Since writing this report a case of scarlet fever has recently arisen (June 18th) in a boy who was a Dick-positive contact with the first case of scarlet fever last term, and who had then been passively immunized. In consequence of this case, all the available passively immunized boys of last term, thirty-two in number, have been Dick-tested again, with the result that thirty-one have been found to be positive and only one negative.

I most gratefully acknowledge my thanks to Dr. J. Alison Glover and Dr. Scott for their advice and encouragement, and to the latter for his practical help in starting the immunization; also to Dr. Pickering, without whose constant help in all the inoculations the task would have been impossible; and, lastly, again to Dr. Griffith, whose investigations were so helpful and interesting, and who has allowed me to make use of his work.

CHRONIC AMOEBIASIS AND CHRONIC APPENDICITIS

BY

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A large number of patients seen at the European Hospital, Nairobi, complained of vague abdominal symptoms, and a number of these had had their appendices removed previously. The main symptoms were: flatulent dyspepsia; biliousness; vague abdominal discomfort; occasional acute pains in the right iliac fossa, often of a dragging-down character; general malaise; headaches; backaches; joint pains. The signs usually found were: a peculiar earthy pallor; a dirty, furred tongue; an enlarged and tender liver; a thickened and tender colon.

While working among natives in the Reserves I found that signs of hepatitis predominated over others in cases of amoebic dysentery. As an enlarged liver was common to all these cases in Europeans in Nairobi, a more careful examination of stools was instituted. The system in vogue at the time, whereby a boy carried the specimens to the laboratory each day, was unsatisfactory; so eventually Dr. Tonking of the Medical Research Laboratory came to examine stools at the European Hospital. This resulted in a startling increase in the number of stools found to contain *E. histolytica*, as not only were stools examined fresh, but repeat examinations could be obtained. In five months Tonking found 125 cases positive at the European Hospital alone, while only 366 among both Europeans and natives had been reported from the Medical Research Laboratory during the previous ten years. The patients who had positive stools improved on anti-amoebic treatment, and so it was decided to treat on similar lines those patients complaining of the same symptoms and showing the same signs, but whose stools were negative. They also improved.

Differential Diagnosis

The differentiation between the so-called chronic appendicitis and amoebiasis is not difficult, providing the prevalence of amoebiasis is borne in mind. The following table gives the points of difference.

Chronic Appendicitis	Chronic Amoebiasis
Discomfort in the right iliac fossa.	Discomfort in the right iliac fossa.
Epigastric pain.	Epigastric pain.
Heartburn uncommon.	Heartburn very common.
Not relieved by alkalis.	Relieved by alkalis.
Nausea and vomiting.	Nausea common, vomiting uncommon.
Constipation.	Constipation.
Pain localized.	Pain diffuse.
The physical examination may be negative.	The physical examination is never negative.
Pressure in the right iliac fossa may cause discomfort in the epigastrium or at the site of pressure.	The pain is marked over the caecum and hepatic flexure; slight over the sigmoid.
Liver not enlarged.	Liver almost invariably affected in some degree.
Does not run in families.	Tendency to family infection.

The main points of differential diagnosis, therefore, are the presence in chronic amoebiasis of: (1) heartburn; (2) relief by alkalis; (3) enlargement of the liver; (4) the method by which the patient indicates the site of pain—in amoebiasis the whole hand is used and the site of pain varies; (5) the tendency for the disease to run in families. The enlargement of the liver is often only upwards, and percussion of the liver with the patient lying on the right side or sitting up will alone demonstrate the increase in its size. Fine crepitations over the liver between the right axillary line and the right nipple line can often be heard. The thickening of the colon is most marked over the caecum, and it is this painful, thickened caecum which is mistaken for appendicitis.

Amoebae in the stools are often found with difficulty, as not all patients will send their stools for examination daily for six to ten days. Amoebae have a periodicity which varies in different individuals, and only at these periods can amoebae be demonstrated in the stools. Further, amoebae may lie dormant for months, and it is this dormant period which is sometimes erroneously described as a cure of the condition, or used as an argument against the presence of amoebic infection.

