abdominal girth was 32½ in. (82.5 cm.) and no foetal heart was heard.

An *x*-ray film of the abdomen in the antero-posterior diameter was taken. It was not very clear owing to the hydramnios, and only one anencephalic foetus was definitely seen, the fact that a second one was present being easily missed. At this stage it was therefore thought that there was only one foetus.

She was admitted to hospital on August 29. A Drew Smythe induction was performed next day and 49 oz. (1,390 ml.) of brown liquor was obtained.

The induction did not stimulate her into labour, and subsequently she had a "pitocin" drip, a re-stretch of the cervix, and dihydroergotamine methanesulphonate, 0.25 mg. intramuscularly three times at four-hourly intervals. She eventually started in strong labour on September 6 and the cervix became fully dilated at 2 a.m. on the 7th.

At 2.35 a.m. the first foetus was born—breech presentation. It was macerated, and showed characteristic signs of an anencephalic female monster. Abdominal examination revealed the presence of a second foetus in longitudinal lie. No foetal heart was heard.

At 3.05 a.m. a vaginal examination was made and the membranes of the second foetus were ruptured. A large amount of brownish liquid was again obtained. On withdrawing the finger the placenta of the first foetus was expelled from the uterus. The second foetus—male—was born soon afterwards, and also showed typical features of an anencephalic monster. The placenta of the second twin failed to separate and had to be removed manually under general anaesthesia. The puerperium was uneventful.

Discussion

An added interest of the case is that it was a binovular pregnancy. The pathogenesis of the condition is that it is an arrest in development of brain and spinal cord due to failure of closure of the neural tube posteriorly about the fourth week. The neural plate, by exposure to amniotic fluid at the cephalic extremity, became atrophic.

No obvious environmental causes were to be found. The patient was a young primigravida with no preceding history of infections, menstrual disturbances, anaemia, nutritional deficiencies, or exposure to possible chemical or physical dangers. The fact that the foetuses differed in sex is against the aetiology being due to derangement of sex chromosomes. There is no family history of this abnormality or of frequent associated abnormalities such as spina bifida, hydrocephaly, or Klippel-Feil syndrome.

Summary

A case of binovular twin pregnancy in which both foetuses were anencephalic is described.

There was no evidence to incriminate any environmental or hereditary factors in this case.

I thank Dr. J. Hallum for permission to publish this case and Dr. M. Israelski for use of the *x*-ray report.

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Medical Memoranda

A Case of Cryptococcosis of the Colon

The following case of cryptococcosis has some interesting peculiarities.

Case Report

A 16-year-old male student from Rize complained of diarrhoea that had persisted for several years. Eight years ago he started having watery, foamy stools, containing some mucus, about five times a day. During the last few years he had abdominal pains, tenesmus, and bloody stools in addition. During this period he had twice been admitted to hospital without any improvement. There was nothing noteworthy in his past or his family history.

Physical examination revealed a mass, the size of an egg and slightly painful on pressure, in the left colic region. The faeces contained mucus, blood, and ascaris eggs. On rectoscopy the rectum was found to be hyperaemic. Blood count: erythrocytes, 3,660,000; Hb, 70%; leucocytes, 5,200; sedimentation rate, 15 at the end of the first hour on December 15, 1958, and 24 on January 3, 1959. The urine was normal and the Mantoux test negative. *X*-ray examination showed normal lungs and some narrowing in the descending colon.

Surgical exploration revealed a hard tumour-like mass adherent to the mesocolon, located in the descending colon next to the splenic angle. The sigmoid was free, and numerous lymph nodes of various sizes were present in the mesocolon. After liberation of the tumour mass from the spleen and kidney, to which it adhered very firmly, an end-to-end anastomosis was carried out. A Pezzer catheter was placed in the colon and the retroperitoneal region was drained. On the fifth post-operative day watery faeces started to ooze from the catheter, which was removed on the tenth post-operative day. Recovery was uneventful, except for the formation of a fistula on the site where the catheter had been inserted; secretions from this fistula however, gradually stopped. Drug therapy consisted of sulphadiazine and "mysteclin."

The resected part of the colon, 22 cm. long, was cut open. The wall of the upper part was hardened and thickened (5–7 mm.) and the lumen was narrowed (2 cm. in diameter). The wall of the lower part was 3–4 mm. in thickness and its lumen 3 cm. in diameter. Though the mucosal folds were well identified in the thin part, they seemed to have been smoothed out in the thicker portion and replaced by eroded areas with pseudopolypous formations. The mucosa was in general very congested, infiltrated, and oedematous. The thickened lower portion contained flat nodular masses, 1.5 to 2 cm. thick, which on section showed soft pinkish and in certain parts myxomatous round foci, 0.5–1 cm. in diameter. These masses were located between hard, fibrous, and whitish-yellow areas. In the serosa of the thin portion of the colon were two small, hard nodes with grey sections, and one red and soft node the size of a lentil.

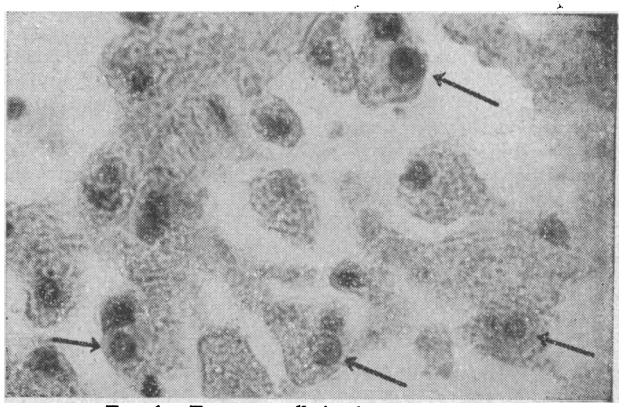

FIG. 1.—Fungous cells in the macrophages.

