Hypotension and intractable vomiting in the first trimester of pregnancy

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Hypotension and intractable vomiting in the first trimester of pregnancy

C Usalan, E Özarslan

A 24-year-old woman, gravida 1, para 1, presented at 12 weeks gestation with abdominal pain, weakness, fatigue, nausea and vomiting of several days duration. Ten days earlier she had been admitted to another hospital with the same complaints; she had been diagnosed as having hyperemesis gravidarum and hospitalised for 3 days, after which she was stabilized. Her medical history was unremarkable and she had no family history of relevant illness. On initial evaluation, she was in rather poor clinical condition and was pigmented to an abnormal degree, particularly around the eyes and in the skin creases of the hands; buccal pigmentation was also pronounced. Blood pressure was 100/60 mmHg, pulse 108 beats/min and body temperature was 38.7°C. Physical examination was unremarkable except for dehydration and hypotension. The laboratory examinations are summarised in the table. The results of urinalysis were: specific gravity 1.016, pH 5, 2+ protein, and urine sediment examination revealed a large number of erythrocytes and leukocytes. The urine contained Gram-negative bacteria.

Within two hours, the patient was confused, sweating and tachypnoeic. A repeat blood sugar level was 52 mg/dl. She was given a rapid infusion of intravenous 5% dextrose in 0.9% sodium chloride, but did not improve. Arterial blood gases showed a profound metabolic acidosis. Her acid-base status was as follows: pH 7.12, HCO_3 6 mmol/l, pO_2 154 mmHg, pCO_2 20 mmHg, base excess −22.

Table Initial laboratory findings

Variables	*Patient*	*Normal range*
Haemoglobin (g/dl)	14.6	12–18
Mean corpuscular volume (fl)	84	80–100
White cell count ($\times 10^9$/l)	16.6	3.6–10
Differential count (%)		
Neutrophils	78	40–70
Band forms	10	0–5
Lymphocytes	12	20–40
Platelet count ($\times 10^9$/l)	452	150–450
Sedimentation rate (mm/h)	90	0–15
Urea nitrogen (mg/dl)	28	8–23
Creatinine (mg/dl)	1.0	0.6–1.2
Total protein (g/dl)	6.8	6–7.8
Albumin (g/dl)	3.8	3.2–4.8
Uric acid (mg/dl)	6.2	2.7–7.8
Glucose (mg/dl)	62	70–110
Sodium (mmol/l)	124	136–147
Potassium (mmol/l)	5.6	3.5–5.5
Alanine transaminase (IU/l)	28	5–40
Aspartate transaminase (IU/l)	32	8–33

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Questions

1 What is the differential diagnosis compatible with the history, clinical and laboratory findings?
2 Which clues suggest an underlying endocrinologic illness?
3 What could be a precipitating factor leading to the aggravation of the underlying illness?

Answers

QUESTION 1
The differential diagnosis is septicaemia, severe hyperemesis gravidarum, adrenal crisis, acute thyrotoxicosis, or hyponatraemic encephalopathy.

QUESTION 2
The classical features of adrenal crisis (box 1) are present in this patient and the severe degree of metabolic acidosis suggests an underlying precipitating factor.

Classical clinical features of adrenal crisis

- symptoms: rapid worsening of preceding symptoms; severe weakness, fatigue, confusion, abdominal pain, nausea, vomiting, diarrhoea
- signs: fever, low blood pressure, dehydration, skin pigmentation
- laboratory findings: hyperkalaemia, hyponatraemia, high blood urea nitrogen, hypoglycaemia, metabolic acidosis (sometimes), low serum cortisol with high ACTH level (primary adrenal insufficiency), or low serum cortisol with low ACTH level (secondary adrenal insufficiency)

Box 1

QUESTION 3
The precipitating factor could be infection (sepsis, urinary tract infection, etc), trauma, surgery, withdrawal of therapy, or drugs (box 2).

Precipitating causes of adrenal crisis

- in diagnosed chronic adrenal insufficiency: withdrawal of therapy, infection, trauma, surgery, dehydration, drugs
- in a normal person: blockage of hormone synthesis (eg, by drugs such as aminoglutethimide, ketoconasole, etc, or as a result of surgery, eg, adrenalectomy), or increased degradation of hormones due to drugs such as rifampicin, dilantin, phenobarbital, etc
- following massive bilateral adrenal haemorrhage as may occur in severe infection (septicaemia, meningiococcaemia, etc), HIV, coagulation disorders, intra-abdominal surgery, adrenal metastasis
- hypophyseal causes: surgery, radiotherapy, apoplexy, thyroid hormone replacement without steroids in panhypopituitarism

Box 2

Outcome

The patient was treated with intravenous bicarbonate infusion, and intravenous ceftriaxone for suspected sepsis due to a urinary tract infection. Because her clinical and laboratory status were suggestive of acute adrenal insufficiency, we performed a rapid adrenocorticotropin (ACTH) stimulation test with a 0.25 mg intravenous bolus of tetracosactrin after taking blood samples for basal serum cortisol and ACTH. The test showed the basal plasma cortisol to be 5 μg/dl, with no evidence of a response to the tetracosactrin after 30 minutes. While the serum cortisol concentration was inappropriately low, the ACTH level was elevated. These results suggested acute adrenal insufficiency. A bolus intravenous infusion of 100 mg hydrocortisone followed by a continuous infusion of hydrocortisone at a rate of 10 mg/h was administered. Within 3 hours, there was restoration of blood pressure and body temperature and a general improvement was seen. Therapy was maintained with a continuous infusion of dextrose in 0.9% saline, antibiotics, and hydrocortisone. On day 2 of the admission the patient continued to improve. Her pH was normal, serum lactic acid levels had dropped significantly, and blood sugar levels were normal. Her electrolyte status also improved. Maternal and foetal monitoring during the pregnancy did not show any profound effects, and she delivered vaginally a healthy infant at 38 weeks gestation.

