ulcerated in October 1950, *i.e.* one year and four months after the appearance of the depigmented patches. Leprotic or pyogenic origin was excluded by the negative clinical and bacteriological findings.

It is a well-known fact that unlike oriental sore post-kala-azar dermal leishmaniasis does not usually ulcerate, so that the occurrence of ulceration in this case was as unusual as the fact of its occurrence in Assam. The case is complicated, however, by the positive Kahn test.*

There were no signs of tuberculous disease and the Mantoux test was negative. No mycobacterium tuberculosis were found in the scrapings from the ulcers, which showed only a heavy infection of L.D. bodies.

Summary

A case of post-kala-azar dermal leishmaniasis with ulceration is described. There was no coexistent leprosy or tuberculosis. The case is complicated by the presence of syphilis.

My thanks are due to Dr. T. Norman, Principal Medical Officer, Jhanzie Tea Association, Assam, for his kind permission to report this case and for going through the case report.

*W.R. is known to be positive in kala-azar.—EDITOR, *I.M.G.*

AMŒBIC INFECTION OF THE FEMALE GENITAL ORGANS WITH REPORT OF A CASE

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Introduction

Entamœba histolytica commonly invades the large gut but sometimes it invades the lower part of the ileum and the appendix. Metastatic infection commonly occurs in the liver but sometimes in the lungs, brain and other parts also.

Amœbic invasion of the vulva, vagina, cervix and uterus has been reported but the number of cases is very small and most of the textbooks give a very meagre account of them. The rarity of the condition would justify placing on record the following case of amœbic vaginitis which the author recently had under his care.

Case report

Mrs. U. K., Hindu female, aged 31, was admitted into the Lake Medical College Hospital on 14th September, 1950. She complained of profuse white discharge per vagina for 2½ months. Recently the discharge had become offensive and blood-stained. There was some lower abdominal pain but no soreness or pruritus of vulva.

O.H.—Patient had one miscarriage at 6 months 14 years ago, and one baby at term 8 years ago.

M.H.—Periods were regular 5/30 days with some pain, L.M.P. 1st September, 1950.

Past illness.—Patient has been having slight leucorrhœa off and on since the birth of her child 8 years ago but the discharge was never very profuse.

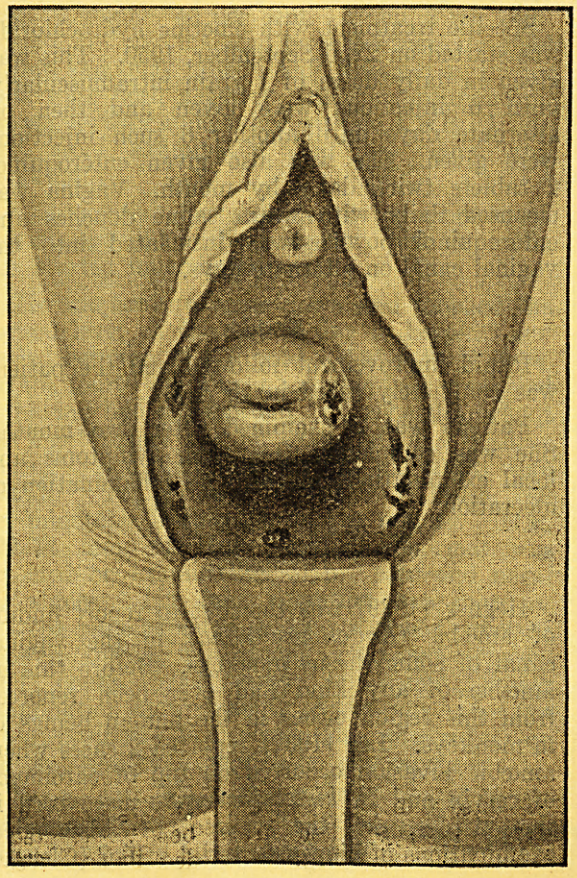
Present illness.—Patient suffered from dysentery for about one month prior to the onset of her present symptoms. She was relieved of her dysenteric symptoms with some medicines by mouth. After the dysenteric symptoms had subsided the vaginal discharge gradually became worse. She was treated with sulphonamides and big doses of penicillin without any relief of her symptoms.

General examination

Liver was slightly enlarged and there was some tenderness over the suprapubic region. No other abnormality detected in the other organs.

Local examination

There was no sign of inflammation or excoriation of the vulva. On separating the labia profuse purulent discharge came out. Vagina was full of purulent discharge. After swabbing the vaginal canal multiple ulcers could be seen on the vaginal walls. There was one big ulcer,



3 cm. × 2 cm., on the left side of the cervix extending from the outer periphery of the cervix to the vaginal vault, between 1 and 5 o'clock positions. The ulcer had irregular overhanging margins. The floor was red with scattered yellowish spots. The ulcer bled easily. The area immediately around the external os was healthy.

