in metabolic and hormonal derangements that lead to anovulation and amenorrhea presumably because of hypothalamic-pituitary dysfunction (5, 6). For example, hypogonadotropic hypogonadism as well as elevated levels of estrogen are seen. Hyperestrogenemia occurs due to aromatization of weak androgens from the portal circulation and consequently ovulation is prevented. Women with alcoholic hepatitis have early menopause. In alcoholic patients between 20–40 years old, decreased numbers of follicles and no corpora lutea are seen. These changes are reversible because fertility is restored after liver transplantation. Full sexual development returns within 3 years of transplant (11, 12). Successful pregnancies can be achieved in anovulatory cirrhotic patients with assisted reproductive technology. Concomitant diseases and nutritional deficiencies may also impair fertility (11).

Pregnancy is also associated with an increase in portal pressure (8). Portal hypertension develops as a result of several etiologies. Liver cirrhosis is the most common cause of portal hypertension. Other causes, like extrahepatic portal vein obstruction, non-cirrhotic portal fibrosis, portal vein thrombosis, Budd–Chiari syndrome, infection or congenital hepatic fibrosis contribute significantly to non-cirrhotic portal hypertension (NCPH). Mostly liver function is much better preserved in women with NCPH and pregnancy is spontaneous in these women (13).

Numerous hemodynamic and physiological changes occur during pregnancy. One of the earliest changes is an increase in plasma output by 40–50%. Maternal cardiac output increases by 30–50% due to increase in stroke volume and the heart rate. There is a decline in systemic vascular resistance as a result of the progesterone effect and development of placental vascular beds. As a result of all of these changes, there is a profound alteration in the systemic hemodynamics, resulting in a hyperdynamic state with increased pulse pressure. These changes can worsen portal hypertension in pregnant patients with portal hypertension and significantly increase the risks of variceal hemorrhage (4). Because TIPS is a rare situation in connection with pregnancy, we cannot assess the possible effect on the placental bed. According to available literature there is limited data about patients who have had TIPS during pregnancy. No effect on TIPS patency has been reported in a few patients, but only a moderate increase in the flow velocity in the portal vein, the stent, and the hepatic artery (14).

The most common cause of maternal death in liver cirrhosis is massive upper gastrointestinal bleeding usually due to esophageal varices (15, 16). The risk of bleeding from varices has been estimated at 20–27 % among pregnant women with cirrhosis; however, this increases to 62–78 % if esophageal varices are present (3, 6). The mortality rate from variceal bleeding is between 2 % to 6 % (5). All pregnant women with cirrhosis should have an initial screening endoscopy, which is recommended during the early second trimester or before pregnancy. Pregnant patients at risk for variceal bleed should receive primary prophylaxis, with either endoscopic variceal ligation or beta-blockers. Although there has been a reported association of beta-blockers with intrauterine growth retardation and neonatal bradycardia, nonselective beta-blocker therapy can be used as variceal bleeding prophylaxis in pregnancy (17). In women with an episode of acute variceal bleeding during pregnancy, treatment should be focused on the resuscitation and hemodynamic stabilization of the mother, antibiotic prophylaxis, and endoscopic therapy. The mainstay of treatment remains endoscopic variceal ligation (13). The use of vasopressin is not recommended during pregnancy due to the risk of arteriolar vasospasm and an increased risk of placental abruption, myocardial infarction, peripheral ischemia and hypertension. The use of octreotide is controversial (13). Third generation cephalosporins can be used for prophylaxis against spontaneous bacterial peritonitis in the event of a variceal bleed but fluoroquinolones are contraindicated in pregnancy (13). TIPS is contraindicated during pregnancy due to the risk of fetal radiation exposure. The placement is considered only if the medical treatment or the endoscopic procedures fail to control the variceal hemorrhage (2, 13).

Another complication in these women is hepatic decompensation leading to hepatic encephalopathy. This can occur during all stages of pregnancy but often occurs after an episode of variceal bleeding. The basis of treatment is using both lactulose and antibiotic therapy. According to available literature, the preferred antibiotic therapy is Metronidazol to Rifaximin, because Metronidazol is safer in pregnancy. Renal dialysis is the modality for hepato-renal shutdown. Terlipressin is contraindicated in pregnancy as it may exert oxytocic effect (13).

Liver transplantation is considered in patients with advanced liver disease before pregnancy. Liver transplant recipients possess an improved quality of life, their hormonal imbalance return to a normal state, ovulation resumes, and pregnancy may ensue (8). In our case, our patient's pregnancy was unplanned, so we did not consider a liver transplantation.

There is limited data about an intercurrence of alcohol-induced liver cirrhosis and lymphomas in the literature, consisting mostly of case reports (18–22). The evidence of the possible causal connection of these two diseases is thus low. Alcohol consumption is known to be a risk factor for developing cancers of the oral cavity, pharynx, larynx, esophagus, liver, colon, rectum, and female breast (23).

Alcohol consumption and the risk of lymphoid malignancies was evaluated in two large prospective cohort studies. One of the studies found a slightly increased risk of developing plasma cell neoplasms (multiple myeloma, lymphoplasmocytic lymphoma) and chronic lymphocytic leukemia in alcohol drinkers. Nevertheless, both of the studies failed to prove a statistically significant association between alcohol consumption and incidence of LBL or other aggressive lymphomas (24, 25).

Conclusion

Cirrhosis in pregnancy is a complex situation which requires a multidisciplinary approach. Neonatal risks are increased as well as maternal, which includes mainly bleeding (due to variceal bleed, splenic artery aneurysmal rupture, postpartum bleed) or liver failure.

We demonstrated the successful pregnancy in a 37-year-old female patient, who, despite having an irregular menstrual cycle and one episode of acute bleeding from esophageal varices in the past, managed the entire course of pregnancy without any bleeding due to TIPS. She gave birth to a healthy baby with physiological measures,