Case report

A 50-year-old obese woman with a five-year duration of type 2 diabetes was referred to our institution for further management of apparently ‘severe diabetic ketoacidosis’ complicated by a left leg cellulitis. Compliance had been poor with oral hypoglycaemic agents preceding this admission.

Clinical examination revealed an erythematosus swollen left leg with an infected ulcer. She was extremely breathless with an unrecordable blood pressure. Blood glucose was 30 mmol/L and urine ketones were strongly positive. Urgent arterial blood gases on high flow oxygen revealed a pH of 7.42, PO$_2$ of 10.6 kPa, PCO$_2$ of 1.9 kPa and bicarbonate of 15 mmol/L. The anion gap was high at 29. The inappropriately normal pH and the degree of CO$_2$ lowering for the given fall in HCO$_3$ made us revise the diagnosis of severe diabetic ketoacidosis.

A chest X-ray revealed pulmonary oligaemia. A 12-lead electrocardiogram showed marked sinus tachycardia with a right bundle branch block. A transthoracic echocardiogram revealed severe right ventricular dilatation. An urgent computed tomography (CT) pulmonary angiogram (Figure 1) showed pulmonary embolism affecting both the right and left main pulmonary arteries. Immediate thrombolysis was administered using alteplase with a dramatic clinical improvement. Ultrasound of both legs the next day revealed a left deep vein thrombosis.

The patient was discharged a few days later on warfarin in a clinically stable condition.

Discussion

There is always an inherent risk in branding a patient with a diagnosis that has a protocol-based management plan, as it may lead to ‘thought inertia’, unless the physician has a high index of clinical suspicion. Arterial blood gases are among the first tests to be performed in suspected diabetic ketoacidosis. Systematic analysis of arterial blood gases in such a scenario can lead the physician to diagnose serious co-morbid illness and this can be life saving.1

Even though there have been previous case reports of thromboembolism complicating diabetic ketoacidosis, such presentations are still uncommon.2–4 However, it is extremely vital not to miss major pulmonary thromboembolism complicating diabetic ketoacidosis, as this combination can be potentially fatal within a few hours if unrecognised.

There were two important clues in our patient that suggested the presence of significant lung pathology. The presence of a pH of 7.42 in a patient with suspected severe diabetic ketoacidosis is extremely unusual and signifies a co-existing significant alkalosis in addition to the metabolic acidosis. The second clue was in the degree of respiratory compensation for a given lowering of bicarbonate. Winter’s formula, named after Dr RW Winters, is a formula used to evaluate the respiratory compensation in a patient with metabolic acidosis. The formula states that the expected PCO$_2$ = (1.5 x HCO$_3$) + 8 ± 2, where HCO$_3$ is in mEq/L and PCO$_2$ is in mmHg (1 kPa = 7.5 mmHg). If the patient’s expected value corresponds to the
actual measured value, then the respiratory compensation is thought to be adequate. If the measured PCO₂ is higher than the expected value, then there is a concomitant respiratory acidosis. On the contrary, if the measured PCO₂ is lower than the expected value, then there is a concomitant respiratory alkalosis. Applying the formula to our case, the measured PCO₂ of 1.9 kPa was much lower than the calculated value (4 kPa), indicating concomitant respiratory alkalosis. The differential diagnosis for respiratory alkalosis commonly includes pain, psychosis and anxiety, pneumonia, pulmonary oedema and pulmonary embolism. The electrocardiogram and the chest X-ray raised enough suspicion for us to get an urgent echocardiogram and CT pulmonary angiogram. Hypotension in the presence of acute pulmonary embolism is widely accepted as criteria for thrombolysis, because successful therapy can be life saving.5

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Declaration of interests
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References