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ABERRANT ENDOMETRIAL TISSUE AND INTUSSUSCEPTION

BY

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Intussusception of the ileo-colic variety in an adult, with aberrant endometrial tissue at the apex of the entering ileum, is an unusual occurrence. V. B. Green-Armytage (1933) reports that a state of pelvic endometriosis existed in 8.9% of a series of 1,000 abdominal operation cases. Most instances in which aberrant endometrial tissue has been found to involve the peritoneal surface of the intestine are reported in locations about the sigmoid colon and recto-vaginal septum. Thierstein and Allen (1946) report 53 cases of intestinal endometriosis. Their series is subdivided into two groups: (1) 41 cases in which the endometriosis involved the rectum and recto-vaginal septum, and (2) 12 cases in which the lesions were found higher in the intestinal tract. The second group consisted of 6 cases of involvement of the sigmoid colon, 3 of caecal involvement, 1 of endometriosis of the ileum, and 2 of lesions involving the appendix.

Relatively few cases of acute or chronic intestinal obstruction due to endometriosis have been recorded. Glenn and Thornton (1940) report two cases of partial intestinal obstruction of the chronic type due to endometrial tissue. A review of the literature revealed only four cases in which the tissue involved small bowel and produced complete intestinal obstruction. I cannot find a single reported case of intussusception with endometrial tissue as a causal factor.

The justification for recording this case lies, however, not only in its extreme rarity but in the pathological histology, in which there is some evidence in support of the serosal theory of endothelial metaplasia as a factor in the origin of aberrant endometrial tissue.

Case Report

A single woman aged 23 was admitted on July 9, 1947. A year previously, while serving in the W.A.A.F., she had complained of attacks of diarrhoea and vomiting, and a diagnosis of acute food-poisoning had been made. Menstruation had started at the age of 14. The flow was moderate, lasting from

five to six days; the cycle was between 27 and 30 days. There had been severe dysmenorrhoea since the menarche, often sufficient to confine the patient to her bed, and the pain had always been associated with vomiting on the first day of the period. There was no history of pregnancy or abortion.

Three months before admission she had complained of several attacks of sharp colicky pain in the lower abdomen, more pronounced in the right iliac fossa. These attacks occurred at irregular intervals of about three weeks and lasted on each occasion for about half an hour. They had recently become more frequent and prolonged, and seemed to be clearly associated with the menstrual periods. The pain had not been relieved by sedation or antispasmodic drugs. The patient vomited during the attacks, but no nausea or vomiting occurred in the periods of intermission. The appetite was consistently poor. On two occasions there had been some diarrhoea (four or five semi-solid stools a day), but at no time was there any blood-stained discharge in the stools. The first day of the last menstrual period was 11 days before admission.

The patient was thin, lacrimose, and miserable, and was in severe pain, drawing up her legs and constantly retching and vomiting. The pulse was rapid, but regular and of good volume. The temperature was 98.6° F. (37° C.). The abdomen was uniformly tender and there was some guarding of the abdominal musculature. An oval sausage-shaped swelling was seen in the right iliac fossa. The swelling was semi-fluctuant, soft, and mobile. The white blood cells numbered 14,500 (polymorphs 80%, lymphocytes 10%).

The external genitalia were normal. The cervix and uterus were of normal size, shape, and position. A cystic swelling was palpable from the right fornix. This was distinctly felt as separate from the uterus. The mass was tender. Rectal examination confirmed the presence of a pelvic mass. Amongst the possibilities that had to be considered at this stage were appendicular abscess, Crohn's disease, pyosalpinx, extrauterine gestation, rotation of the pedicle of an ovarian tumour, and obstruction from bands or internal hernia.

Operation (July 9).—Under anaesthesia the mass felt like an appendix abscess. A McBurney incision revealed an intussusception of the ileo-colic variety, extending half-way up the ascending colon, which had to be delivered by a separate right paramedian incision. All attempts at reduction failed. A hemicolectomy was performed and an end-to-end anastomosis of the ileum to the transverse colon was carried out. Both ovaries appeared normal at operation and there was no evidence of pelvic endometriosis.

The patient made an uneventful recovery and was discharged symptom-free on July 29. On Sept. 17 she was found to be well, and the two periods since operation had passed without pain or vomiting.

