INTRODUCTION

Premature separation of a normal inserted placenta or placental abruption is a very serious obstetrical pathology determined by placental disruption from the wall of the uterus, after 20 weeks of gestation and before the expulsion of the fetus. The condition’s gravity resides in its rapidity of onset and in its severe consequences, resulting in maternal-neonatal morbidity, and sometimes even death.1,2 The etiology is still largely unclear and would, once clarified, represent a valuable aid in providing the most appropriate prevention and treatment. Some authors raise the question of vascular changes in the pathogenesis, but there are still not enough data to fully support this hypothesis.3 Stimulated by these controversies and by the clinical importance of helpful etiological insights, we designed a study to investigate the placental morphological vascular aspect in patients with placental abruption.

MATERIALS AND METHODS

The study was designed as a prospective cohort on 95 subsequent pregnant women, aged between 15 and 42 years, who were hospitalized and have delivered in our department during 2001-2005, with
singleton pregnancies and placental abruption, after 22 - 42 gestational weeks. Arterial hypertension was the major concomitant pathology, reported in 64% of the included patients. All cases were emergencies; were considered severe and all patients were delivered by cesarean section.

Placentas were collected and morphologically analyzed. Placental weight varied between 260 g and 650 g, for most of them (60 cases) between 450 g and 600 g. The volumes of the hematomas were between 50 - 1000 ml. The placentas were injected with Glycol Methacrylate Technovit® 4173 (Kulzer GmbH, Wehrheim, Germany), according to the manufacturer’s instructions. (Fig. 1) We have used the red color for placental arteries and blue for placental veins. The specimens were, than, immersed in sodium hydroxide solution and corroded. Placental vascular structure was, thus, obtained. We analyzed the morphologic vascular patterns and we looked for correlations of these data with the patients’ outcome: uterine status and maternal – fetal jeopardy.

**RESULTS**

Placental vascular tree could be adequately observed in all cases. (Fig. 2) The major vascular observation was, in the study group, the Hyrtl anastomosis, which was encountered in 68 cases. (Fig. 3) This counts for 71.57% of all the 95 patients with placental abruption included in the study. (Fig. 4)

No important vascular morphological abnormalities were otherwise noted. In some cases, at the place of the hematoma possible vascular disruptions were observed, especially in the placental arterial territory. Results were, however, inconclusive, and a firm conclusion could not be stated.

Some of the cases had a very severe outcome. Within the group, two cases of maternal death and 23 cases of fetal death were registered. Maternal hypertension was present in all of the cases which resulted in maternal or fetal death. In nine patients extensive uterine alterations were noted (a majority of the myometrium infiltrated by the blood – up to the serosa), Reeb test was negative, some had consumptive coagulopathy and the medical decision was to perform
hysterectomy, after extracting the fetus. The extension of the lesions imposed supracervical hysterectomy in one case and total hysterectomy in eight patients: two cases with adnexal preservation, two cases with left adnexectomy and four cases with bilateral adnexectomy.

Figure 4. Incidence of the Hyrtl anastomosis among the 95 patients with placental abruption included in the study.

Hyrtl anastomosis was present in one of the cases of maternal death and in 14 of the placentas corresponding to fetal death, meaning 60.87% of all corroded placentas. (Fig. 5) This vascular pattern was noted in 4 of the 9 cases which necessitated hysterectomy. (Fig. 6)

Figure 5. The proportion of placentas with presence of the Hyrtl anastomosis among the cases resulting in fetal death.

Figure 6. The proportion of placentas with presence of the Hyrtl anastomosis among the cases necessitating hysterectomy.

DISCUSSIONS

Placental abruption represents a very severe pathology, even today, and the results of our study are in concordance with this assertion.\textsuperscript{1,2} The fact that the cohort included only patients in which delivery was performed through cesarean section may imply that the only the more severe cases of abruptio placentae were considered. Otherwise it would seem reasonably to consider that milder cases could have been delivered vaginally – or even diagnosed at the morphological inspection of the placenta, after the delivery.\textsuperscript{3} Maternal death is the worse scenario - and, unfortunately, we report two cases. The number of fetal loses are, equally, in the range of that accepted by the international literature – about 25%.\textsuperscript{3,4} The third severity criteria that we considered, the necessity of the removal of the markedly damaged uterus, was also described by others and was relatively frequent in this high severity pathology group.\textsuperscript{5,6} The analysis of this attitude and the different surgical options reported above are beyond the scope of this article.

The morphology of the placentas was similar to the description of other authors.\textsuperscript{7} There could be a rationale for vascular changes in placental abruption, even if there is no consensus on this topic.\textsuperscript{7} The morphological hypothesis was considered, especially as we could use a method to reveal the placental vascular circulation by a method that already proved efficient until very recently, in other vascular territories, in animal experiments.\textsuperscript{8}

Hyrtl anastomosis is probably the most frequently encountered anatomic variant in the morphology of the placental circulation. It represents a transverse connection between the two umbilical arteries, immediately before or just after entering the chorionic plate.\textsuperscript{3,7} This loop is rarely missing – at most up to 15%.\textsuperscript{1,3} It may have some physiological role as to equalize the arterial pressure between the two correspondent arterial territories.\textsuperscript{1}

It is not clear what significance could have the reduced frequency of Hyrtl anastomosis in our group and, hence, in the pathogenesis of placental abruption. It is evident, however, that this inter-arterial connection was far less frequent in our patients overall (28.43%, compared with the “normal” placentas: below 15%). The difference was even bigger when we considered the proportion of its absence in severe cases, which resulted in fetal mortality (39.13%), maternal mortality (one in two cases) or excision surgery (five of nine cases). Of course, fetal mortality could have been caused by other factors as well (e.g. prematurity, distress) but one could speculate that the absence of a “regulation” within the arterial placental circulation could impede helpful flow in a damaged territory or prevent regularization in a pathologically reactive one.\textsuperscript{3}
As all severe cases were noted in hypertensive patients, we could equally consider a possible correlation between the reduced frequency of Hyrtl anastomosis and hypertension or we could think that the absence of this loop in a hypertensive patient may induce placental abruption or may aggravate its severity.

We cannot comment on the vascular disruptions observed at the place of the hematoma in some cases; as we previously stated, results were inconclusive and a firm conclusion could not be stated.

CONCLUSIONS

Placentas retrieved from patients with placental abruption seem to have at least a morphological vascular particularity: the decreased frequency of the Hyrtl anastomosis. This phenomenon is more evident in the most severe cases. Even if it could be difficult to say that this vascular macroscopic placental change represents an etiological factor, it could be a factor of aggravation in abruptio placentae and/or in cases of hypertensive patients.

REFERENCES