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Review

Starvation ketoacidosis in pregnancy

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ABSTRACT

Starvation ketosis outside pregnancy is rare and infrequently causes a severe acidosis. Placental production of hormones, including glucagon and human placental lactogen, leads to the insulin resistance that is seen in pregnancy, which in turn increases susceptibility to ketosis particularly in the third trimester. Starvation ketoacidosis in pregnancy has been reported and is usually precipitated by a period of severe vomiting. Ketoacidosis is likely to have important implications for fetal survival as ketoacidosis in women with type 1 diabetes mellitus is associated with intrauterine death.

This article features four cases of women with vomiting in the third trimester of pregnancy associated with a severe metabolic acidosis. The mechanism underlying ketogenesis, the evidence for accelerated ketogenesis in pregnancy and other similar published cases are reviewed. A proposed strategy for management of these women is presented.

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1. Introduction

The development of a metabolic acidosis in pregnancy poses a threat to both mother and fetus and is a medical emergency. Severe acidosis occurs rarely in pregnancy and may result from diabetic ketoacidosis in women with type 1 diabetes mellitus, lactate accumulation secondary to sepsis or rare conditions with metabolic consequences such as acute fatty liver of pregnancy. This requires immediate resuscitation and prompt management to remedy the acidosis and consideration of delivery after stabilisation of the mother. Emergency caesarean section in a woman who has not been adequately resuscitated may result in further deterioration in her condition.

Ketoacidosis in the non-pregnant population is most commonly seen in type 1 diabetes mellitus in association with inadequate insulin treatment, intercurrent illness or as the first presentation of

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the condition. Ketoacidosis is occasionally seen in people with a history of alcohol excess. Starvation-induced acidosis in the non-pregnant population is rare and usually causes a mild acidosis reaching maximum severity after approximately 14 days [1]. It has long been noted that pregnant women are more prone to ketosis and a number of cases of starvation ketoacidosis have been described previously [2–8]. Here we present four cases of starvation ketoacidosis in pregnant women who presented with vomiting in the third trimester of pregnancy.

2. Case 1

A 22-year-old woman presented at 32 weeks of gestation with a 24 h history of severe vomiting [9]. The day before presentation she had been treated with prednisolone and erythromycin for worsening breathlessness attributed to an infective exacerbation of her known mild asthma. She had previously had a termination of pregnancy and a normal pregnancy with delivery at term. She did not take any regular medication, use illicit drugs or drink alcohol. She continued to smoke 20 cigarettes per day.

At presentation she was unwell, tachypnoeic, tachycardic and appeared dehydrated, but the remainder of the examination was normal. Her blood pressure was 145/70 mmHg. Urinalysis showed 1+ protein and 4+ ketones. The capillary blood glucose was 4.2 mmol/L, with capillary ketones of 4.0 mmol/L. Arterial blood gas measurement revealed a partially compensated metabolic acidosis (pH 7.27, $PaCO_2$ 1.14 kPa, HCO_3^- 8.6 mmol/L, base excess −22 mmol/L), with an increased anion gap (20 mequiv./L) and lactate of 0.6 mmol/L. Blood tests were normal except for a neutrophilia (23.8×10^9/L) with mildly raised CRP (15 mg/L) and an elevated urate (691 μmol/L).

There was no improvement in the metabolic abnormalities after fluid resuscitation using compound sodium lactate, so, after intramuscular betamethasone administration, an emergency caesarean section was performed under general anaesthetic and a 1.9 kg male baby was delivered. Umbilical cord arterial pH was 7.23 and 5-min Apgar score was 10. The mother was taken to Intensive Care, the metabolic parameters normalised within 6 h and she was extubated 12 h after delivery.

3. Case 2

A 40-year-old woman presented at 35 weeks of gestation with a 3 day history of a sore throat and 24 h history of vomiting, epigastric pain and no oral intake. She had had two previous miscarriages and two other pregnancies. The first pregnancy had been complicated by placenta praevia and antepartum haemorrhage that was managed conservatively: she remained well and underwent an emergency caesarean section at term for fetal distress. Her second pregnancy had been uncomplicated, with a ventouse delivery at term.

In her present pregnancy, gestational diabetes mellitus was diagnosed after an abnormal oral glucose tolerance test at 28 weeks' gestation (fasting glucose 4.5 mmol/L, 8.1 mmol/L after 120 min). Good glycaemic control was achieved with dietary modification.

