line was started at 1 mg/h. Hydroxocobalamin 5 g was given intravenously over 30 minutes, followed by another 5 g over 12 hours. Systolic blood pressure rose rapidly with the initial infusion of hydroxocobalamin and the patient became responsive to painful stimulation but never regained consciousness. He developed pneumonia, with septic shock, and died 12 days after the ingestion.

Blood samples were simultaneously collected from femoral artery and internal jugular vein catheters for measuring arterial and venous blood gas tensions and blood cyanide,³ and plasma lactate concentrations.⁴ Toxic and lethal blood cyanide concentrations are in the range of 40 µmol/l and 100 µmol/l, respectively.⁵ The upper limit of normal for lactate is ≤2 mmol/l. Decay in plasma lactate and blood cyanide concentrations was derived with non-linear regression analysis (PRISM, GraphPad Software).

On admission, blood cyanide and plasma lactate concentrations, before hydroxocobalamin infusion, were 256 µmol/l and 53 mmol/l, respectively (figure). These values decreased to 40.4 µmol/l and 10 mmol/l, respectively, six hours after admission. The time course of blood cyanide concentration is described by a monoexponential decay ($r^2=0.97$) with a blood half life of 1.14 hours (95% confidence interval 0.84 to 1.80); the time course of plasma lactate concentration is described by a monoexponential decay ($r^2=0.99$) with a blood half life of 3.94 hours (2.98 to 5.78).

Until five hours after admission, despite normalisation of systolic blood pressure, lactate concentrations

remained raised and arterial and venous oxyhaemoglobin saturation did not differ significantly.

Comment

Systolic blood pressure was not indicative of blood cyanide levels and oxyhaemoglobin saturation gradient did not parallel decreases in blood cyanide concentrations. Concentration decay curves suggest that plasma lactate concentration is closely related to blood cyanide concentration before and after hydroxocobalamin treatment. If this is confirmed in other cases of pure cyanide intoxication serial plasma lactate concentrations could be used as a marker of the evolution of cyanide toxicity and perhaps of the adequacy of treatment.

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Assessment of urine analysis for the diagnosis of tuberculosis

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The number of reported cases of tuberculosis has been increasing steadily over the past few years. Due to the complex and time consuming procedures needed for the detection and isolation of *Mycobacterium tuberculosis*, the workload of the microbiology laboratory is substantial. Many recently developed technologies—including blood cultures performed with lysis-centrifugation, genomic detection by polymerase chain reaction, or detection of mycobacterial antigens by immunoassays—will modify the diagnostic approach of tuberculosis.¹ As most of these new diagnostic procedures are not yet available routinely, sputum and urine remain the most common clinical specimens submitted to conventional microbiological study. To improve the quality and efficiency of medical care, we tried to assess the usefulness of urine analysis for the diagnosis of tuberculosis.

Patients, methods, and results

In this retrospective study, conducted between July 1983 and July 1993 in a 500 bed hospital, all patients with positive urine cultures for *M tuberculosis* were identified from microbiology records. During this 10 year period, 7200 midstream morning urine specimens obtained from 2814 patients (mean (SD) age 48.9 (22.9) years; 63% male) were submitted to microbiological examination for a presumptive diagnosis of tuberculosis. All urine specimens were inoculated onto Lowenstein-Jensen medium. Only 65 (0.9%) urine samples obtained from 33 patients (1.2%) yielded positive cultures for *M tuberculosis*. In this group, acid

fast bacilli in direct Ziehl-Nielsen stained smears were detected in eight of the 19 samples processed. Urine analysis performed on non-centrifuged samples of 29 of these 33 patients gave normal results for 21 of them, including three of the seven patients with genitourinary tuberculosis. The urinary sediment of the remaining patients showed pyuria ($\geq 10\,000$ cells/ml) in all eight and haematuria (≥ 5000 cells/ml) in five.

Of the 33 patients with positive urine cultures for *M tuberculosis*, 22 presented with pulmonary tuberculosis as the main site. Direct smears of sputum were positive for acid fast bacilli in 15 of them (68%). Seven patients had tuberculosis limited to the genitourinary tract as diagnosed by clinical signs and symptoms. No additional patients with genitourinary tuberculosis were identified from pathology records during the study period. The remaining patients had gastrointestinal, pleural-peritoneal, miliary, and meningeal involvement (table). Except for the patients with tuberculosis of the genitourinary tract, for whom urine culture was diagnostic, identification of *M tuberculosis* in urine did not allow a more prompt diagnosis of tuberculosis already established with other specimens.

