

between the antimony resistant and non-resistant cases was observed. It appears that an antimony resistant case is not necessarily pentamidine resistant and therefore does not require relatively higher dose for cure. So far as relapse rate is concerned the drug is inferior to stilbamidine or neostibosan.

10. No neurological late toxic manifestations, as seen in some cases treated with stilbamidine, were seen.

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TUBERCULOSIS OF THE KIDNEY

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THE writer had during the course of last 12 months opportunities for treating a number of cases of tuberculosis of the kidney and ureter, and at one time he had a series of three very similar cases under his care. In the course of management of these cases he made a number of observations and learnt a few facts concerning the progress of the tuberculous lesion in the kidney and the subsequent post-operative period that may be worth recording.

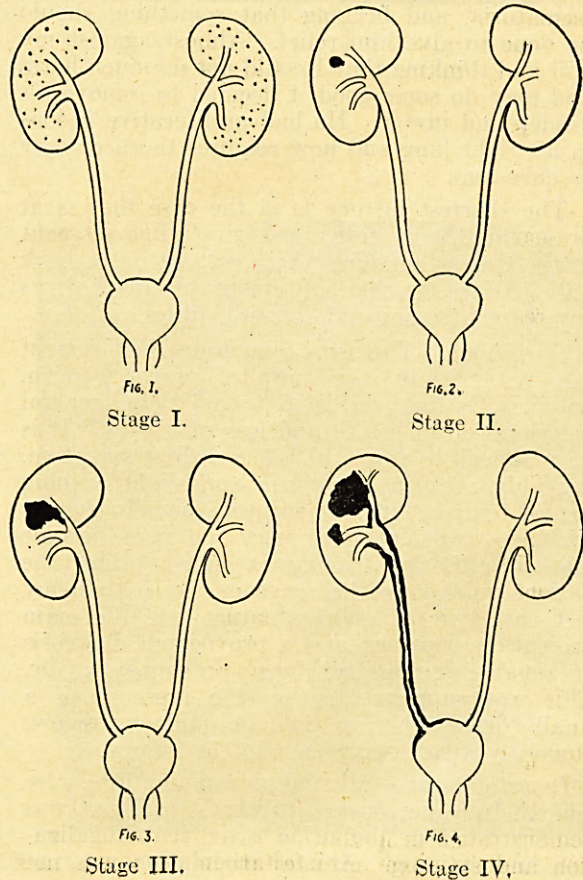
He thinks it will be generally agreed that tuberculosis of the urinary tract is always secondary to a primary thoracic or glandular infection, though at the time the patient presents himself for investigation, the primary infection may not always be obvious. The initial lesion in the lungs may have completely healed leaving only a tiny dense shadow discernible only by very careful examination or there may not be any shadow at all. In his present series there were 5 cases in which there were evidences of old proliferative lesions in the lungs. In the other two there was no evidence of primary infection. Dukes (1948) of St. Mark's Hospital, London, has written recently on the life history of the tuberculous lesion in the kidney and has postulated a very interesting series of events. According to him, the lesion in the kidney passes through four well-demarcated stages and it is possible by critically analysing signs and symptoms to determine the stage of the disease.

In stage I, according to Dukes, there are multiple showers of tubercle bacilli in the substance of both the kidneys in the form of multiple emboli from a primary source of infection most commonly in the lung (figure 1). Not all these multiple emboli are destined to grow and flourish. In stage II (figure 2) one finds that almost all the emboli have disappeared and only one focus in one of the kidneys has thrived and has continued to grow. The multiple emboli and the persistent enlarging focus are situated in the cortex and the pelvis and the calyces are yet completely free.

During these two stages symptoms are not marked and are mostly of general nature. There may be certain amount of frequency, fever and constitutional disturbance but urine is otherwise normal and pyelographic appearances show no change.

In stage III, the persistent focus has become much bigger (figure 3), perhaps has caseated and formed a cavity but the important thing is that it now ulcerates into the calyces and pelvis and it is at the stage that urinary symptoms become profound and disturbing; hæmaturia is often present, and pyelographic appearances show profound change.

Stage IV is only a further extension of the tuberculous process down the pelvis and the ureter and affecting the orifice of the ureter and the lumen of the bladder (figure 4). During



this stage bladder symptoms become very marked and the patient passes very agonizing and painful days with constant dysuria, frequent hæmaturia and pyuria, and rapidly loses ground.