Discussion

A wide variety of metabolic and endocrine disorders may complicate pregnancy. Acute adrenal insufficiency is an emergency and is caused by a sudden, marked decline in levels of adrenocortical hormones.[1] In some cases, the condition first appears during the pregnancy, and in others acute adrenal insufficiency can occur in the course of a chronic insufficiency. Rarely, acute adrenal insufficiency may be the initial manifestation of new adrenal disease in pregnancy.[2] Acute adrenal crisis during pregnancy may mimic hyperemesis gravidarum.[3]

Previous case reports have suggested that acute adrenal insufficiency does not become manifest until the postpartum period. This may be partly due to a delay in diagnosis due to the similarity of symptoms common to pregnancy with those of Addison's disease, and partly because placental foetal steroid production protects the mother from crisis, although this latter point is controversial.[1 4–7] Alternatively, patients may develop acute adrenal crisis during pregnancy in the presence of precipitating factors such as severe infection, as in our patient.

Clinical symptoms, signs and laboratory findings in adrenal crisis are presented in box 1. In cases of adrenal crisis, the serum cortisol concentration is inappropriately low, and the ACTH levels will be elevated if the disease is primary and low in secondary adrenal insufficiency.[2] However, in the pregnant patient with hypoadrenalism, cortisol levels can be within the normal range for non-pregnant patients and the diagnosis will be based on the lack of a rise of plasma cortisol after ACTH stimulation.[5 6] In a patient with no known history of adrenal insufficiency, it can be difficult

to distinguish between Addison's disease and acute adrenal insufficiency, especially during the first trimester of pregnancy. It is important to remember that mild cases can go undetected during pregnancy and become manifest as crises at parturition or in the presence of other illness such as urinary tract infection, dehydration, pre-eclampsia, etc.[6 7]

In conclusion, acute adrenal insufficiency is a rare disorder whose diagnosis can be difficult during pregnancy. On the other hand, it is associated with high maternal and/or foetal morbidity and mortality if allowed to progress. For this reason, early recognition and intervention are critical.

Final diagnosis

Acute adrenal insufficiency precipitated by urinary tract infection.

Keywords: adrenal insufficiency; pregnancy; urinary tract infection; vomiting; hypotension

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Fatal complication of coincidental operative finding

J S McCourtney, S Karim, M Rahilly, R Dalling

A 65-year-old man with an established diagnosis of Crohn's colitis presented as an emergency with peritonitis and free subdiaphragmatic air on erect chest X-ray. At laparotomy, a grossly distended colon was noted, with two sites of perforation at the rectosigmoid and splenic flexure areas. Multiple non-dilated jejunal diverticula were noted coincidentally. Subtotal colectomy with cross-stapling of the rectum and formation of an end ileostomy was performed. He made an uneventful early postoperative recovery but during the fourth postoperative week he developed intermittent, colicky abdominal pain, vomiting and reduced ileostomy output. Subacute small bowel obstruction secondary to adhesions was diagnosed after clinical and radiological examination. Conservative treatment with intravenous fluids and nasogastric decompression produced an initial improvement but on the 32nd postoperative day the patient suddenly collapsed and died from a cardiorespiratory arrest secondary to acute renal failure and septic shock. At post-mortem examination, evidence of proximal jejunal diverticular disease with signs of acute diverticulitis and perforation were noted along with widespread peritonitis. No histological features of Crohn's disease were identified in the segment of perforated jejunum, or elsewhere in the bowel, and the inflammatory process was centred around the diverticulum (figure).

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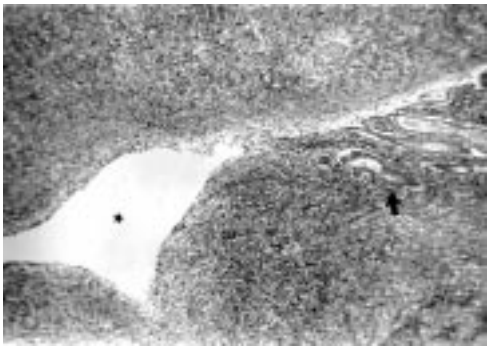

Figure Perforated jejunal diverticulum lined by inflamed granulation tissue. Some residual diverticular mucosa is visible on the right (arrow). The lumen of the perforation is indicated by ⋆

Questions

1 What is the incidence of small bowel diverticula?
2 What is the aetiology of small bowel diverticulosis?
3 List the common pre-operative clinical manifestations of small bowel diverticula.