 SOME LONG-TERM RESULTS OF TREATING URINARY SCHISTOSOMIASIS IN KUWAIT

D. M. FORSYTH*
Formerly Physician
AND
MERVYN HUGHES†
Lately Radiologist,
Kuwait Oil Company Limited.

Before 1948 fresh water was imported into Kuwait by boat from Iraq. Possibly some snails shedding cercariae of *Schistosoma haematobium* were occasionally brought in with the water, and rarely a person may have been infected by this means. Normally however there was no transmission of *S. haematobium* infection in Kuwait. Nevertheless, urinary schistosomiasis was not uncommon in Kuwait Oil Company Ltd. (K.O.C.) employees, many of whom came from other countries. Altogether in 1960 there were 89 male patients directly employed by K.O.C. who had been treated by one of us for urinary schistosomiasis and who were considered to have been cured. We do not know when or where many of them acquired their infections, for the histories given were often unreliable.

No systematic urinary egg-counts were made, but nearly all patients were carefully investigated clinically to define exactly the pathological changes caused by *S. haematobium* infection. Detailed findings in 26 of them were earlier reported in this journal (FORSYTH, 1961).

In 1970, 32 of the 89 patients remained in uninterrupted service with K.O.C. This total includes 10 of the patients described in the previous report (*op cit*). 4 other patients were treated by one of us for urinary schistosomiasis before 1960, but left K.O.C. and later rejoined the Company. These 36 patients were examined at irregular intervals between 1961 and 1970, and all of them were carefully reviewed during 1970 and 1971 to assess the residual effects of infection with *S. haematobium*. The period of observation after urinary schistosomiasis was diagnosed varied from 11 to 18 years, with a mean of 13.8 years.

Results

Urinary findings

One patient was treated for urinary schistosomiasis in 1959 and believed to be cured. He left K.O.C. temporarily. During a routine follow-up examination in 1962 he was found to be voiding viable *S. haematobium* eggs in the urine. A second course of treatment was given, and afterwards no viable eggs were found in repeated urinary examinations.

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*present address, Leighton Hospital, Cheshire.
†present address, St. Helen's Hospital, Hastings.

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A few patients had occasional frank haematuria during the early period of follow-up after treatment, microscopic haematuria was more common. These findings were associated occasionally with the passage of dead *S. haematobium* eggs.

Several patients continued to void neutrophil and/or eosinophil polymorphs in the urine for many years after parasitological cure. However none of them had white cells in the urine when specimens were examined in 1970 and 1971.

Repeated and systematic urinary examinations in all of the other patients showed nothing abnormal.

**Clinical findings**

In 1960, 32 of our patients were young adult males who were apparently well and had no symptoms attributable to past *S. haematobium* infection.

During 1961 one of them had recurrent attacks of right renal colic. A stone was removed surgically from the right kidney, and other stones were passed spontaneously in 1962 and in 1963, after which he was symptom-free.

Another of the 32 patients had pain and frequency of micturition associated with bacteriuria during 1967. The symptoms responded promptly to treatment with specific anti-bacterial therapy. There was no recurrence of urinary symptoms or of bacteriuria.

None of the other patients had symptoms which could be attributed to past infection with *S. haematobium* during the follow-up period or when they were finally reviewed in 1970 and 1971.

The intravenous pyelograms and cystoscopic appearances in 13 patients*** were normal in 1960 and in 1970-71.

In 4 other patients the intravenous pyelograms were normal in 1960 and in 1970, but at cystoscopy in 1970 the ureteric orifices were rigid and punched out.

In 1958 the intravenous pyelogram of 1 patient showed bladder calcification with irregularity and dilatation of the right ureter and right hydronephrosis. Minute sandy patches were seen through the cystoscope. In 1971 the intravenous pyelogram was normal.

One patient* had minimal bladder calcification in 1959, but no other radiological abnormality. The cystoscopic appearances were normal. No bladder calcification was seen radiologically in 1970. In another patient* bladder calcification was less pronounced in 1970 than it was in 1959.

In 5 patients** the intravenous pyelogram in 1960 showed calcification of the bladder with ureteric deformity. The appearances were very similar in 1970-71.

In 1 other patient* the 1959 pyelogram showed bilateral ureteric deformities and bladder calcification. In 1971 the bladder seemed unchanged but the walls of the ureters were calcified.

In 2 patients the intravenous pyelograms in 1959 and 1960 were normal, but ureteric deformities were seen in 1970.

In 2 other patients the only abnormalities seen in the 1960 pyelograms were bilateral ureteric deformities. The radiological appearances in 1970-71 were very similar.

In 3 patients* marked ureteric deformities were seen radiologically in 1960. In 1970 the deformities were less severe.

One patient's 1959 pyelogram showed bilateral ureteric deformity and unilateral hydronephrosis. In 1970 the ureters and kidney seemed to be normal radiologically, but bladder calcification was seen.

*indicates 1 patient described in the 1961 paper.
Another patient* had unilaeral hydroureter and unilateral hydronephrosis in the 1957 pyelogram. Cystoscopic examination then showed many sandy patches and extensive ulceration of the bladder. In 1970 the pyelograms showed unilaeral hydroureter but no hydronephrosis. The cystoscopic appearances of the bladder were normal.

In our last patient the 1960 pyelogram showed bladder calcification, minor deformities of both ureters, bilateral hydronephroses and a stone in the left kidney. The bladder wall was pale but intact when seen through the cystoscope. In 1970 the radiological appearance of the bladder was unchanged, the ureteric deformities were marked and there was a possible reduction in the size of the hydronephroses. The stone remained, unchanged.

Discussion

These results are satisfactory, for our patients suffered few ill-effects from infection with *S. haematobium*. In some of them urological lesions regressed during the follow-up period, and in none did the lesions appear to increase in severity. However these case histories are very different from those of most people who are infected with *S. haematobium*. K.O.C. employees are well-paid, live in modern houses with good sanitation and have excellent medical and social services. For them there is little chance of reinfection, because *S. haematobium* is not transmitted in Kuwait. Indeed the case histories are not even representative of the K.O.C. patients whom we treated for urinary schistosomiasis.

In the earlier series (op cit) the urological findings in 28 patients were reported and 12 of them were thought to have suffered important urological damage. Only 1 out of the 12 is included in the present series. In 5 other patients in the earlier report it was thought that urological damage was permanent and progressive; none of these 5 patients is included in the present series. The patients whose case histories are given above are clearly those whose urinary tracts were slightly damaged as a result of *S. haematobium* infection. We do not know what happened to our patients whose lesions were more severe, for we were unable to trace them after they left K.O.C. None of the 89 patients whom we treated for urinary schistosomiasis later died whilst in K.O.C. service.

Our findings show only that under favourable circumstances the prognosis is good for some patients with urinary schistosomiasis who are given prompt and efficient treatment.

Summary

36 patients were followed up for periods of 11–18 years after treatment for urinary schistosomiasis. Clinically they remained well; generally their urological lesions regressed and in no patient did the lesions increase significantly in severity.

Under favourable conditions the prognosis may be very good for some patients with *S. haematobium* infection.

REFERENCE