Hypoglycaemia and septicaemia

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Summary

A patient with severe hypoglycaemia complicating staphylococcal septicaemia is reported. No recognized cause of hypoglycaemia was detected during life or at autopsy. This case adds to the existing evidence that through several possible mechanisms septicaemia can give life-threatening hypoglycaemia.

KEY WORDS: *Staphylococcus pyogenes*, infective psoriasis, endocarditis, pericarditis, osteomyelitis.

Introduction

Spontaneous hypoglycaemia complicates several systemic disorders including severe congestive heart failure and diffuse hepatic disease (Fajans and Lloyd, 1976). A case of staphylococcal septicaemia complicating pustular psoriasis and associated with unsuspected hypoglycaemia is described.

Case report

A 69-year-old female was admitted because of generalized pustular psoriasis. There was a 24-year history of palm and sole psoriasis with one episode of generalized pustular psoriasis 4 years previously at which time seropositive arthropathy affecting the hands and feet was diagnosed. Systemic corticosteroids and methotrexate had never been used. Previous drug therapy involved non-steroidal anti-inflammatory drugs and intermittent dilute topical corticosteroids and these were continued. Response was poor. Hospital course was uneventful until 8 weeks after admission when she complained of left-sided chest pain and within 12 hr became hypotensive, oliguric and comatose. Physical examination of the chest revealed left-sided consolidation, confirmed on chest X-ray. White cell count was $17.4 \times 10^9/l$ with 76.0% neutrophils. Blood cultures grew penicillin-sensitive *Staphylococcus pyogenes*. Initial blood glu-

cose (Trinder method—Autoanalyser I) was unrecordable (repeated). Following an intravenous bolus of 50 ml of 50% dextrose, and continuous infusion of 5% dextrose/water at 120 ml/hr, blood glucose had risen to 2.6 $\mu\text{mol/l}$ (normal fasting range 3.5–5.5 $\mu\text{mol/l}$) 2 hr later. Five hours after the first specimen, blood glucose was 7.2 $\mu\text{mol/l}$. Plasma cortisol drawn with the first glucose was 3560 $\mu\text{mol/l}$ (normal (9 a.m.) 170–720 $\mu\text{mol/l}$). Serum immuno-reactive insulin (Morgan and Lazerow, double antibody radioimmunoassay supra-Regional Assay Service) drawn with the second blood glucose was less than 1 $\text{m}\mu\text{l}$. Despite intensive resuscitative measures including intravenous cloxacillin, death occurred 48 hr after the onset of hypotension. Post-mortem examination revealed left-sided bronchopneumonia with central suppuration, mitral and aortic endocarditis, pericarditis with purulent effusion and osteomyelitis of five ribs. Pancreas and adrenals were normal. Random pancreatic sections showed no evidence of islet-cell tumour. The liver had some centrilobular fatty change.

Discussion

Septicaemia is a recognized complication of pustular psoriasis (McFadyen and Lyall, 1971). There was no personal or family history of diabetes mellitus or hypoglycaemic episodes. The patient was not taking any drugs associated with hypoglycaemic and post-mortem examination did not reveal known occult causes of hypoglycaemia (August and Hiatt, 1958). Serial pancreatic sections would have been valuable but insulinoma is unlikely as serum insulin levels in excess of 10 $\text{m}\mu\text{l}$ would be expected (Harrison, 1978). The time-course suggests the hypoglycaemia to be a consequence of overwhelming sepsis.

Hypoglycaemia has been reported with pneumococcal septicaemia in splenectomized patients (Francioli, Schaller and Glanser, 1981) and in 50% of cirrhotic patients with septicaemia (Nouel *et al.*, 1981). Hypoglycaemia associated with overwhelming sepsis (mortality 67%) in a general medical popula-

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tion (Miller *et al.*, 1980) has been observed and in four of nine cases, there was no explanation other than sepsis. It also occurs with infections in neonates (Young, 1970). In animals, injection of endotoxin is known to inhibit gluconeogenesis (Francis and Watson, 1977) and deplete hepatic glycogen (Sandford, Barnett and Gott, 1980). Acidosis impairs gluconeogenesis (Mallis *et al.*, 1972) and both hypotension and reduced tissue perfusion increase peripheral glucose utilization (Lehninger, 1975). The pathophysiology of hypoglycaemia in severe septicaemia is likely to be complex as acidosis and impaired tissue perfusion are common and hypoglycaemia is probably the result of impaired glucose production by the liver combined with increased peripheral utilization. Unfortunately, liver sections could not be stained for glycogen in our case. Blood glucose should be monitored in all cases of severe septicaemia.

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