Dukes' present classification like the one he propounded in neoplasm of the rectum is interesting and at the same time practical and certainly better than the ones mentioned in pathological textbooks, the caseous type, the cavitory type, the cretaceous type and the miliary type. In my small series, 4 cases were in stage IV and 3 in stage III.

Age incidence.—The average age incidence was twenty-nine, the youngest being 18 and the oldest 39. This is in keeping with the figures of other observers and it may be said that tuberculosis of the kidney is a disease of the 2nd, 3rd and 4th decades of life and is uncommon in the very young or the very old.

Sex.—Five cases were in males and two in females.

History.—The longest history was 7 years. This was a case whom I had admitted previously in the hospital 4 years ago for the same condition and the surgeon under whose care he was at the time had deemed operative interference too hazardous and not advisable in view of the affection in his lungs. He came to me again early last year complaining of incessant polyuria, unbearable dysuria and frequent hæmaturia, and begging that something should be done to give him relief. Almost against my will and thinking that I could not do much harm and may do some good, I decided to remove his kidney and ureter. He had proliferative lesions in his right lung and now requires thoracoplasty to cure him.

The shortest history is in the case that is at present under my care, an Anglo-Indian sergeant in the Calcutta Police whose history dates back only to last October. Previous to this he was asymptomatic and did his full duties.

Symptoms.—The usual symptoms that brought the patients to the hospital were dysuria, polyuria, hæmaturia, fever, renal pain, general deterioration of health and loss of weight. It is very difficult to say what is the earliest symptom, probably nocturnal polyuria and slight evening rise of temperature but surgeons usually do not see these cases until dysuria and pain become marked. Early hæmaturia may bring the patient quickly to the surgeons' notice. In my last case pain of colicky nature was the main presenting symptom and a provisional diagnosis of renal calculus had been previously made. This was supplemented by the presence of a small dense shadow in the kidney region, obviously a calcareous spot in the kidney.

Investigations.—All routine examinations were made. In four cases tubercle bacillus was demonstrated in the urine after centrifugalization and staining. Animal inoculation was not

considered feasible in view of the long delay required for such examination. Intravenous pyelography was done in all the cases and revealed various pyelographic appearances from total non-excretion to abnormal appearances of the calyces or to hydronephrosis. Cystoscopy was done in all the cases except the last one. Almost in every case the bladder showed evidences of chronic cystitis, engorgement and œdema. Ureteric catheterization and retrograde pyelography were possible in only two cases.

Operation.—In all the cases except one, the writer has extirpated the diseased kidney together with almost the whole of the ureter (figures 5 and 6, plate XV). He put the patient on his back in the beginning, made a 3-inch midline suprapubic incision, isolated, ligated and divided the ureter as close to the bladder as possible. Then the patient was turned on his side and after the kidney pedicle was divided and secured, finger separation brought the whole of the ureter out together with kidney. The importance of removal of the ureter together with the kidney is nowadays being widely stressed and seeing that all the cases belonged to stage IV or late stage III it seemed very necessary.

Post-operative period.—A troublesome post-operative complication that occurred in three of the cases and is in course of occurring in another is the slow breaking down of the wound, starting at about the 12th, 13th or 14th day. In all the cases the wound seemed to heal without trouble and only after the stitches had been taken out slight serous discharge and slow gaping became evident and continued for a long time. In a particularly serious case (figure 5, plate XV) practically the whole of the incision line gave way, the muscles seemed to fall apart and one was looking into a deep hole. The patient began to run slight temperature, granulation tissue was very slow in growing and progress was almost at a stand still. The patient seemed on the downward path. Many drugs including penicillin were tried without the slightest response.

At this time he was given a course of streptomycin and the response even after the first gramme was very pleasing. The fever quickly responded and the refractory wound quickly showed signs of healing. The course of streptomycin had to be repeated twice or thrice and in the writer's opinion made the essential difference between recovery and death. This particular complication has been happening so constantly in the last few cases and the beneficial response with streptomycin has been so marked that the writer now intends in his next series of cases to administer the drug from the beginning of the post-operative period as a prophylactic measure.