Two typical histories illustrating the confusion between the two diseases are as follows:

CASE I

A female, aged 22, was quite well until 1930, when she suffered from indigestion and flatulence. For this her appendix was removed in March, 1930. She felt no better for it. At the end of 1930 one stool was examined and reported as negative. A test meal showed hyperacidity. She was next x-rayed for duodenal ulcer, but nothing was found. An operation was suggested, but refused. She then went to England, where further x-ray photographs were taken: a diagnosis of acute rheumatism was made. The patient was given steam baths for six months, and massage; still no improvement.

In October, 1931, she returned to Kenya, and was then having two to three attacks of acute pain daily. In April, 1933, her stools were examined and found to be full of *E. histolytica*. Treatment for amoebiasis restored her health.

CASE II

A female, aged 29, had had, in 1923, diarrhoea in Sydney, Australia, followed by constipation, soreness of the abdomen, and periodical attacks of diarrhoea. Her condition gradually got worse, until in February, 1927, she had violent abdominal pains accompanied by vomiting. The attack passed off. During the next two months she had recurrences of abdominal pain, mucus in her stools, and occasional attacks of diarrhoea. A diagnosis of colitis was made. At the end of 1927 the appendix was removed for a return of abdominal pains. She felt better after this, but the colitis continued. She next suffered from urticaria.

In 1930 the patient had pleurisy; the sputum was examined repeatedly for tubercle, but none was ever found. Later she had a maculo-papular rash, for which no diagnosis was given. In August, 1930, there was another attack of pleurisy. X-ray investigation and sputum tests were all negative, but a low fever persisted (about 99° F.), accompanied by a slight cough. In 1931 she came to East Africa, and remained well for eight months. At the end of 1931 there was another attack of colitis, associated with rheumatic pains and bad headaches. In 1933, after a further attack of colitis, the stools were examined. The first was negative, but the second and subsequent ones showed *E. histolytica*. The patient's condition improved immediately on the institution of treatment for amoebiasis.

This case also illustrates other features of chronic amoebiasis besides the appendicular type of pain. These are (a) the rashes—signs of toxæmia; (b) cough and pleurisy, which are usually present in amoebic hepatitis; and (c) rheumatic pains, probably toxæmic in origin.

Treatment

The treatment of chronic amoebiasis is not satisfactory. Emetine is the one drug that will control the condition

and cause an abatement of symptoms. Its most pleasing results are in cases in which the liver is most affected. Drugs by mouth, such as carbarsone, stovarsol, and kurchi bark, help to control the disease. Treatment by these drugs and emetine will not, however, effect a cure unless the secondary bowel infection, which is always present in chronic cases, is dealt with. Most cases will produce an agglutinating coliform bacillus, and an autogenous vaccine will materially assist in controlling the infection.

Another reason why this disease is disappointing to treat in East Africa is the frequent reinfections which occur, since amoebiasis is far from uncommon among Africans, and cooks and houseboys may be carriers of the disease. As an example, I quote the figures from Nairobi Prison: in 1933, one case reported; in 1934, fifteen cases; during the first four months of 1935, eighty-eight cases. The clinical diagnosis for the 1935 cases rested primarily

on the enlargement of the liver. Several examinations of stools were necessary in most of the cases to confirm the clinical diagnosis: in one instance thirty examinations were carried out. In conclusion, provided the prevalence of amoebiasis is borne in mind, the condition commonly ascribed to chronic appendicitis will be found to have a protozoal origin.

Summary

1. Many cases of so-called appendicitis, and in which the appendix has been removed, were found to respond to anti-amoebic treatment.

2. A differential diagnosis between chronic appendicitis and chronic amoebic dysentery is given.

3. The prevalence of amoebiasis among Europeans and natives is shown.

4. Two typical cases of amoebiasis in which the appendices had been removed are recorded.

Clinical Memoranda

Cavernous Sinus Thrombosis with Recovery

The following case of recovery from cavernous sinus thrombosis, following nasal infection, seems worthy of mention.

Case Record

A youth of 18 years was first seen on April 5th, 1935, complaining of left-sided nasal obstruction and recurrent epistaxis, becoming increasingly worse since an accident in boyhood. On examination his nasal septum was found to be markedly displaced to the left, with a deep concavity on the right side. Investigation of the nasal sinuses and post-nasal space was negative, there being no adenoids. A submucous resection of the nasal septum, which presented no difficulties, was performed under general anaesthesia on April 10th, but it was noted on completion that the right mucoperichondrial flap sagged towards the right, tending to form a pocket.

