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# Case study

# Placental cyst – case study



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# **Abstract**

We present a case of a large and growing protruded subchorionic placental cyst noted during routine ultrasound evaluation at 25.6 weeks in a pregnant woman with polycystic ovary syndrome (PCOS) and simple left ovarian cyst. We observed evidence of progressive and persisted be weekly repeated reduction of fetal cerebral-placental blood flow. Prenatal ultrasound evaluation, including detailed fetal echocardiography, allowed for safe pregnancy continuation and avoidance of the prematurity. Wide placental infarction, fibrin exudation, decalcifications, and severe disturbances in fetal and maternal circulation were reported based on pathologic postnatal evaluation.

Key words: placental cyst, PCOS, fetal echocardiography, prenatal echocardiography

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### Introduction

Subchorionic/chorionic or membranous placental cysts are echo-free cavities with no blood flow within, and they have a prevalence of approximately 2–7%. A large placental cyst located near the umbilical cord insertion or multiple cysts (with more than three cysts) could induce partial occlusion of umbilical cord blood flow and possible haematomas formation, infarction, fetal growth restriction, preterm birth, and/or neonatal morbidity [1–20].

# Case study

A G2P0 30-year-old pregnant woman was referred at 26 w 5 d to our Institution for fetal echocardiography due to a diagnosis of a placental cyst at a routine second-trimester scan performed elsewhere. She had gynaecological history of PCOS

and had been under gynaecological oncologist observation for two years due to a simple left ovarian cyst:  $53.5 \times 27.7$  mm (width  $\times$  height) with no growth progression nor higher risk of malignancy algorithms, and she had had hormonal therapy before pregnancy, without any ovulation induction nor in vitro fertilisation.

The first trimester combined test for common trisomy was negative. The second trimester routine anomaly-scan was negative for congenital anomalies. The patient had recurrent infections in her pregnancy: bronchitis and persistent vaginal infections, treated with antibiotics, so she had a very tight obstetric schedule.

The first ultrasound anomaly was detected at 25.6 weeks when we saw a single cyst at the fetal side of the placenta with the size of  $38.4 \times 37.4 \times 29.9$  mm and volume of 22.484 cm<sup>3</sup>,

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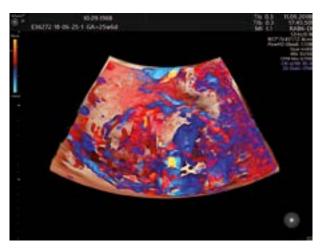


Figure 1. Placental villous capillaries at 25.6 weaks

the placenta was positioned at the posterior wall, and the cyst was located near the placental marginal cord insertion. The cyst was anechoic with no masses, with very thin wall, and no c-Doppler flow within it. The enlargement of the cyst in the

pregnancy was as follows: cyst size ranged from  $43.2 \times 41.3 \times 39.5$  mm, volume of 36.9 cm<sup>3</sup> at 27.5 weeks to, finally:  $72.8 \times 68.1 \times 55.2$  mm, volume of 143.29 cm<sup>3</sup> at 37.5 weeks (Figures 1–5).

The placenta was growing adequately until 34.5 weeks of gestation, when infection and oedema of the placenta was suspected: the thickness/length/width of the placenta ranged from  $47.1 \times 16.17 \times 13.56$  mm, with volume of 540.741 cm<sup>3</sup> at 34.5 weeks to  $83.5 \times 15.28 \times 12.01$  mm, volume of 802.327 cm<sup>3</sup> at 38.2 weeks (Figures 4, 5).

Figure 4 shows the chart of growth of the placental cyst volume and placental plate volume in gestation (25.6–37.5 weeks). Figure 5 shows the ratio of the placental cyst volume to the placental plate volume in the pregnancy (36.5–38.2 weeks). Apart from the placental abnormalities, the fetal growth charts were adequate throughout the time of gestation.

The echocardiographic evaluation was performed three times, at 20 weeks, 27.5 weeks, and 29.6 weeks, and apart from the reduced contractility of the ventricles, with the 18% of shortening fraction for RV and 25% for LV, the fetus was cardiovascularly stable with a 9/10 CVP score (cardio-vascular profile).



Figure 2. Placental cyst at 25.6 weaks

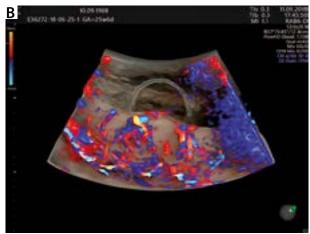






Figure 3. Placental cyst at 27.5 weaks, located near the placental marginal cord insertion

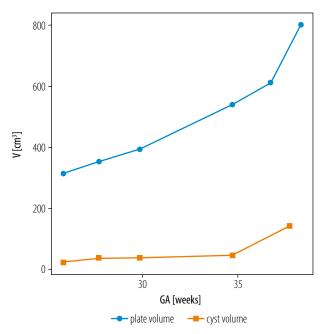


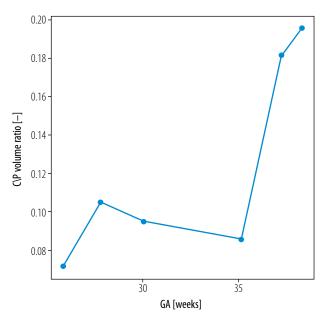
Figure 4. The picture of growth of the placental cyst volume and placental plate volume in gestation (25.6–37.5 weaks)

Table 3. PI UMBA, RI UMBA, PI MCA, PS MCA, RI MCA values in pregnancy complicated by placental cyst