Pathological Report

The specimen consisted of the resected portion of the terminal ileum, which was intussuscepted into the ascending colon (Fig. 1). The ensheathing and returning tubes had been laid open. For microscopical sections a piece was cut from the ileum at the apex of the intussusception.

Microscopical Description.—Section of the ileum (Fig. 2) showed congestion of the mucosa and submucosa and some necrosis of the mucosa. In the muscularis there were small areas of typical endometrium, composed of glands lined with tall columnar cells in an abundant stroma that was characteristic of endometrial stroma (Fig. 3), but the unusual and interesting feature in this section was that on and underneath the serosa, at the terminal tip of the intussusception and exactly opposite the intramuscular area of endometrium, there was an area showing a most unusual degree of hyperplasia of the peritoneal endothelial cells (Fig. 4). Here the cells were cuboidal, and were arranged to form branching tubules of varying size, giving the appearance of an adenoma. Proliferations of peritoneal endothelial cells, with resulting tubular and cystic peritoneal inclusions, are common in chronic pelvic inflammation, but are very rarely found to the degree shown in this section. The appearance therefore suggested the possibility that this area of peritoneal hyperplasia was an early stage in the metaplasia of peritoneal cells into "endometrial" glandular elements. Further, at a point between the endothelial tubules, areas of

typical endometrial stroma might be seen. The picture produced was thus one of invagination of the serosa with accompanying stromal changes.

Discussion

The presence of aberrant endometrial tissue in certain conditions of the ovary was recognized by Russell in 1899

FIG. 1.—Resected specimen of ileo-colic intussusception. (A) Apex of ileum with endometrial tissue.

FIG. 2.—Section cut from apex of the entering ileum, with (A) area of endometrial tissue and (B) area of hyperplasia of peritoneal endothelial cells. ($\times 5$.)

and yet the histogenesis of endometriosis is not completely explained. J. A. Sampson (1921) maintained that these lesions were due to implantations of the living endometrial cells over the surface of the peritoneum of the ovary, etc. These cells were supposed to be cast into the cavity of the uterus during menstruation, passed along the Fallopian

tubes, and finally settled at the site of the future lesion. Sampson has produced a great deal of evidence in support of this theory, and yet many objections to the general application of his views have been raised.

Emil Novak (1926) mentions the following: (1) Though retrograde menstruation may occur, it is a rarely observed phenomenon compared with the great frequency of endometriosis. (2) It is difficult to believe that endometrium thrown off in the uterus could enter the small orifice of the tube, travel outwards, and still be capable of implanting itself and growing upon the pelvic structures. (3) Endo-

FIG. 3.—Aberrant endometrial glands with abundant endometrial stroma within the muscularis of the ileum. ($\times 182$.)

FIG. 4.—Great hyperplasia of peritoneal endothelial cells forming tubules, suggestive of an early stage of metaplasia. ($\times 350$.)

metrium thrown off at menstruation is already degenerated or dead, so that it is not easy to conceive of it taking root in the peritoneum. (4) Experiments such as those of Jacobsen (1922), showing that endometrium can grow in the peritoneum, have dealt with the normal healthy endometrium of animals. (5) Experiments such as those of Heim (1929), in monkeys, in which a utero-abdominal fistula was created, have failed to show any development of endometrium, in spite of the fact that the menstrual blood was emptied freely into the abdomen.

It was suggested by Iwanoff (1898), and supported by Meyer and Kitai (1924), that the origin of the aberrant endometrial tissue was a serosal metaplasia. This was based on the fact that the entire epithelial apparatus of the female genital tract (endometrium, germinal epithelium of the surface of the ovary, etc.) is derived from the primitive coelomic epithelium of the urogenital fold and is a mere modification of the peritoneum. According to this theory, under the influence of ovarian hormonal stimulation the pelvic peritoneum forms tubular invaginations which sink into the subjacent tissue, and the flat endometrium of the involved peritoneum becomes columnar, thus producing an adenomatous condition.

The hypothesis of serosal metaplasia has received influential support, especially for the endometrial cysts of the ovary and endometriosis of the umbilicus, laparotomy scars, uterine ligaments, intestine, and recto-vaginal septum.

