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Nephrology Dialysis Transplantation

Nephroquiz

Abdominal pain and hypertension in the third trimester of pregnancy. Is pre-eclampsia the only explanation?

Keywords: coagulation deficit; hypertension; intestinal infarction; pre-eclampsia; pregnancy

The case

A 29-year-old woman, at the 27th week of her fifth pregnancy, called the emergency room because of severe abdominal pain since the previous day, emesis and vaginal bleeding. At the home visit, blood pressure was 140/90 mmHg. She was previously normotensive. Her four previous pregnancies ended at term with vaginal delivery and the course of the present one was uneventful. No personal or family history of diabetes, hypertension, thrombophilic diathesis or other severe illness was reported. She was not on any oral therapy and denied alcohol and drug use. At the beginning of pregnancy her BMI was 34.

Pre-eclampsia was suspected and she was referred to an obstetrics and gynaecology referral centre for high-risk pregnancies.

At referral, blood pressure was 180/110 mmHg and was only partially responsive to nifedipine. At the first hospital visit, the patient was alert, afebrile but suffering: the abdomen was diffusely tender with peritonism. Slight oedema was present and costovertebral angle percussion elicited sharp bilateral pain. Patellar reflexes were normal. She reported no stool or gas passage in the last 24 h.

The main biochemical data are reported in Table 1.

The development of the fetus was adequate for gestational age, with normal amniotic fluid and normal uterine and umbilical blood flows. Maternal ascites were detected at ultrasound.

Table 1. Biochemical data at admission

	Results	Normal range
WBC (μl)	33 140	4000–11 000
Hb (g/dl)	14.2	12.0–16.0
Hct (%)	40.7	36.0–46.0
PLT (μl)	283 000	150 000–400 000
Neutrophils (%)	87.5	30–70
Prothrombin activity (%)	101	70–120
INR	0.97	0.8–1.2
aPTT (s)	25.3	26–36
aPTT ratio	0.80	0.8–1.2
Fibrinogen (mg/dl)	730	200–400
AT III (%)	92	80–120
BUN (mg/dl)	22	5–25
Serum creatinine (mg/dl)	0.79	0.60–1.20
Uric acid (mg/dl)	4.74	2.40–6.00
Amylase (U/l)	15	0–100
Lipase (U/l)	69	<60
AST (U/l)	25	<35
ALT (U/l)	29	<35
GGT (U/l)	10	<30
ALP (U/l)	143	100–280
Bilirubin tot (mg/dl)	0.69	0.30–1.10
LDH (U/l)	400	210–460
Total proteins (g/dl)	5.52	6.50–8.50
Albumin (g/dl)	2.83	3.92–5.18
CRP (mg/l)	159	0.0–10.0
Proteinuria (dipstick)	+/−	Absent.

WBC: white blood cells; Hb: haemoglobin; Hct: haematocritus; PCT: platelets; INR: international normalised ratio; aPTT: active partial thromboplastin time; ATIII: antithrombin III; BUN: blood urea nitrogen; AST: aspartate aminotransferase; ALT: alanine aminotransferase: GGT: gamma glutamyltransferase; ALP: alkalyne phosphatase; LDH: lactate dehydrogenase; CRP: C reactive protein.

Quiz

- What is your differential diagnosis?
- What would you suggest to do?

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Answer to the quiz in the preceding page

The young lady suffered from haemorrhagic intestinal infarction.

Follow-up

Her clinical condition progressively worsened in the 8 h following hospitalization. A CT scan was performed to further investigate the abdominal picture and it confirmed the presence of moderate ascites and coecal oedema. Control of hypertension became critical. Heart rate increased to 140 beats per minute, oxygen saturation decreased to 92% (ambient air).

An acute abdominal surgical problem was suspected (the first hypothesis being intestinal occlusion due to a volvulus or intussusceptions), and an exploratory laparotomy was performed.

At surgery, sero-haematic ascites were drained; intestinal loops were ischaemic for a tract of ~80 cm, from the duodeno-jejunal angle to 25 cm from the ileo-coecal valve. The jejuno-ileal ischaemic tract was removed and a termino-terminal anastomosis was performed. At the same time, the baby was delivered by Caesarean section, as we feared that the critical maternal conditions and possible post-surgical complications might compromise fetal well-being. The baby was a female of normal weight for gestational age (weight 980 g, Apgar index 6 at 10 min).

The pathological specimen (Figure 1) showed mesentery with haemorrhagic infarction and disseminated vascular thrombosis of small venous and arterial vessels. The intestinal mucosa was ischaemic but not yet necrotic. The picture suggested an acute vascular event, in the absence of chronic ischaemia.