Results.—There has been no mortality in these 7 cases; five of them have markedly improved. Two have gone back to work. One case who is an employee of the Calcutta Fire

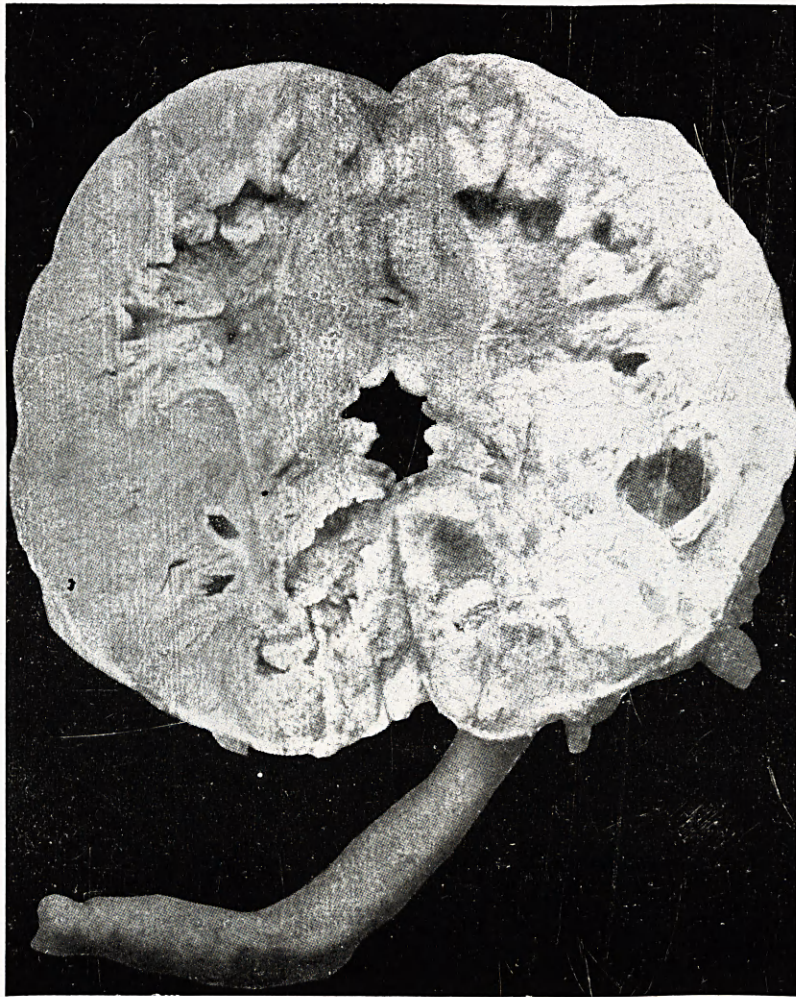


Fig. 5.—Showing ulceration, caseation and cavitation of the kidney.
The ureter is thickened.

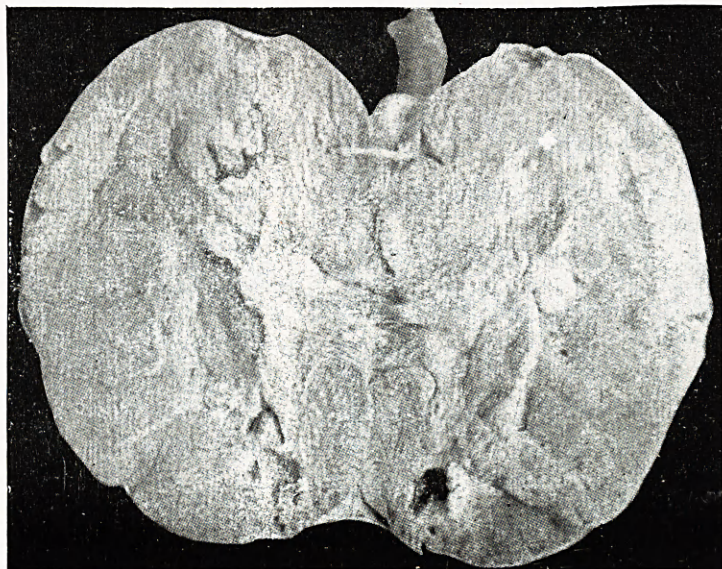


Fig. 6.—Showing ulceration and cavitation. But less pronounced
than in figure 5.

Brigade has started his duties. The man with the worst kidney and lesion in his lung has been keeping good health and now has no urinary symptoms. He passes urine 5 or 6 times a day and has undisturbed sleep at night. One case, youngest, has, I am told, not been keeping well and still has frequency and dysuria.

