evaluations of cognitive behaviour therapy are desirable, including comparisons with treatments other than basic medical care. Nevertheless, we believe that our results have potentially important implications for the management of patients presenting to medical clinics with chronic disabling fatigue.

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Relation between plasma lactate and blood cyanide concentrations in acute cyanide poisoning

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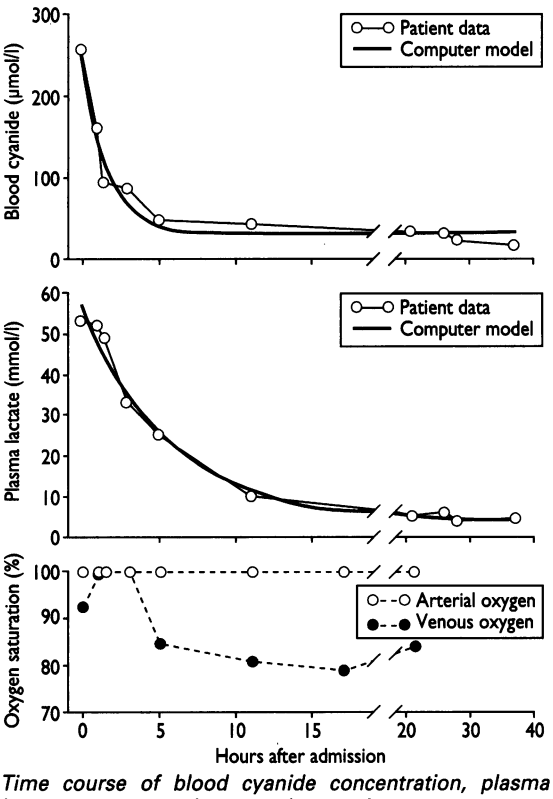
Cyanide poisoning produces rapid blockade of cellular respiration due to binding to cytochrome$_{aa3}$, resulting in accumulation of lactate. Lactic acidosis is a recognised hallmark of acute cyanide poisoning in humans.[1,2] The time course of lactic acidosis, however, has not been well described in relation to evolving blood cyanide concentrations. We studied the relation of blood cyanide to plasma lactate concentrations in a patient with pure acute cyanide poisoning.

Case report

A 63 year old man called for help immediately after suicidal ingestion of a single potassium cyanide capsule. He was conscious on arrival of ambulance staff, but apnoea rapidly supervened, followed by cardiac arrest. Cardiopulmonary resuscitation, endotracheal intubation with 100% pure oxygen, and advanced life support were started. He regained a pulse, with response to painful stimuli.

On arrival at hospital the patient was completely unresponsive and severely hypotensive, with a systolic blood pressure of 35 mm Hg measured by indwelling catheter; his heart rate was 72 beats/minute. Arterial blood gas tensions showed severe metabolic acidosis: pH 7·15, arterial carbon dioxide pressure 24 mm Hg, and arterial oxygen pressure 447 mm Hg; bicarbonate ion concentration 8·2 mmol/l. Gastric lavage and a single dose of activated charcoal were given immediately after the first blood samples were drawn. Intravenous fluids were given and intravenous adrena-



Time course of blood cyanide concentration, plasma lactate concentration, and arteriovenous oxygen saturation in a case of pure cyanide poisoning

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 (≥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