The subsequent post-surgical follow-up was characterized by segmental thrombosis of the peroneal vein, responsive to subcutaneous heparin therapy. The patient was discharged 12 days after surgery and is presently in good clinical condition.

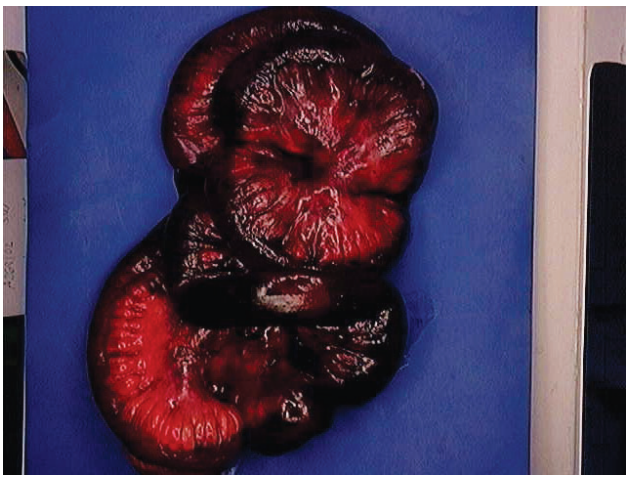

Fig. 1. Mesentery with haemorrhagic infarction and jejuno-ileal ischaemic tract. The distal loop presents a normal aspect, in contrast to the infarcted middle loop (see arrows).

A complete thrombophilic screening was performed to search for predisposing factors. The major finding was a low protein S level (46.4%), with heterozygosity for a prothrombin mutation (G20210A), while all other data tested negative (protein C, APCR, V Leiden factor mutation, MTHFR mutation, APA, ACA, A-ANA, ENA, ANCA, anti-nucleus antibodies).

The daughter was hospitalized in the Neonatal Intensive Care Unit for ~2 months, because of her prematurity and low birth weight. She is now in her 12th month of life and developing normally.

Comment

A clinical picture of hypertension and abrupt abdominal pain during the third trimester of pregnancy is usually considered highly suggestive of severe pre-eclampsia syndrome [1–2].

Some of the diagnostic data were missing in this patient (no proteinuria, only slight oedema, normal patellar reflexes) and the abdominal pain was diffuse, without the emblematic feature of epigastric pain. However, atypical cases are not exceptional, in particular at initial presentation [1–2].

Although inflammatory or infectious signs are not rare in pre-eclampsia (high C-reactive protein levels, leukocytosis), the extremely high values in this case suggested a different acute infectious-inflammatory state [1–2]. The absence of fever suggested a Gram-negative sepsis, a hypothesis supported by the low oxygen saturation and high heart rate, but completely discordant with the hypertensive picture. The acute bilateral pain elicited at costovertebral angle percussion could be misleading in this context, adding acute pyelonephritis to the differential diagnosis (however, no symptom was present and bilateral pictures are rare). The finding of ascites at obstetric echography definitively indicated an intestinal problem.

Pregnancy is a high-risk condition for the development of acute vascular events [3]. There were additional risk factors in this patient, despite her young age: obesity and multiparity. The finding of low protein S levels and a heterozygous mutation at the G20210A locus of the prothrombin gene provided a genetic basis for the pro-coagulant status.

In addition to pre-eclampsia, the differential diagnosis of acute abdominal pain during pregnancy takes into account major abdominal catastrophes (the same as those occurring in non-pregnant persons) and rare events like the one described here, possibly triggered by the particular metabolic, hormonal and mechanical situation of pregnancy [3–4].

A recent review of the 'imitators of pre-eclampsia and eclampsia' suggested considering sepsis (a possible consequence of intestinal infarction), autoimmune diseases (for the sake of this discussion, their complex links with congenital coagulopathies can be mentioned), haemolytic uraemic syndrome, as well as

thrombotic thrombocytopaenic purpura and acute fatty liver of pregnancy. Since perinatal mortality is relatively high and the maternal risks are appreciable in all these conditions, the imitators of pre-eclampsia should be considered at least as severe as eclampsia itself [4–5].

In all rare diseases, each case should be managed separately, and cumulative risk assessment or therapeutic guidelines are not feasible. Nevertheless, referral to a tertiary centre specialized in high-risk pregnancies is currently one of the few demonstrably effective therapeutic tools for materno-fetal survival [6].

Conflict of interest statement. None declared.

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