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JEJUNAL ULCER ASSOCIATED WITH TESTICULAR PAIN.

BY

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THE following case appears to be worth recording on account of the unusual symptoms.

A man, aged 37, had mumps as a child, but otherwise no illness worthy of note till December, 1915, when he was invalided from the army with dysentery. Shortly after returning to duty he was buried by a shell. He was apparently concussed, and immediately afterwards the thyroid gland was seen to be much enlarged; it gradually became smaller, and now seems to be normal. Shortly after the concussion he began to have attacks of epigastric pain, associated with hunger, about half an hour after meals, and tenderness in the epigastrium to the right of the mid-line. At this time he was having irregular meals and smoked heavily; the pain was relieved by food for about half an hour, and then returned increased, being frequently associated with vomiting. At the beginning the attacks lasted about a week, pain appearing after every meal during this time; there were intervals of two or three months during which he was entirely free from symptoms. The attacks were specially severe during the winter, and were usually accompanied by enlargement of the thyroid gland. They gradually became more frequent and of longer duration.

In July, 1919, he had an attack of unusual severity, and three weeks later, while working in his office, he experienced sudden acute abdominal pain associated with repeated vomiting. An operation was performed for perforated duodenal ulcer, but he was apparently given no instructions as to diet, and he ate what he pleased. He remained well for about two years, after which the symptoms appeared again more acutely, and the pain and tenderness extended across the epigastrium. The stools now showed the presence of occult blood. He was treated for six weeks by dieting, but was not confined to bed. After a short interval the symptoms returned, and in February, 1925, gastro-jejunostomy was performed; following this, he was put on a moderately restricted diet for two months.

Six months later he again had symptoms, but they were not so acute as before the operation; they came on particularly when the weather was cold. The pain became gradually longer in duration, increasing in intensity during the day, independent of meals, and was not now relieved by food. On the other hand, for the first time he started to be wakened by pain between 2 and 3 a.m. This only occurred if he had taken a meal after 7 p.m.

In August, 1926, he first felt a dull aching pain in the testes at the end of the day, when the epigastric pain was most acute. A suspensory bandage gave little, if any, relief. Gradually the pain in the testes became greater, and appeared earlier in the day, together with pain in the loins. He never had these symptoms except when the epigastric pain was present, and there was no abnormality in micturition.

In May, 1928, he was awakened about 3 a.m. by attacks of pain in the testes, so severe as to necessitate injections of morphine. There was general tenderness in the upper abdomen, especially at a point immediately above and to the left of the umbilicus, where a small lump could be felt. It seemed to be fixed to the deeper tissues, and moved with the pulsations of the aorta. Pressure on the lump reproduced the pain in the back and in the testes. The testes themselves were tender, and there was slight hyperalgesia in the loins. The urine was normal, and examination of the urogenital tract revealed no abnormality. An x-ray examination with a barium meal showed a jejunal ulcer in the situation of the tumour. Occult blood was present in the stools. The patient was confined to bed, and treated medically on a strict diet. The pain was rapidly relieved and the tumour gradually disappeared. Since then he has remained on a modified diet, and has become a non-smoker. He has remained free from symptoms, has put on 2 st. in weight, and remarks that he has never felt better in his life.

The two main points of interest in this case are: the association of thyroid enlargement with attacks of epigastric discomfort due to duodenal ulcer, and the pain in the testes and the loins. With regard to this pain, four points may be emphasized. (1) Testicular pain was never

present except when there was pain in the epigastrium. (2) Pressure on the tumour produced pain in the testes. (3) Subsidence of the pain, both in the testes and in the epigastrium, coincided with the disappearance of the tumour. (4) The tumour moved with the pulsations of the aorta, to which it appeared to be fixed. The first three of these points suggest that both testicular and epigastric pain had a common origin.

X-ray examination showed that the jejunal ulcer and the tumour were situated just to the left of the mid-line, at the level of the intervertebral disc between the third and fourth lumbar vertebrae. The sympathetic nerve supply, both of the kidneys and the testes, is derived from the twelfth thoracic and first lumbar segments of the cord. These nerves form a plexus in front of the aorta, and branches from this plexus accompany the renal arteries to the kidneys. Branches from the same plexus accompany the spermatic vessels to the testes, and are also distributed to the ureters. Thus the inflammatory mass associated with the ulcer was situated where the sympathetic plexus, in front of the aorta, would be involved, and the patient's symptoms suggest that pain can be referred to a viscus through sympathetic nerves. The phenomenon also suggests an explanation for the testicular pain in ureteric colic, other than the one advanced by Mackenzie, that both these structures are supplied by branches of the genito-crural nerve.