There was another big ulcer on the upper half of the anterior vaginal wall between 9 and 11 o'clock positions, about 4 cm. × ¾ cm. The character of the margins and the floor of the ulcer was similar to those described above. There were a few more smaller superficial ulcers scattered over different parts of the vaginal mucous membrane. The vaginal mucous membrane as a whole looked œdematous.

Uterus was retroverted normal size with restricted mobility. Tubes and ovaries were not palpable. The perineum was relaxed.

Laboratory findings

Hb. 75 *per cent* (*Hellige*).—W.B.C. 10,800 per c.mm.

Smear of the pus.—No intracellular Gram-negative diplococci were found nor any A.F.B. Wet preparations of the pus showed no amœba but similar preparations of swabs taken directly from the ulcers showed *Entamœba histolytica* in its vegetative forms.

Treatment

Specific treatment with emetine hydrochloride was started on 22nd September, 1950. This was given in daily doses of 1 grain intramuscularly until 6 injections were given and then on alternate days until another 6 such injections were given. She was also given enteroquinol 2 tablets twice daily by mouth. Vagina was cleansed daily with warm saline douches and enteroquinol powder was insufflated into the vaginal canal on alternate days.

Result

Rapid improvement followed and the patient was completely cured in 15 days.

Patient was re-examined after two months. She was completely free from symptoms and local examinations revealed no inflammation or ulceration.

Discussion

Hegner (1929) was the first to report finding of amœba (*Endolimax nana*) in the vaginal canal of some rhesus monkeys. Before this no amœbæ of any kind had ever been reported from this region either in the human beings or in monkeys. Lee (1932) reported 2 cases where amœbic invasion occurred on top of carcinoma of cervix and *E. histolytica* were discovered in the tissues, in the course of histological examinations of biopsy material. Hsu (Cleland, 1944) reported cases of amœbic ulceration implanted upon condylomatous growths of vulva and on carcinoma of cervix. Since then cases of amœbic vaginitis were reported by Bacigalupo *et al.* (quoted by Weinstein and Weed, 1948), May (1943), Morse and Seaton (1943), Garin (1947), Sen (1949) and Misra (1950). De Rivas (1944) reported a case of amœbiasis of uterus and Cleland (1944) reported a case of amœbic infection of vulva. In both these cases diagnosis was made at autopsy by the finding of *E. histolytica* in the tissues. In Cleland's case amœbic infection complicated granuloma pudendi.

Bickers (1943) studied 200 cases of leucorrhœa and observed that 0.5 per cent were caused by *E. histolytica,* but he did not report any case.

So far about 20 cases of amœbic infection of the female genital organs have been reported, of these 18 had vaginal lesions. In about 70 per cent of these cases the cervix was involved. In 25 per cent of the cases the uterus was soft and enlarged and uterine infection was suspected but only in 15 per cent (3 cases) uterine infection was definitely proved. Amœbic invasion of vulva appears to be most uncommon and the only reported cases are those of Hsu and Cleland.

From a study of the reported cases it appears that amœbic infection of the female genital organs has no relation to age or parity. The youngest patient in the reported series is 16 years and the oldest is 70 years. It has been suggested by some authors that relaxed outlet in multipara would favour ascending infection from the perineal region, but several cases have been reported in nulliparous women.

The reported cases though small in number show a geographical distribution which generally corresponds with high incidence of intestinal amœbiasis. In about 80 per cent of the reported cases a history of chronic dysentery could be traced but in about 20 per cent no such history was present and in about 50 per cent stool examination did not show any *E. histolytica.*

Amœbic infection of the female genital organs seldom occurred as a complication of acute dysentery. It is possible that treatment with emetine in these cases of acute dysentery not only cures the bowel condition but also cures any microscopic lesion, in the genital organs, before it can produce any symptom.

No fistulous tract communicating with the rectum was present in any of the reported cases. Fæcal contamination from the perianal region was undoubtedly responsible for the infection of the genital tract. What additional factors were responsible is not quite clear. But pathological states of vulva, vagina or cervix were present in a proportion of the cases and these have no doubt some ætiological significance. 'Relaxed outlet', 'Coitus', 'Sitting in a bath tub' or 'Method of ablution practised in India' have all been suggested as factors that helped to

carry the infection upwards through the vaginal introitus.

