

## CASE REPORT

# Solvent abuse, toluene acidosis and diabetic ketoacidosis

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### SUMMARY

Solvent abuse in adolescents and young adults has been reported to cause a metabolic acidosis with a normal or increased anion gap (Streicher *et al.*, 1981; Voights & Kaufman, 1983, Anonymous, 1988). We report a particularly severe clinical problem produced by the combination of toluene intoxication and diabetic ketoacidosis.

### CASE REPORT

A 20-year-old girl who had insulin dependent diabetes mellitus for 6 years and a history of habitual solvent abuse was admitted to hospital in an unconscious state. There was a marked smell of acetone on her breath and glue stains were present on her face, hands and clothing. She was afebrile, tachypnoeic and peripherally vasodilated, pulse 120 beats/min and blood pressure 120/80 mmHg. Dipstick urinalysis on several occasions revealed glucose (>55.5 mmol/l), ketones (>10 mmol/l), protein (>5 g/l) and blood. Plasma glucose was 21.4 mmol/l, standard bicarbonate 12 mmol/l and blood pH 7.12. The anion gap was increased to 20 mmol/l. Serum toluene was 6.8 mg/l and serum acetone 505 mg/l (normally undetectable). No evidence of local or generalized sepsis was identified. She was treated using a standard ketoacidosis regimen of intravenous fluid and potassium replacement with frequent intramuscular administration of short acting insulin.

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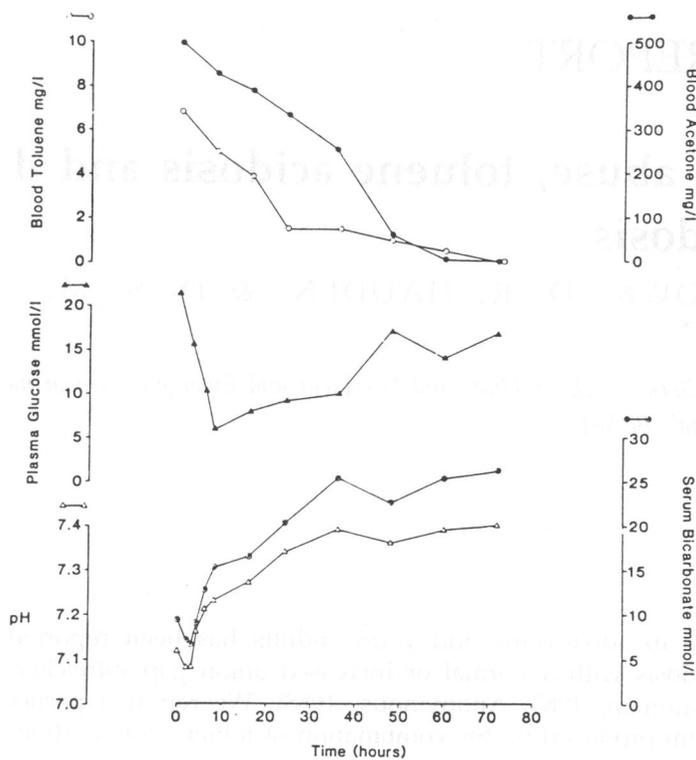


Fig. 1 Blood toluene and acetone levels during management of mixed solvent induced and diabetic acidosis.

Clinical recovery was prompt. The return to biochemical normality is shown in the Fig. 1. Dipstick urinalysis returned to normal. Following stabilization of diabetic control she was transferred to a psychiatric unit for treatment of her addiction, but unfortunately this remains a major problem.

## DISCUSSION

Toluene intoxication can induce a metabolic acidosis either indirectly by causing renal tubular damage with secondary distal tubular acidosis, or directly by increasing the circulating level of organic acids following conversion of toluene to benzoic acid (Streicher *et al.*, 1981, Voights & Kaufman 1983). A transient renal tubular acidosis was probably present in this patient as haematuria and proteinuria was detected on admission. She had very high circulating levels of both toluene and acetone, which fell as the pH and bicarbonate returned to normal, in keeping with an acidaemia due to the combination of diabetic ketoacidosis and toluene-induced metabolic acidosis. It is probable that toluene intoxication in this instance

precipitated the diabetic ketoacidosis. Blood toluene levels in excess of 10 mg/l are usually fatal (Baselt, 1982).

This case illustrates the additional problems caused by solvent abuse in an insulin-dependent diabetic and the complex acid-base disturbances that may result.

## REFERENCES

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