Periodic stimulation of the sympathetic plexus by the inflammatory mass might be due to periodic vascular engorgement or to actual traction on the plexus. The latter seems the more likely explanation. The traction could be produced by gastric movement, the site of the ulcer being fixed. If this explanation is correct, the coincidence of the epigastric pain with that in the testes suggests that pain due to an ulcer is associated with movement of the diseased viscus, whether the movement be tonic or peristaltic in origin. The persistent character of the pain suggests an alteration in tone as the more likely explanation.

TRAUMATIC FAT NECROSIS OF THE BREAST.

BY

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THE condition of traumatic fat necrosis of the breast was first described in America in 1920 by Lee and Adair,¹ who published two cases. They wrote further on the subject in 1922² and in 1924,³ and since then several cases have been described. The first published in this country was by Keynes.⁴ Later, Hadfield⁵ wrote on the subject.

In these cases, following trauma, there developed in the breast a lump which clinically appeared to be a carcinoma, but on microscopic examination areas of fat necrosis only were found, and no evidence of malignant disease was present. Farr⁶ was able to produce experimentally the characteristic lesions by pinching with forceps the subcutaneous fat of young pigs. Sections of the injured areas showed the typical histological picture of fat necrosis. Farr concluded that in an area of traumatized fat the lipolytic enzyme present in fat and blood is liberated, and acts on the neutral fat, breaking it down into its component parts—fatty acid and glycerol. These substances form the irritant which gives rise to the foreign body reaction producing the lesions of traumatic fat necrosis. Although trauma seems to be the usual factor in the published cases, Gottesman and Zemansky⁷ came across a case in which this cause could be eliminated. Further, they analysed 78 cases of non-malignant breasts, and in 14 found some stage or degree of fat necrosis. They concluded that factors other than trauma may explain the phenomenon, and that the escape of fatty material from a dilated duct and its subsequent decomposition plays a role in the cause of this condition.

Professor M. J. Stewart calls the condition subacute panniculitis. He tells me that in his experience it is not uncommon, and he has met with it in the omentum,

in fat removed from the femoral canal, and in the subcutaneous fat in different situations in the body as well as in the breast. In a paper on xanthema and xanthosis⁸ he writes:

"When adipose tissue is involved in a subacute inflammatory process it is not uncommon for aggregations of large, foamy endothelial cells to make their appearance in the interstices of the tissue between the oil droplets. Like foamy cells elsewhere, they are filled with cholesterolin fat, and often they may be seen going on to the formation of foamy giant cells. Rarely pure cholesterolin crystallizes out, and a remarkable histological picture results, in which foamy cells, giant cells, cholesterolin clefts, and foci of necrosis are mixed up together. Similar changes may be met with in the quieter forms of fat necrosis."

That this condition is not so uncommon as supposed is also evidenced by a table published by Lee and Adair² in the article already referred to. During the period in which their five cases of fat necrosis occurred they found that, compared with primary breast cancer, the incidence was 1.8 per cent., with operable cases of breast cancer 4.2 per cent., and compared with benign lesions the incidence was 7 per cent.

The following case recently came under my care:

Clinical History of Case.

Mrs. E. A., aged 53, no children. Rather an obese subject. She knocked her right breast two months previously to noticing a lump therein. On examination, there was a nodule about 1½ by 1½ in. in the upper and outer quadrant of her right breast; it was hard and fairly well defined, and could be easily felt with the flat of the hand. The centre of the nodule was attached to the skin, but there was no deep fixity, nor could any enlarged glands be felt in the axilla. The nipple was not retracted, nor was the breast elevated. The diagnosis of early carcinoma of the breast was made, and with this diagnosis two of my colleagues agreed. A radical removal of the breast was carried out. The convalescence was straightforward, and the patient was discharged three weeks later with a soundly healed wound.

Microscopic Examination.

The breast lesion is a subacute inflammatory and necrotic one, without evidence of neoplastic change, and involving the adipose rather than the glandular tissue.

1. In the central portion of the affected area there are several small necrotic patches of irregular outline, as yet free from calcification.

2. Active fibroblastic proliferation is the chief feature of the central area. Intermingled therewith are fat-droplet spaces derived from the original adipose tissue, many of them being of large size from fusion of the contents of disintegrating fat cells. Scattered endothelial cells, lymphocytes, and "foamy" cells (fat- and lipid-filled phagocytes) are present in small numbers.