Amœbæ have been demonstrated in ovarian abscess and in inflamed tubes (quoted by Stitt and others). The infection of these organs was probably the result of direct extension from the bowel lesions. There is no evidence to suggest that ascending infection from vagina was responsible.

Blood-stained vaginal discharge was the most prominent symptom in the majority of the reported cases. Sometimes there was frank bleeding and sometimes only a purulent discharge per vagina. Some lower abdominal pain or burning during micturition was sometimes present.

The ulcers when well developed have very characteristic appearance. They are punched out in character with slightly overhanging irregular margins which bleed easily. The floor is red but sometimes it contains yellowish sloughs. The cervical lesions are usually eccentric in position. Both the vagina and the cervix are usually soft and œdematous. The uterus when involved is soft and enlarged.

Vegetative forms of *E. histolytica* were identified in all the reported cases. In some of the cases the amœbæ were found in the tissues, in others they were demonstrated in the discharge. A wet preparation of the discharge as well as of the scrapings from the ulcers should always be examined in all suspected cases.

Rapid cure with emetine injections with or without local treatment has been the rule in all the diagnosed cases.

Some authors reported more than one case of amœbic vaginitis and Weinstein and Weed reported as many as four. It may be concluded that if cases of leucorrhœa be thoroughly investigated for *E. histolytica* as a routine, we would probably find that amœbic vaginitis is not as rare as it appears.

REFERENCES

Bickers, W. (1943) .. *Virginia Med. Monthly*, **70**, 135.

Cleland, J. B. (1944) .. *J. Trop. Med. and Hyg.*, **47**, 54.

De Rivas, D. (1944) .. *Amer. J. Trop. Med.*, **24**, 185.

Garin, H. (1947) .. *South African Med. J.*, **21**, 32.

Hegner, R. (1929) .. *J. Parasitol.*, **16**, 91.

Lee, S. W. (1932) .. *Chinese Med. J.*, **46**, 1096.

May, M. Y. (1943) .. *Indian Med. Gaz.*, **78**, 250.

Misra, S. (1950) .. *R. G. Kar Med. College Magazine*, **19**, 139.

Morse, E. M., and Seaton, S. P. (1943). *Amer. J. Trop. Med.*, **23**, 325.

Sen, N. C. (1949) .. *Brit. Med. J.*, *i*, 808.

Weinstein, B. B., and Weed, J. C. (1948). *Amer. J. Obstet. and Gyn.*, **56**, 180.

PNEUMOCOCCAL MENINGITIS TREATED WITH PENICILLIN AND AUREOMYCIN

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V. Goriah, Hindu male, aged 30 years, was admitted on 26th February, 1951, with history of fever from the day before, pain on the right side of chest and cough with scanty expectoration.

The onset was sudden with chill and rigor.

Nothing significant could be elicited from the family history or past history except an attack of malarial fever a few months previously.

On examination.—The patient was moderately built and poorly nourished. He was very restless with an irritating cough. There was no cyanosis, no jaundice and no glandular enlargement. Anæmia was not very marked. Temperature 103.6°F. Pulse 116 per minute, regular. Respiration 48 per minute. B.P. 120/64 mm. of Hg. There was no neck stiffness. Kernig's sign was negative. There was evidence of consolidation of the mid and lower zone of the right lung. His abdomen was slightly tympanitic. His spleen and liver were both palpable one finger beneath the costal arch.

Blood examination.—Hb. 65 per cent (Tallqvist). W.B.C. 3,600 per c.mm. Poly. 58 per cent. Lympho. 40 per cent. Mono. 2 per cent. Eosino. nil. No malarial parasites were detected in thin and thick films. Aldehyde and antimony tests were both negative. Sputum examination showed a rusty appearance with no A.F.B. Urine and stool examination did not show any abnormality.

A diagnosis of right-sided lobar pneumonia was made and specific treatment with penicillin was started immediately.

His temperature came down and touched the normal level on the second day in hospital and varied between 98.4°F. and 100.6°F. for 2 days. On the fourth day in the morning he complained of having had a persistent headache throughout the night before and on examination a positive Kernig's sign and stiffness of the neck were found. Temperature 100.6°F. Pulse 68 per minute. Respiration 32 per minute. B.P. 120/74 mm. of Hg. Nothing abnormal was detected on examination of his ears and nasal air sinuses. His lung condition was more or less the same as noted before. Lumbar puncture showed turbid fluid under increased tension. Cell count 5,000 cells per c.mm. Smear examination showed plenty of pus cells and a few Gram-positive diplococci. The organisms on culture were confirmed to be pneumococci.

Treatment.—Administration of penicillin by intramuscular injections was continued and a total of 42 lacs of units was given in all, in 10