(Fig. 1E). Moreover, our research team performed another test by using sodium tanshinone II a sulfonate to inhibit the expression of P-gp in normal pregnant rats with higher digoxin transfer rate, which confirmed the efflux function of P-gp for digoxin in healthy condition (data not show).

In clinical practice, digoxin has been used as the first line medication to treat fetal heart arrhythmia and failure [3]. But digoxin demonstrated a lower transplacental distribution with down-regulated P-gp expression. So that P-gp is not the main cause inducing adverse transplacental distribution under fetal heart failure, while many studies focused on the digoxin transplacental transfer previously reported that digoxin could diffuse through placenta facilely in normal animal and human researches [4]. Besides, in the investigation using a perfused human placental lobule model it showed that adverse transplacental transfer in the condition of increased pressure load of fetal circulation inducing lower digoxin concentration in fetal circulation [5]. Thus, P-glycoprotein was not a factor inducing lower transplacental digoxin transfer, and we simply considered that the damages of placenta and the increased interstitial hydrostatic pressure may play a more important role in digoxin transportation across maternal-fetal barrier than the function of P-gp in fetal heart failure.

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Early plasmapheresis as a successful treatment in hypertriglyceridemia-induced acute pancreatitis in first trimester pregnancy following in vitro fertilization

Dear Editors,

We describe (with the informed consent of the patient) the successful treatment of hypertriglyceridemia induced acute pancreatitis (AP) with early plasmapheresis in a pregnant diabetic woman who had no familial hypertriglyceridemia, during the first trimester of pregnancy after in vitro fertilization.

A 42-year-old multipara woman, in the 9th week of gestation following in vitro fertilization, was referred to the emergency ward for an acute abdominal pain. The epigastric pain was continuous and radiating to the back, with aggravation in supine position and relief on crouching forward. There was no history of familial hypertriglyceridemia but the patient was treated for diabetes by dietary therapy. Her physical examination was unremarkable excepted for abdominal sensibility; hemodynamic parameters were stable but the patient was hyperventilating to compensate a metabolic acidosis (bicarbonates 10 mEq/L). The TG (9850 mg/dL – normal 15–158 mg/dL) and lipase (1592 U/L – normal <60 U/L) levels were high without hyperleucocytosis (6130/mm^3 – normal 4500–10,500/mm^3). Other biological parameters were normal ruling out etiologies such as hypercalcemia; no pharmacological treatment such as valproic acid, azathioprine or sulfonomides could explain the pancreatic enzymes abnormalities. Magnetic resonance imaging findings were consistent with AP. Despite good clinical condition on admission to the hospital, she was admitted in the intensive care unit for early plasmapheresis to prevent further complications under optimal follow up. We used cristalloid solution and albumin for therapeutic plasma exchange with a volume of 40 mL per kg along with heparin infusion 10 U/kg/h for anticoagulation. The daily plasmapheresis treatment was ceased after four days as there was a significant drop of TG after four sessions (Fig. 1). In 4 days time, with TG levels of 712 mg/dL (and cholesterol 181 mg/dL – normal <200 mg/dL), she was discharged from ICU. No other treatment was necessary to stabilize the patient in ICU. After discharge from the hospital, she regularly visited the outpatient department without any complications during this pregnancy till delivery.

Commment

Hypertriglyceridemia is a serious complication during pregnancy. Plasma TG increase two-fold to four-fold in pregnancy, principally in the third trimester, because of increased TG-rich lipoprotein production and decreased lipoprotein lipase activity [1]. In women with diabetes and abnormal lipoprotein metabolism, this can lead to severe hypertriglyceridemia, precipitating pancreatitis risk. Nutrition and pharmacologic therapy may be essential in preventing further complications and improve the outcome.

Hypertriglyceridemia-induced pancreatitis during pregnancy or following in vitro fertilization ovulation induction have been reported previously [2]. Family history of lipid abnormalities should be elicited, and an attempt to identify secondary causes made. A serum TG level >1000 mg/dL is an identifiable risk factor. Reduction of TG levels to well below 1000 mg/dL effectively prevents further episodes. There was in our patient, previous history of, diabetes mellitus without lipid abnormality, but no alcohol ingestion, or any other precipitating factors apart in vitro fertilization. To prevent AP, hospitalization for intravenous fluid therapy and plasma exchange could be required. Plasmapheresis and extracorporeal lipid elimination are therapeutic alternatives and can dramatically reduce excessive lipid levels [3], as seen in our case.