On the second day following the operation the temperature rose to 102° F., but rapidly subsided twelve hours later, with profuse perspiration. The temperature again rose to 101° on the fourth day, but it gradually subsided, becoming normal by the seventh day. A mild influenzal epidemic was at this time prevalent in the ward, and in the absence of any apparent intranasal infection this was believed to be the cause of the pyrexia. On the eleventh day, however, the patient complained of frontal headache, "like a weight on his eyeballs," especially severe around the left eye. Ocular movements, particularly on looking upwards or downwards, were painful; the temperature was 97.8°. The interior of the nose showed some generalized mucosal oedema, with no particular localization to the septum. Nothing abnormal was discovered in the central nervous system.

On the twelfth day the morning temperature was 102.8°. There was complaint of severe frontal and occipital headache, with photophobia, marked neck rigidity, and a positive Kernig's sign. Slight left external rectus ocular paresis was present: the headache was increased by the slightest movement. On nasal examination a boggy swelling was observed on the right of the nasal septum opposite the middle turbinate, which on incision revealed about a teaspoonful of creamy pus. A septal abscess was drained by a looped strand of silkworm-gut. Culture of pus was sterile.

On the thirteenth day the temperature was 101.4°. Headache was more severe, and the patient was becoming lethargic. There was oedema of both left eyelids, with proptosis, and later in the day the right eye became similarly affected, though to a lesser degree. Gradually increasing left chemosis developed later, with increased neck rigidity and a more marked Kernig's sign. The left external rectus ocular paresis became complete. The abdominal reflexes were diminished on the left side, and there was an indefinite left plantar response. The optic disks showed slight papilloedema, more marked on the left side. Slight bilateral tactile hyper-

aesthesia was noted in the supra- and infra-orbital regions. Lumbar puncture gave a clear fluid under pressure. The findings on examination of this fluid were as follows: albumin, 60 mg.; globulin, a trace; sugar present; chlorides slightly decreased; cells 11 per c.mm. (lymphocytes). Culture was sterile.

The appearances, therefore, were those of a cavernous sinus thrombosis—probably septic—with meningeal irritation. Mr. Rowbottom kindly saw the case in consultation, and expressed the opinion that, as the condition was not localized, surgical approach was inadvisable. In the belief that the infection was in all probability streptococcal in origin, from the nasal infection, 20 c.cm. of polyvalent antistreptococcal serum was administered intramuscularly on the thirteenth day, and a course of hexamine (10 grains four-hourly) started.

On the fourteenth day the morning temperature was 100.6°, the headache being slightly better and the patient less lethargic. Chemosis and proptosis were still present and the left abdominal reflexes absent. The optic disks were normal. Antistreptococcal serum (20 c.cm.) was given intravenously, followed half an hour later by a lumbar puncture. The cerebro-spinal fluid was now slightly turbid and under pressure, but was again sterile. The silkworm-gut drain was renewed. Considerable improvement was observed by the evening, and the chemosis and proptosis were diminishing; temperature 98.8°.

On the fifteenth day there was much improvement. The headache was better and the left eye was becoming normal. The abdominal reflexes had returned. There was no neck rigidity and no Kernig's sign. The silkworm-gut drain was removed on the sixteenth day. The condition continued to improve.

On the nineteenth day the temperature was 100.4°, and there was slight headache. Intravenous antistreptococcal serum (20 c.cm.) was again administered.

On the twentieth day the septal abscess had re-formed. To facilitate drainage a small window resection of the right wall of the septal abscess was performed under local anaesthesia.

Subsequent progress continued satisfactorily, and on May 24th, 1935, the patient was discharged from hospital.

Summary

1. The late onset of cavernous sinus thrombosis—namely, thirteen days after what appeared in the early stages to be an uncomplicated submucous resection of the nasal septum.

2. A satisfactory response following treatment by incision and drainage of the septal abscess, together with intravenous antistreptococcal (polyvalent) serum therapy and lumbar puncture.

My thanks are due to Mr. F. Holt Diggle for permission to publish the notes of this case.

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