An explanation of the stimulus of metaplasia may be found, in the above case, in a study of the behaviour of epithelium in the presence of inflammation. Inflammation beneath a mucous or serous surface produces an infiltrative proliferation in the adjacent epithelium. This epithelial proliferation is at first a protective process, but it can overstep the limit of physiological hyperplasia by producing epithelial tubules which have all the appearances of glandular invasion. Peritoneal endothelium behaves in the same way, and in the present case, in which a chronic intussusception probably gave rise to such a stimulus, the glandular elements are seen to reproduce the exact structure of normal endometrial glands and stimulate a surrounding stroma which is an exact replica of endometrial stroma. Furthermore, these areas of typical endometrial tissue are seen in the sections to lie in immediate apposition to the hyperplastic tubules which are shown to be derived from the peritoneal endothelium.

It would seem that reversions of the epithelium under inflammatory, and perhaps ovarian, stimuli are possible in the adult peritoneum, and that the embryonic cells can reproduce endometrial structure.

Summary

A case of intussusception, with tissue showing all the appearances of endometrium situated at the apex of the entering ileum, is presented.

The microscopical appearances are suggestive of a metaplastic origin of the endometrial tissue and may be held to support the observations of Meyer and Kitai (1924), Novak (1926, 1947), Moench (1929), and others.

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NEONATAL BACT. COLI MENINGITIS AFTER PROLONGED LABOUR

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It is well known that *Bacterium coli* meningitis is relatively commoner in the newborn than in childhood or later life (Barron, 1918; Cruickshank, 1930; Craig, 1936; Rauch and Krinsky, 1940; Barrett *et al.*, 1942). The explanation given is that the newborn infant has not developed adequate resistance against the organism, although in later life it carries the same organism about in large numbers in relative safety (Cooke and Bell, 1922).

The route taken by the infecting organism has been the subject of some speculation, and numerous suggestions have been made. It is generally agreed, however, that the meninges are more commonly infected via the blood stream than by direct spread from adjacent areas. To support this contention Fothergill and Sweet (1933) demonstrated a *Bact. coli* bacteraemia at the same time that *Bact. coli* were found in the cerebrospinal fluid.

The original portal of entry is difficult to determine, but the large number of suggestions made, incriminating widely separated sources, indicate that infection may enter at any of many sites. The following portals of entry have been mentioned: middle ear (Scherer, 1894; Barron, 1918; Leathart, 1932; Craig, 1936), gut (Cooke and Bell, 1922; Barrett *et al.*, 1942; Ravid, 1935), lungs (Aschoff, 1897; Goldreich, 1902; Herrman, 1915), genito-urinary tract (Neal, 1926; Köplik, 1916), spina bifida (Forbes, quoted by Rauch and Krinsky, 1940), umbilicus (Hinsdale, 1899), and morbid conditions of skin, scalp, mucosae, and subcutaneous tissues (Craig, 1936). In addition transplacental bacteraemia, possibly arising from maternal pyelitis, has been suggested by Jahkola (1935), Karplus (1927), and Athenstaedt (1933). Aspiration of liquor infected with *Bact. coli*, causing a bronchopneumonia followed by bloodstream spread to the meninges, was suggested by Goldreich in his case. Aschoff held similar views. And, it might be noted, F. J. Browne (1921) considers aspiration of infected liquor a cause of bronchopneumonia.

In the following case it would seem that this was also the method and route of infection. The foetal head lay in the maternal pelvis, deeply engaged, for at least four days, and during the latter two days the cervix was between half and full dilatation. Further, both mother and child showed clinical signs of infection three days after delivery—the mother with *Bact. coli* pyelitis and endometritis and the child with fatal *Bact. coli* meningitis and bronchopneumonia.

Some previous cases have been reported in which the same organism was found in the maternal urine and lochia as was found in the child's cerebrospinal fluid (Jahkola, 1935; Trillat and Notter, 1939; Davis and Fernando, 1935).

Case History

A primipara aged 25 had labour surgically induced by a Drew Smythe catheter when 38 weeks pregnant. The indication was pre-eclamptic toxæmia with a rising blood pressure.