an instance of quite acute parotitis is perfectly evident.

I think, in view of the paucity of well-examined cases of this kind (a very natural paucity, it is true, considering the short duration of acute parotitis, its slight dangers, and manifest tendency to spontaneous recovery), any contribution to its pathology deserves to be placed on record; for the absence of a grave prognosis will surely be admitted as not necessarily excluding pathological interest. The pathologist may find ample material for study and reflection where the clinician fails to recognize either importance or interest.

After this digression, we will resume the discussion of the nature, origin, and growth of the morbid process in the case engaging our attention. The first stage of the disease was evidently one of congestive hyperæmia. The arterioles and capillaries were visibly distended and packed with blood elements. Simultaneously with or immediately after this increased blood supply, exudation of liquor sanguinis and

Fig. 8.—From a process bulging from the gland of the right side; interstitial hyperæmia and hæmorrhage. (Oc. 2, obj. 5.)

emigration of numerous leucocytes, with occasional red globules, took place. About this time rupture of capillaries also

occurred. These are familiar phenomena, and require no further elucidation.

Until now the epithelia of the secreting parenchyma remained intact, though the salivary ducts were already filling with material, and contained many formed bodies. The latter were partly of hæmic origin, and partly derived from the cell-elements of the ducts themselves. Now, under the abnormal trophic stimulus of excessive blood-supply, a pathological growth of each individual cell took place, at the same time the nucleus divided into two, and subsequently segmentation of the whole cell ensued. The epithelia thus rapidly underwent numerical increase. This was the period of hypertrophy and hyperplasia. Soon, however, the acinous secreting constituents quickly underwent cloudy swelling, then fatty degeneration, and finally complete disruption. The confluent products of this necrobiotic metamorphosis accumulated in the meshes of the interstitial tissue (see Figs. 3, 4, 5, and 6). The more succulent ingredients of this connective material were themselves totally destroyed in the general breaking-down. Some of its fibers, however, and notably the elastic fasciculi, opposed a firm resistance to the last, so that there was no diffuse infiltration, but circumscribed aggregation of a detritus of very complex origin. This, in our case, was the final phase (Fig. 3).

That, prior to this dénouement of the retrogressive metamorphoses, there had been established a condition of progressive hypertrophy and active proliferation, has already been noted. In the collections of tissue-débris just mentioned, there might still be recognized, as a pooof of their origin, epithelial cells in a state of advanced degeneration, numerous free nuclei and commingled leucocytes, fat-granules, and, in quantity the most conspicuous, a finely molecular mass, already described. Examples illustrative of the different phases of this extremely rapid pathological action were readily found in the various portions and regions of both glands.

An examination of the left testicle showed the same to be free from structural alteration, barring only an ordinary general atrophy. The right one was found to be in a condition of moderate hyperæmia, in conjunction with which slight

exudation had taken place. Since the existence of a certain peculiar "*consensus*" between glandular bodies is a well-established empirical fact, I dismiss these changes as undeserving of further comment. Incidentally, it may be mentioned that serous exudation had also occurred into the cavity of the tunica vaginalis propria of the right testicle.

Reviewing the series of morbid transformations which constituted the parotid affection in our case, we must at once concede the analogy between this disease and certain forms of very acute and intense inflammation of mucous surfaces; conjunctival blennorrhœa, for instance, say from gonorrhœal infection, would furnish an example characterized by similar successive stages of hyperæmia and proliferation, followed by rapid tissue destruction, with the eventual possibility of a *restitutio ad integrum*. This is no similarity arbitrarily adduced, but an analogy springing from the histogenetic structural likeness of the glands of this order to mucous surfaces generally. It is therefore something beyond the fundamental resemblance of all inflammatory processes. Though I wished to emphasize this point, it seems needless to enlarge on it here.

In conclusion, it may be interesting and instructive to take a cursory glance at the opinions of authors on the nature of this affection, and in this way institute a brief comparison of their conclusions with the deductions derived from my own examination. Formerly, the circum-glandular areolar tissue was supposed to be the principal seat of the disease. The gland itself, it was held, rarely if ever participated in the morbid process. This view, based chiefly on clinical evidence, is still retained by some authors. Others, from inferential analogy, rather than actual observation, maintained the complete identity of parotitis with inflammation of the lymphatic glands. Virchow ("Annal. d. Charité," Berlin, 1858, viii, 3) showed the opinion to be erroneous which located the affection in the conjunctive tissue surrounding the gland. He regards parotitis as essentially a catarrh of the salivary ducts and secreting parenchyma, with frequently a secondary participation of circum-glandular connective tissue. My case, it will be seen, therefore, only partially bears out his interpretation of

the subject, the ducts in this instance being the seat of secondary, not primary change. Förster ("Handbuch d. pathol. Anat.," Leipzig, 1863, p. 48) described several distinct phases of the disease. According to this author, there is initial hyperæmia, then rapid exudation, followed by purulent infiltration of the inter-acinous spaces. The terminal secreting vesicles undergo fatty degeneration. Re-absorption may take place, but diffuse necrosis, causing abscess, is more common. This, then, is a view which receives corroborative illustration from my case. The chief point of difference is that he makes no mention of the stage of cloudy swelling preceding that of fatty degeneration of the epithelia. But this is probably only an omission, and not a fault of observation.

Klebs ("Handbuch d. pathol. Anat.") thinks the affection invariably begins with swelling and redness of the interstitial tissue. The subsequent and consequent proliferation is most active in immediate proximity to the secreting vesicles. Diffuse purulent infiltration precedes parenchymatous necrosis. Gangrene of various regions of the gland is established. In many cases there is only periparotitis. His "swelling and reddening" correspond to the initial hyperæmia of Förster, as well as the first stage of the disease in my case. But in the latter the infiltration of pus was not diffuse; moreover, the limited necrosis was preceded by distinct periods of cloudy swelling and fatty degeneration.

Rindfleisch ("Lehrb. d. path. Gewebelehre," Leipzig, 1873, p. 510) found hyperæmia, œdema, *and* cloudy swelling, *followed by* puro-catarrhal secretion, proceeding partly from the epithelia of the acini, partly from the surrounding conjunctive tissue, with a simultaneous cellular infiltration of the interlobular spaces. These changes preceded a breaking through of the pus into the alveoli, thus causing their destruction. In my case, it will be remembered, the alveoli were broken up by intra-alveolar, not extra-alveolar processes.

Vogel, in Ziemssen's "Cyclopædia," states that only hyperæmia and serous imbibition can form the anatomical basis of mumps, since other products are not capable of rapid re-absorption. He also describes a secondary purulent catarrh, with accumulation of a viscid, tawny secretion in the tubules of the

gland, usually followed by sloughing. He invalidates the acceptability of his views by admitting them to be inferred from analogy instead of based on actual observation.

Amid the existence of this great diversity of opinion, a certain underlying similarity still prevails, yet this only tends to show that the subject is not the simple one it might seem. I will not add to the length of this paper by citing other authors. Nor does it appear needful to append a résumé of conclusions deducible from my case. Corroboration, chiefly of the results of Förster's investigations, being implied, I refrain from an explicit demonstration of the points of likeness and difference.