3. In the outer zone of the lesion the most notable feature is the accumulation of the same foamy macrophages in large numbers throughout the adipose tissue. They occur in irregular groups and strands, separating the adipose tissue cells from one another, and are associated with slight degrees of fibrosis. The examination of frozen sections shows that they contain both neutral and cholesterol fat, taken up by them from disintegrating fat cells, many of which have their oil vacuoles filled by the products of cell degradation.

4. A few giant cells with "foamy" cytoplasm and up to six or eight nuclei are present here and there throughout the lesion.

5. The scanty gland acini present appear to be involved secondarily in the inflammatory process.

The appearances correspond to those which have been described in America under "traumatic fat necrosis." The lesion differs from the fat necrosis of pancreatitis in being of slow evolution, so that phagocytosis of fat-cell disintegration products and fibroblastic proliferation keep pace with cell destruction. The axillary glands show no pathological changes. The condition is met with in many different sites, but in the breast it is of special importance because of the danger of confounding it with scirrhus carcinoma.

The importance of traumatic fat necrosis from a clinical viewpoint lies in its simulation of carcinoma of the breast; in many of the published cases a radical operation has been carried out, and only later, on microscopic examination, has the true diagnosis been made. The recognition of the condition is therefore important and should be borne in mind, especially in what appears to be an early case of mammary cancer. In arriving at a diagnosis the traumatic nature of the lesion should be remembered, as injury seems to have been the starting point in most of the published cases. It is interesting to note, that in

three of the five cases of Lee and Adair² the disease developed after the giving of saline infusions into the breast. Although patients with carcinoma of the breast frequently state that they have had a knock or blow on the breast, or think the corset may have rubbed them, the exact nature and date of the injury is usually rather indefinite. In cases of fat necrosis, however, the exact trauma is clearly remembered.

The time interval between the injury and the development of the tumour varies from a week or two up to several years. The condition is usually met with during the fourth and fifth decade, and mostly in women with large and fat breasts. The tumour itself, although hard like a cancer, is better defined. The superficial fixity of the tumour to the skin exactly resembles that found in cancer, but deep fixity is not as a rule present. The axillary glands are not enlarged, though this does not help in the differential diagnosis from an early cancer. In a suspected case it will often be necessary to perform a local excision of the tumour for microscopic examination, since the mimicry of early mammary cancer is so close. If the condition is borne in mind, however, and only the lump removed, adequate treatment will have been carried out and an extensive operation obviated.

I am indebted to Professor M. J. Stewart for his advice in the preparation of this article, and for the report on the microscopic examination of the specimen in the case quoted.

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SEVERE HAEMORRHAGE FROM THE RUPTURE OF A CORPUS LUTEUM CYST.

BY

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HAEMORRHAGE into the peritoneal cavity from a ruptured ovarian cyst is met with occasionally. Haemorrhage from a ruptured luteal cyst in a young woman, simulating an ectopic pregnancy, is a rare condition. Edward Moore¹ reviewed the literature in 1922 and could find only fourteen cases recorded, a list of which is given in his paper. Since that time records of several patients suffering from this condition have been published, notably in Germany by Kaboth,² Hesselberg,³ Eick,⁴ Schickele,⁵ and others. But in the English⁶ and American literature little mention has been made of this condition. It has been my good fortune to have been associated with three cases, descriptions of which are given below. As a ruptured ectopic pregnancy is closely simulated, a pre-operative diagnosis is difficult, and in this series the condition was not diagnosed before operation, and afterwards only when portions of the tissue removed had been sectioned and examined microscopically. By careful attention to the history, however, it should be possible to make a diagnosis. The most important points in the history are as follows.

The patient is a young woman, usually single; the average age in the following cases was 24. There is a history of a sudden attack of severe abdominal pain localized to the lower abdomen on the right or left side, depending probably on the ovary affected; this is accompanied by nausea, and occasionally by vomiting. The pain gradually passes off, and subsequently, following some strain producing increased intra-abdominal tension such as straining at stool, the patient has a feeling of faintness or actually does faint. The menstrual history is normal; there is no missed period, and the catastrophe bears no relation to the menstrual flow. On examination the patient is pallid, and sometimes restless and dyspnoeic. The pulse, generally rapid, thin, and running, may be slow. I have seen severe intra-abdominal haemorrhage with a pulse of 76. The temperature is usually subnormal, unless a considerable interval of time has elapsed between the