Pancreatitis in pregnancy is associated with a maternal death and significant fetal loss rate. These rates are declining with earlier diagnosis and treatment of the disease. The commonest reasons for maternal and fetal mortality are AP itself and, very rarely preeclampsia–eclampsia or HELLP syndrome. In our case, pregnancy had not to be terminated. The incidence of preterm delivery and perinatal death in these patients is higher than that in the general obstetric population [4]. Ramin et al. demonstrated that, of pregnancies complicated with pancreatitis, 19% had AP in the first trimester, 26% in the second, 53% at more than 28 weeks gestation,

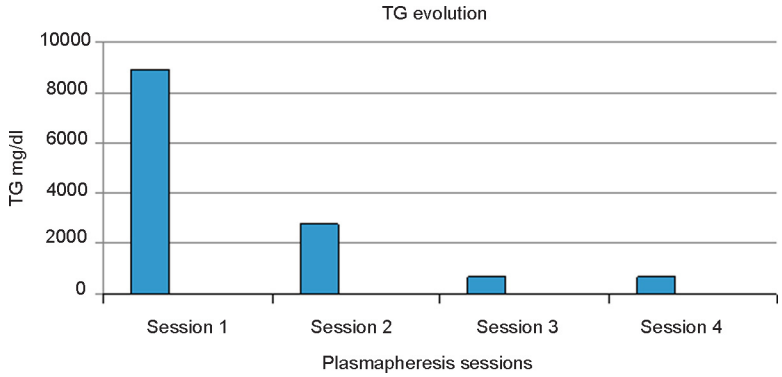

Fig. 1. The time-course of plasmapheresis therapy: after the first session of plasmapheresis triglycerides (TG) 8900 mg/dL reduced to 2800 mg/dL, after second session to 705 mg/dL, and after third session to 712 mg/dL. The patient was taken to the ward following the four sessions – plasmaphresis to continue with supportive treatment.

and 2% postpartum [5]. The incidence of pancreatitis during pregnancy is low, certainly during the first trimester.

Plasmapheresis is an alternative treatment for the cases with hypertriglyceridemia resistant to the medical therapy but the early application of plasmapheresis in parturients with hypertriglyceridemia-induced AP may improve the clinical course and prevent maternal and fetal complications. No side effects of plasmapheresis were noted in our case; this technique seems to be safe in pregnant woman but admission in ICU could be recommended for optimal follow up.

This case again confirms the importance of pre-screening for triglyceride levels and careful physical examination before initiation of estrogen-related medical treatment.

Conflict of interest

None.

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Bromocriptine-induced coronary spasm in postpartum

Dear Editors,

We report the case of a 26-year-old woman with a postpartum coronary spasm probably induced by bromocriptine. Her medical history was unremarkable, as was her pregnancy. She had no personal or familial cardiovascular risk factors. A caesarean was performed at term for a breech presentation, and her immediate postoperative recovery was uneventful. Oral administration of bromocriptine (2.5 mg twice daily) for lactation suppression began on day 1. Her only other medication was trimebutine (100 mg, three times daily). On day 2, she suddenly developed tetraparesis, predominantly on the left side, and bilateral blindness. Laboratory tests, including blood glucose level, were normal. Cerebral magnetic resonance imaging (MRI) showed diffuse bilateral cortical lesions compatible with hypoxic lesions. The electrocardiogram (ECG) showed T-wave inversions over the inferior leads. Computed tomography of the thorax showed no evidence of pulmonary embolism. The troponin I level (41 ng/l, $N < 0.01$) was elevated. Echocardiography showed a small pericardial effusion, inferior akinesia, and a normal left ventricular ejection fraction. While coronary angiography appeared normal, the cardiac MRI showed a transmural infarction of the right coronary artery (Fig. 1). Its transmural and segmental extent combined with the perfusion defect confirmed the diagnosis of myocardial infarction. One month later, echocardiography and cardiac MRI both confirmed residual inferior necrosis.

Subendocardial and nontransmural lesions on cardiac MRI are pathognomonic of myocardial infarction, a diagnosis also supported by elevated troponin I. The apparently normal coronary angiography, showing no atherosclerotic lesion in the artery associated with the MRI-documented infarct, strongly suggests a coronary spasm. This event happened the day after initiation of bromocriptine. These findings are consistent with the diagnosis of a prolonged coronary spasm induced by bromocriptine that caused a myocardial infarction, despite healthy coronary arteries. Heart rhythm disorders secondary to this inferoseptal infarction might have reduced cerebral blood flow, which might in turn explain the neurological deficit. Heart monitoring in the cardiac intensive care unit showed no atrial fibrillation. Transoesophageal echocardiography confirmed the absence of any valvular tumour or intracavitary thrombus. The latter was also ruled out by MRI.

Bromocriptine is an ergoline derivative sometimes used to prevent postpartum lactation. Its dopamine receptor agonist activity can induce coronary vasoconstriction [1]. In 1986, Iffy reported the first case of postpartum myocardial infarction