Comment

Of patients with pulmonary tuberculosis, 5-8% have positive urine cultures for *M tuberculosis* even though there are no signs, symptoms, or laboratory data that suggest genitourinary tract involvement.²⁻⁴ No predictive factors of bacilluria in pulmonary tuberculosis have been identified so far. In our study, among the 19 patients with bacilluria and pulmonary tuberculosis in whom urinary sediment analysis was available, only three (16%) had abnormal results on urinalysis, which is consistent with published reports.²⁻⁴

These data suggest that submission of urine specimen to the microbiology laboratory for identification of *M tuberculosis* is rarely useful in the diagnostics of tuberculosis. Except in patients with genitourinary

Patient No	Sex	Age	No of urine samples	Positive direct smear	No of positive cultures	Pyuria*	Haematuria†	<i>M tuberculosis</i> isolation	Positive direct smear in sputum	Tuberculosis
1	M	77	1	Not done	1			Sputum; gastric aspirate; pleural biopsy		Pulmonary-pleural
2	M	51	3	1/3	3	+	+			Genitourinary
3	F	46	3	Not done	1			Sputum; gastric aspirate	+	Pulmonary
4	F	66	2	Not done	1			Sputum; gastric aspirate	+	Pulmonary
5	M	78	2	Not done	2			Gastric aspirate		Pulmonary
6	M	43	3	Not done	3	Not done	Not done	Bronchoalveolar lavage		Pulmonary
7	F	26	3	Not done	2	+		Sputum; gastric aspirate	+	Pulmonary
8	F	80	3	Not done	1			Gastric aspirate; bronchial aspirate		Pulmonary
9	M	40	3	Not done	3			Sputum	+	Pulmonary
10	M	58	3	Not done	1					Genitourinary
11	M	34	3	Not done	1			Gastric aspirate; bronchial aspirate		Pulmonary
12	M	40	3	Not done	3			Sputum		Pulmonary
13	M	54	3	0/3	1			Sputum	+	Pulmonary
14	M	47	4	Not done	3			Sputum; gastric aspirate	+	Pulmonary
15	M	25	3	Not done	2			Draining sinus		Gastrointestinal
16	M	29	7	Not done	6	+	+	Sputum; gastric aspirate		Pulmonary
17	M	18	4	Not done	1	Not done	Not done	Sputum; gastric aspirate	+	Pulmonary
18	M	28	3	Not done	2			Sputum; gastric aspirate	+	Pulmonary
19	M	31	2	Not done	2			Sputum; gastric aspirate	+	Pulmonary
20	M	34	3	Not done	2			Sputum; gastric aspirate	+	Pulmonary
21	M	59	3	Not done	1			Sputum; gastric aspirate	+	Pulmonary
22	M	54	2	Not done	1	+		Ascites; pleural biopsy		Pleural-peritoneal
23	F	87	1	1/1	1	+	+	Sputum	+	Miliary
24	M	58	3	Not done	1	+		Sputum	+	Pulmonary
25	F	74	2	Not done	1					Genitourinary
26	M	75	3	Not done	2	+	+			Genitourinary
27	F	47	2	2/2	2			Bronchial aspirate	+	Pulmonary
28	M	52	3	0/3	1			Spinal fluid		Meningeal
29	M	53	3	Not done	1					Genitourinary
30	M	54	3	Not done	3			Sputum; gastric aspirate; myocardial biopsy	+	Pulmonary-mycocardial
31	F	51	3	Not done	3	+	+			Genitourinary
32	M	50	3	2/3	3	Not done	Not done	Sputum	+	Pulmonary
33	M	41	4	2/4	4	Not done	Not done			Genitourinary

*Leukocyte count $\geq 10\,000$ cells/ml. †Red blood cell count ≥ 5000 cells/ml.

tuberculosis, who usually present with clinical signs and symptoms justifying the sampling,⁹ urine analysis does not provide an earlier diagnosis. Moreover, bacilluria does not indicate the need to modify the recommended therapeutic regimens for the patients with pulmonary tuberculosis, nor does it purport a worse prognosis.

The cost of this diagnostic procedure should also be considered. In our study, the 7200 urinalyses incurred a cost of about 72 000 ecu (approximately £58 000) and were beneficial only in the seven patients with genitourinary tuberculosis.

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WHEN I USE A WORD

Curious, yellow

A young man, drunk, had been worsted in a dust up with his landlord. He presented a week later with neurological signs and symptoms that made us do a lumbar puncture. Sure enough, the spinal fluid was uniformly blood stained. Later I discussed the case with the medical students. Talking about the possibility that the fracas and the clinical presentation had been unrelated, we discussed the changes that you expect in the spinal fluid after subarachnoid haemorrhage. What does xanthochromia mean? they asked. I saw the chance for a brief diversion. In Greek, I explained, xanthos means yellow, any kind of yellow, or even brown; one of Achilles's horses was called Xanthos, or Bayard—the other was called Balios, Dapple—and Xanthippe, the name of the spirited wife of Socrates, means "bay filly." Chroma means colour, originally the colour of the skin, but then extended to mean colour generally.

Now, I asked them, can you think of any other words with xanthos in them? They recalled the xanthines, derivatives of xanthic acid, so called because it leaves a lemon yellow residue when reacted with nitric acid, and for a minute or two we discussed caffeine, theophylline, and the treatment of asthma, and then progressed to hypoxanthine, purine metabolism, and the treatment of gout. They had not heard of xanthopsia but were interested in digitalis toxicity. We stored up those subjects for future tutorials.

Any others? I asked, hoping for xanthoma and xanthelasma. They thought hard. A light came into the eyes of one of them. "Yes," she said triumphantly, "chrysanthemum."

I decided that it was time to return to subarachnoid haemorrhage.—JEFF ARONSON is a clinical pharmacologist in Oxford