GA [weeks]	PI UMBA	RI UMBA	PI MCA	PS MCA [cm/s]	RI MCA
25.6	1.08	0.66	1.98	26.91	0.84
27.5	1.10	0.70	1.54	37.23	0.77
29.6	1.08	0.69	1.29	41.71	0.74
31.6	0.77	0.54	0.79	30.63	0.51
34.5	0.69	0.50	1.46	19.42	0.74
36.5	0.69	0.48	1.01	22.39	0.64
36.6	0.71	0.50	1.03	22.47	0.62
37.0	0.75	0.53	1.01	38.67	0.65
37.5	0.67	0.50	1.09	23.40	0.7
38.2	0.64	0.46	1.03	21	0.61

The prenatal course, except of the placental enlargement and growing of the placental cyst, was uneventful up to 36.5 weeks. Then we discovered evidence of progressive and persistent biweekly repeated reduction of fetal cerebral-placental blood flow with the CPR (cerebroplacental ratio): 0.67 at 36.5 weeks (PIUMBA = 0.69; PSUMBA = 42 cm/s; PIMCA = 1.02; PSMCA = 28 cm/s) and 0.62 (PIUMBA = 0.64; PSUMBA = 53 cm/s; PIMCA = 1.03; PSMCA = 21 cm/s) at 38.2 weeks (Table 1) with fetal tachycardia of 180 bpm and a probable false umbilical knot near the fetal face documented by ultrasound (Figure 6).

A scheduled caesarean section was performed at 38.5 weeks because of these findings and with the intention to remove the left ovarian cyst during the same operating cycle. A 3300 g baby girl was delivered; Apgar scores were 10, 10, and gasometry: pH: 3, 7.31; pH: 7,35; BE = -0.7; BE = -1.2. The



**Figure 5.** The picture showing the ratio of the placental cyst volume to the placental plate volume in the pregnancy (36.5–38.2 weaks)

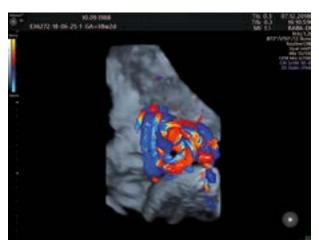


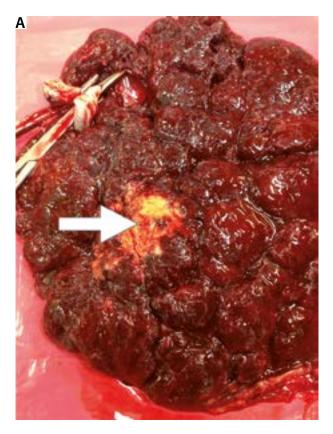
Figure 6. False umbilical knot at 38.2 weaks

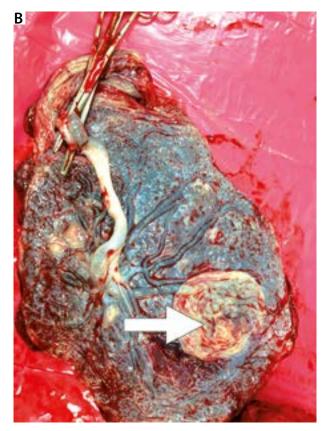
baby girl had a neonatal erythema with good reaction to the potassium permanganate and jaundice with a bilirubin value at the third day of life, on the day of discharge, of about 9.6 mg/dl.

Pathologic postnatal evaluation revealed that the placenta weighted 600 g, measured  $15 \times 118 \times 2$  cm, and with an umbilical cord length of 40 mm of the marginal cord insertion. The microscopic examination showed wide limiting of the exchange placental surface: wide placental infarction, fibrin exudation, decalcifications, severe disturbances in fetal and maternal circulation, and a sole membranous cyst located near the marginal placental cord insertion (Figure 7). The left ovarian cyst was reported by pathologists as cystadenoma mucinosum.

#### Discussion

The thickness of the anterior placenta greater than 33 mm and 40 mm for the posterior placenta should be considered as abnormally thick placenta, based on the pilot study of Lee et al. [21]. In our case, we observed a posterior positioned thickened placenta with a placental cyst. Many studies show the correla-





**Figure 7.** The placenta weighted 600 g, measured 15  $\times$  118  $\times$  2 cm, with an umbilical cord length of 40 mm of the marginal cord insertion, with no true umbilical knot, with wide placental infarction (maternal side – A), and with the sole membranous cyst located near the marginal placental cord insertion (fetal side – B)

tions between abnormally thick placenta and poor outcome, with higher risk of perinatal mortality and growth restriction. Placental alterations have been associated with a wide variety of maternal and fetal conditions, including infections, diabetes, PCOS, and hydrops [21–33].

Placental chorionic plate cysts are usually benign, but such large and growing placental cysts, as presented in our study, are reported very rarely. They are often associated with progressive reduction in fetal blood flow and placental-maternal floor infarction [2, 4, 11, 31, 32].

In pregnant women with PCOS gestational androgen excess and insulin resistance might alter and contribute to endovascular trophoblast invasion. Adipokines produced by placenta and ovaries have a significant effect on fetal and placental growth, and programming of insulin sensitivity. Placental growth factor is increased in women with PCOS and might play a role in placental cyst pathogenesis and the angiogenic placental dysregulation mentioned in our case [33–37].

Recurrent symptomatic infections observed in our case supported the evidence of the presence of chronic low-grade inflammation in women with polycystic ovarian syndrome [38]. If any recurrent infections occur in pregnancy, we recommend a schedule of prenatal visits: every two weeks after 26 weeks, to monitor fetal growth, repeated, detailed fetal echocardiography, and ultrasound placental evaluation for the continuation of a safe pregnancy and avoidance of prematurity.

#### **Conflict of interest**

The authors declare no conflict of interest.

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