7

A 28-Year-Old Male Fisherman from Malawi With Shortness of Breath

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Clinical Presentation

History

A 28-year-old Malawian man presents to a local hospital with progressive shortness of breath over the past 5 days. He reports orthopnoea and paroxysmal nocturnal dyspnoea. He has also developed bilateral flank pain in the past days, which is continuous and dull, and there is constant nausea. There is no cough and no fever.

He was diagnosed with arterial hypertension 2 years earlier and prescribed antihypertensive drugs, which he never took. No investigations were done at that time.

His medical history and family history are otherwise unremarkable. A recent HIV test was negative. The patient is a fisherman from a town on the southern shore of Lake Malawi.

Clinical Findings

The 28-year-old man is not looking chronically ill, but is in respiratory distress. His conjunctivae are notably pale. His blood pressure is 200/130 mmHg, pulse 66 bpm, temperature 36.8°C and respiratory rate 32 breath cycles per minute.

His apex beat is slightly displaced, but his heart sounds are clear and regular. The jugular venous pressure is not raised. There are bilateral fine crackles over the lung bases. The abdomen is flat and non-tender. There is bilateral renal angle tenderness, and the kidneys are ballottable. There is no peripheral oedema.

Laboratory Results

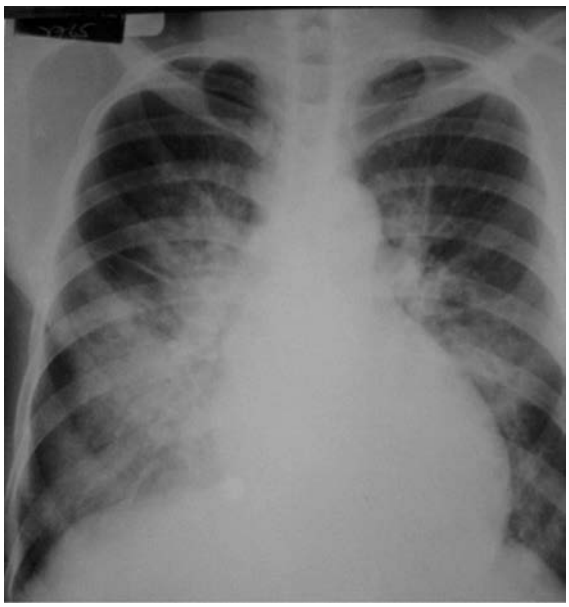
His laboratory results on admission are shown in Table 7.1.

Imaging

His chest radiograph on admission is shown in Figure 7.1.

TABLE 7.1 Laboratory Results on Admission

Parameter	Patient	Reference Range
WBC ($\times 10^9$/L)	3.8	4–10
Haemoglobin (mg/dL)	6.0	13–15
MCV (fL)	92	80–99
Platelets ($\times 10^9$/L)	187	150–400
Creatinine (μmol/L)	1200	<120
BUN (mmol/L)	89.3	<17.9
K^+ (mmol/l)	7.2	3.5–5.2



• **Fig. 7.1** Chest radiograph on admission.

Questions

1. What is your clinical impression?
2. What further investigations would you do to establish the diagnosis?

Discussion

A young Malawian fisherman presents with signs and symptoms of left ventricular heart failure, hypertension and anaemia. There is renal angle tenderness, and his kidneys appear enlarged. His creatinine is very high and he is hyperkalaemic.

Answer to Question 1

What Is Your Clinical Impression?

This young man presents with combined left-sided cardiac failure and renal failure. He was diagnosed with hypertension 2 years prior. It is unclear if his renal incompetence is a cause or the result of his raised blood pressure.

The reason for the enlargement of his kidneys could either be primary (e.g. polycystic kidneys) or secondary as a result of post-renal obstruction with hydronephrosis. In renal compromise secondary to hypertension, one would expect the kidneys to be small.

Being a fisherman, the patient has been in regular contact with *Schistosoma haematobium*–infested water in Lake Malawi. Chronic schistosomiasis with hydronephrosis is one of the top differential diagnoses to suspect.

Answer to Question 2

What Further Investigations Would You Like to Perform to Establish the Diagnosis?

The most useful investigation at this point is an ultrasound of the kidneys to differentiate between, primary renal pathological condition and a post-renal problem. If there was hydronephrosis, obstruction at the level of the bladder seems most likely because both kidneys appear enlarged.