On admission blood pressure, pulse and temperature were normal. Examination demonstrated bilateral submandibular lymphadenopathy and erythematous tonsils. She had mild epigastric and right upper quadrant tenderness on abdominal palpation. Urinalysis showed 4+ ketones only. Blood tests showed an elevated urate (450 μmol/L), mild derangement in liver transaminases (bilirubin 8 mmol/L, ALT 59 IU/L, AST 105 IU/L) and raised inflammatory markers (WCC 16.7×10^9/L, CRP 154 mg/L). Intravenous antibiotics were given to cover the possibility of acute cholecystitis, but abdominal ultrasound was normal.

Over the next 24 h she continued to vomit intermittently. Intramuscular betamethasone was administered. She became progressively more unwell and tachycardic with cool peripheries. Urinalysis showed 2+ protein and 4+ ketones. Blood glucose was between 2.9 and 4.7 mmol/L. Arterial blood gas analysis showed a partially compensated metabolic acidosis (pH 7.29, $PaCO_2$ 2.7 kPa, HCO_3 13.7 mmol/L, base excess −14.4 mmol/L) with an increased anion gap (18.9 mequiv./L) and normal lactate (0.7 mmol/L).

After fluid resuscitation with compound sodium lactate there was little change in her metabolic derangement so she underwent emergency caesarean section under general anaesthesia. A 2.6 kg female baby was delivered, with an Apgar score of 5 at 5 min. Umbilical cord arterial and venous pH measurements were 7.15 and 7.17 respectively. She was transferred to the Intensive Care Unit and extubated 12 h after delivery after rapid resolution of the metabolic derangement.

4. Case 3

A 22-year-old woman presented at 32 weeks of gestation with severe vomiting and pain in her lower abdomen and back. She had been vomiting once daily for the majority of her pregnancy but this had worsened significantly over the preceding 24 h. Past medical history included two miscarriages, irritable bowel syndrome and migraine.

Examination on admission was normal except for tenderness over the symphysis pubis. Blood tests were within normal limits except for a mild neutrophilia (14×10^9/L). Urinalysis showed 1+ protein and 2+ leukocytes. Abdominal ultrasound was normal.

Vomiting continued after admission despite increasing doses of antiemetics and she remained nil by mouth. She was given intravenous 0.9% saline and compound sodium lactate. Urine dipstick on days 3 and 5 of admission showed 4+ ketones and 1+ protein. Arterial blood gas measurement was performed on day 5, which showed a partially compensated metabolic acidosis (pH 7.33, $PaCO_2$ 2.5 kPa, HCO_3 13.2 mmol/L, base excess −15.4 mmol/L) with a normal lactate (0.7 mmol/L) and normal anion gap (13.1 mequiv./L).

Intramuscular betamethasone was administered and emergency caesarean section with regional anaesthesia was performed after repeat arterial blood gas analysis showed the metabolic acidosis was worsening (pH 7.28, $PaCO_2$ 1.98 kPa, HCO_3 10.6 mmol/L, base excess −18.8 mmol/L). The baby was delivered in good condition with umbilical cord arterial and venous pH of 7.23 and 7.27 respectively. The Apgar score was 9 at 5 min. Following delivery, the vomiting immediately ceased and the acidosis resolved in the next 4 h without further intervention.

5. Case 4

A 32-year-old woman in her third pregnancy presented at 29 weeks of gestation with a 2 day history of severe vomiting that occurred even after sips of water. She had no abdominal pain, but had been constipated throughout this pregnancy and had last opened her bowels a week prior to admission. Past medical history included an appendicectomy aged 17 and two previous pregnancies complicated by hyperemesis gravidarum. She was on no regular medication but had continued to smoke 10 cigarettes a day whilst pregnant.

On admission, examination was normal apart from mild tenderness across her lower abdomen. Observations were normal. Blood tests showed a neutrophilia (25×10^9/L) with mildly deranged liver function tests (bilirubin 31 μmol/L, ALT 46 IU/L, AST 52 IU/L) and elevated urate (370 μmol/L). Urinalysis showed 4+ ketones and no protein or glucose. She continued to vomit despite the prescription of regular antiemetics and intravenous

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