In the slides prepared from myxomatous masses of the serosa accumulations of histiocytes and, in some places, localized fatty tissue among bundles of collagenous fibres were observed. In the macrophage infiltrations there were a few lymphocytes, plasma cells, and, very rarely, polymorphonuclear leucocytes, but no giant cells.

Many macrophages (Fig. 1) contained one or, rarely, two round mycotic cells (Figs. 2 and 3), the size of a lymphocyte.

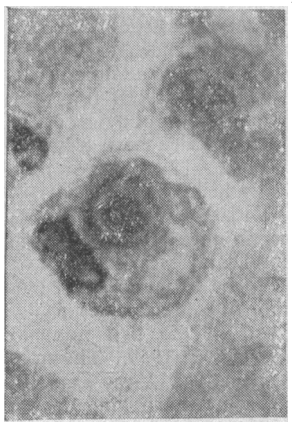

FIG. 2. — One intracellular fungous cell.

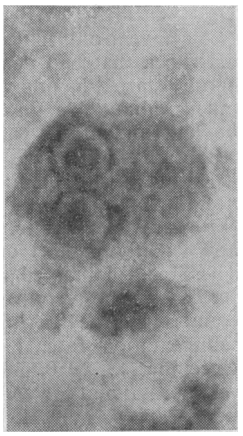

FIG. 3.—Two intracellular fungous cells.

Some of the fungous cells were surrounded by a clear transparent capsule. Extracellular fungous cells could be found with difficulty and only after a long search (Fig. 4). The diameter of the fungous cells was usually about 5–7 microns without the capsule and 7–10 microns with the capsule. Smaller cells, whose capsule could not be identified, were also observed. No mycelium formation was detected. When stained with haematoxylin and eosin the cytoplasm of the fungous cells was mainly pinkish grey, though some with grey-blue cytoplasm were also seen. The periphery of some cells was basophilic, and basophilic staining parts could be found. The capsules were very clearly visible in the slides stained with mucicarmine.

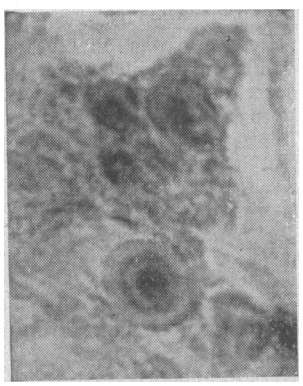

FIG. 4. — One extracellular fungous cell.

On account of the above-mentioned characteristics, which differentiate the fungous cells from *Blastomyces dermatitidis, Blastomyces brasiliensis, Coccidioides immitis, Candida albicans* and other *Candida* species, and *Histoplasma capsulatum*, it was concluded that the fungus observed was a cryptococcus.

The slides prepared from the pseudopolypous regions of the thickened wall of the colon showed fairly large ulcerated and eroded areas with little fibrin formation, but with an accumulation of macrophages and a few scattered multinucleated giant cells. A chronic inflammatory infiltration with histiocytes was present among the glands. The fungous cells were more numerous in the cell agglomerations of the serosa than in the macrophages of the ulcerated areas. In no giant cell was any fungous element found.

The regional nodes showed only characteristics of a sinusal inflammation, and no fungous element could be detected.

COMMENT

Cryptococcosis can be divided into two groups, localized and generalized. The case here reported belongs to the first group, and the lesion is localized to the descending colon. We could not find a case of similar location in the literature available to us. However, in generalized cryptococcosis mycotic elements can be detected in all tissues, including the intestines; for instance, in the first case of cryptococcosis reported from Turkey (Soysal, Unat, and Tahsinoglu, 1954) the parasites were detected in the lymphatic system of the intestines. In the present case no lesions attributable to cryptococcosis could be found either clinically or radiologically in any other part of the body.

In this case we are inclined to think that the portal of entry was the colon, though generally the respiratory tract, the skin, and the mucous membranes are considered the portal of entry (Littman and Zimmerman, 1956). Takos (1956) proved by experiments on monkeys that the digestive tract can be the site of primary inoculation. *Cryptococcus neoformans* was repeatedly recovered from soil (Emmons, 1951, 1955; Ajello, 1958) and milk (Carter and Young, 1950), besides normal skin and the gastro-intestinal tract of man (Benham, 1935).

Another interesting point in this case is the absence of gelatinous masses of free cryptococci and the rarity of giant cells. However, it has been known for a long time that cryptococci can be found in histiocytes. Baker and Haugen (1955), who studied the pathology of the disease in 26 cases, identified two principal types of tissue lesions. According to them, early lesions are gelatinous, while older lesions are granulomatous. These parasites are initially inert in tissues and form masses of fungous cells with but little surrounding inflammatory reaction; later they are phagocytosed by macrophages and giant cells. The course of the infection depends on the virulence of the strain and the resistance of the host. It has been proved that the thicker the capsule the more difficult the phagocytosis and the more virulent the strain of *Cryptococcus neoformans* (Drouhet, Segretain, and Aubert, 1950; Drouhet and Segretain, 1951). In our case we did not find fungous cells with very thick capsules, and this fact seems to explain their phagocytosis by histiocytes. While diarrhoea existed for eight years, it is difficult to claim that cryptococcosis was the cause from the very beginning, but it certainly accounts for the bloody diarrhoea during the last few years. It can be assumed that the resistance of the body and the colon was lowered by diarrhoea and thus the fungous cells, with limited pathogenicity, had a chance to penetrate and proliferate in the tissues without generalization.

We were unable to make cultures because the specimen had been fixed with formol.

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