Summary

1. The life history of tuberculous disease of the kidney and its surgical treatment as judged from a small series of cases is presented.

2. Cuthbert Dukes' views as regards the pathology of the disease are mentioned.

3. An important post-operative finding, *viz*, late and gradual disruption of the wound, is discussed and its rapid response to streptomycin is stressed.

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TWO CASES OF AMŒBIC LIVER ABSCESS WITH COMPLICATIONS

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AMŒBIC abscess of the liver when not properly treated tends to increase in size and often ruptures into the adjacent tissues, *viz*, lung, pleura, peritoneum, pericardium or abdominal wall, and less commonly into the stomach, duodenum or hepatic flexure. An effusion may occur into the pleural or peritoneal cavities as a result of direct spread of the inflammation from the advancing abscess. Abscess in the lung may result from extension of inflammation through the diaphragm or as a metastatic condition. Amœbic abscesses have also been observed in brain and spleen. Two cases that illustrate some of the important complications of an amœbic abscess of the liver are described below.

Case reports : Case 1

The patient was admitted to hospital on the 14th July, 1942, with a complaint of pain in the abdomen. He was operated upon for perinephric abscess on 19th July, 1942. No pus was found and the wound closed. On 29th July, 1942, he showed a cystic swelling in the lower abdomen, extending up to within 1 inch of the umbilicus. His total leucocytic count was 14,800 per c.mm. with polymorphs 82.0 per cent. A diagnosis of pelvic abscess was made and the patient was operated upon on 30th July, 1942. Two pints of pus were evacuated. There was some improvement in his general condition. The total leucocytic count came down to 9,000 per c.mm. and polymorphs to 66.0 per cent. He died on 16th August, 1942.

Post-mortem findings.—Both the paracolic gutters showed free purulent fluid. The cæcum,

ascending colon and descending colon were slightly adherent to the posterior abdominal wall. The liver showed adhesions to the diaphragm at its upper surface and to the lesser curvature at its posterior surface. Left lobe showed an abscess, 3 inches by 1 inch, at its posterior surface. Contents of the abscess consisted of necrotic tissue and its wall was shaggy. The cæcum and ascending colon showed no ulceration. The descending colon was slightly atrophied, adherent to the posterior abdominal wall and its upper part showed a few ostia slightly larger than pin-points in size. Depth of these ostia appeared to be up to the thickness of the mucosa only and there was no undermining of the edges of these ostia.

Histopathology : (1) *Large intestine.*—The pin-point ostia referred to above were tiny amœbic ulcers. They were most marked on the splenic flexure in which relatively large number of parasites were observed. The mucous membrane was less affected than the deeper layers and much of the muscle had been replaced by fibrous tissue infiltrated with chronic inflammatory cells.

(2) *Liver.*—The lesion was typical of an amœbic abscess except that no amœbæ could be identified in the sections examined. The abscess cavity was lined by a shaggy layer of pink-stained, acellular and structureless material representing necrotic liver tissue. Outside this was a well-marked zone of lymphocytes and a few plasma cells and monocytes. Polymorphs were conspicuous by their absence. Just beyond this was a broad indefinite layer of compressed liver tissue in which the lobular arrangement had been lost and the columns of hepatic cells had become broken up into groups of 1, 2 or 3 cells undergoing degeneration, the cells being swollen, rounded and with karyolysis and vacuolation of the cytoplasm. This area was permeated by a variable number of chronic inflammatory cells. The chronicity of the lesion was shown by areas of collapse and fibrosis in which the small bile ducts had survived though the polyhedral cells had been destroyed rather like the appearance in sub-acute yellow atrophy. The surrounding liver had normal architecture but the portal tracts showed round-celled infiltration, the capillary sinuses were congested: the liver cells showed cloudy swelling and the Kupffer cells were loaded with minute granules of dark brown pigment (old malarial pigment).

(3) *Lung.*—An abscess was present. The wall of the abscess consisted of pink-staining necrotic debris centrally. Outside this was a zone in which the architecture of the affected lung could just be distinguished. The blood vessels and bronchioles had survived longer than the more delicate alveoli, and throughout this zone were very large number of amœbæ containing granules, vacuoles, RBC's and altered blood pigment. Beyond this was a broad zone of