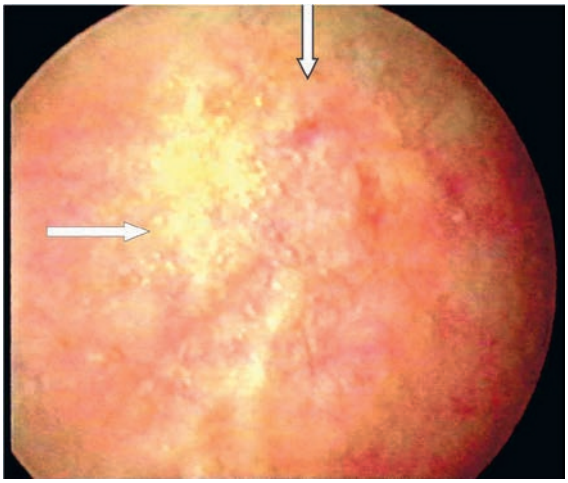
On cystoscopy, endoscopists can macroscopically establish the diagnosis, but ideally, biopsies should be performed to look for evidence of granulomatous inflammation and *S. haematobium* ova in the tissue and to rule out neoplasia. In chronic infection, urine microscopy may be negative for ova of *S. haematobium.*

The Case Continued. . .

Despite high doses of furosemide, the patient remained oliguric. Glucose and insulin were administered for his hyperkalaemia.

Ultrasound showed bilateral hydronephrosis with massive dilatation of the renal pelvis and calyces. The remaining renal parenchyma was very thin in both kidneys.

Cystoscopy revealed a hyperaemic mucosa with multiple 'sandy patches' suggestive of granulomatous lesions in the mucosa. No tumour was seen. An endoscopic diagnosis of urogenital schistosomiasis was made. Histology was not available.



• **Fig. 7.2** Cystoscopy findings of a patient with urogenital schistosomiasis. The white arrows show 'sandy patches' (*left*) and hyperaemia of the bladder mucosa (*top*). (Courtesy Iran Mendonça da Silva.)

The next day the patient deteriorated. He became drowsy and vomited repeatedly. He got progressively bradycardic, and there was a new pericardial friction rub suggestive of uraemic pericarditis.

He was taken to theatre and a bilateral percutaneous nephrostomy was done. The patient was transferred to the intensive care unit and peritoneal dialysis (PD) was started (haemodialysis was not available).

The patient improved rapidly, vomiting and drowsiness ceased and his friction rub disappeared. He was discharged home on PD.

Two months later he was readmitted with fever and abdominal pain. He was treated for suspected bacterial peritonitis but sadly died 3 weeks later still in the hospital.

SUMMARY BOX

Genitourinary Schistosomiasis

Genitourinary schistosomiasis caused by *Schistosoma haematobium* is endemic in large parts of Africa and in the Middle East. Schistosomiasis is one of the most common tropical diseases in migrants from endemic countries and should actively be screened for.

About 50% of *Schistosoma* eggs are shed through urine, the other half remain trapped in the tissue causing granulomatous inflammation. Around 10% of people infected progress to chronic late-stage disease. Risk factors for disease progression are poorly understood and include intensity and duration of infection, host genetic factors and parasite strain differences.

Chronic infection leads to granulomatous inflammation of bladder wall and ureteral mucosa resulting in obstructive uropathy, hydronephrosis, recurrent bacterial pyelonephritis and end-stage renal disease. It is also suspected to contribute to the development of squamous cell carcinoma of the bladder. The pathological changes can long go unnoticed.

Egg deposition in the female genital tract may result in dyspareunia, chronic lower abdominal pain, ectopic pregnancy and infertility and has also been associated with an increased risk of HIV infection. Men may present with scrotal swelling, orchitis and prostatitis, haematospermia and oligospermia.

In advanced stages of schistosomiasis, the proof of *Schistosoma* eggs in the urine is challenging because the adult flukes may have long died and egg production may have stopped. Biopsy is often unavailable and serology is unhelpful in an endemic setting, because it can only detect exposure without indicating the activity, duration or quantum of infection. A promising point-of-care diagnostic tool may be circulating anodic antigen (CAA), which is under development.

The investigation of choice is ultrasound. Hydronephrosis and bladder wall abnormalities can easily be demonstrated.

On cystoscopy a hyperaemic mucosa with 'sandy patches' may be seen (Fig. 7.2). Sandy patches are raised, yellowish mucosal irregularities associated with heavy egg deposition surrounded by fibrous tissue pathognomonic for schistosomiasis.

Praziquantel may reverse the early stages of infection but has little role to play in advanced hydronephrosis. It may still be given to kill remaining adult flukes. Otherwise, late-stage urogenital schistosomiasis has to be managed symptomatically, which can be challenging in settings where renal replacement therapy is not an option.

Further Reading

1. Bustinduy AL, King CH. Schistosomiasis. In: Farrar J, editor. Manson's Tropical Diseases. 23rd ed. London: Elsevier; 2013 [chapter 52].
2. Colley DG, Bustinduy AL, Secor WE, King CH. Human schistosomiasis. Lancet 2014;383:2253–64.
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4. Asundi A, Belavsky A, Liu XJ, et al. Prevalence of strongyloidiasis and schistosomiasis among migrants: a systematic review and meta-analysis. Lancet Glob Health 2019;7:e236–48.