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Obstetrics and Gynaecology Section

Images of Placental Aging with Calcification in Pregnancy-induced Hypertension

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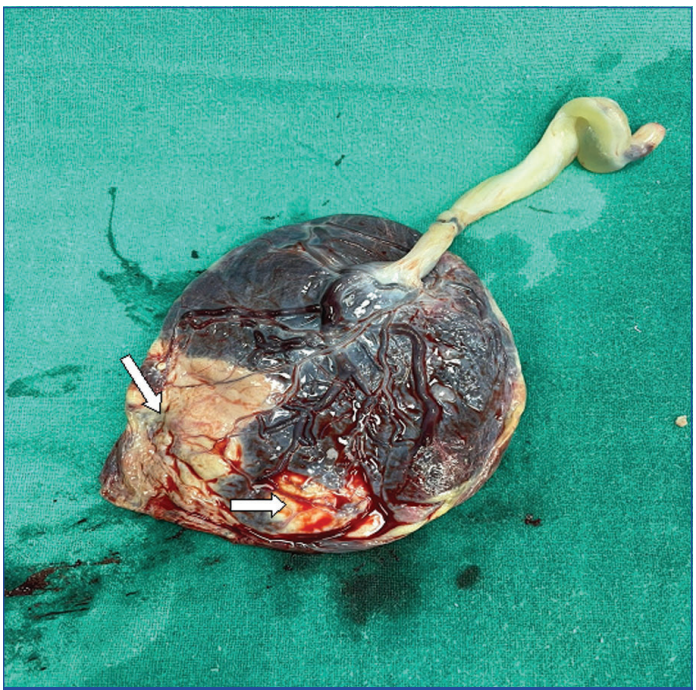
A 28-year-old primigravida with a history of Pregnancy-Induced Hypertension (PIH) diagnosed at 24 weeks of gestation, came to the emergency department at 37 weeks of gestation with the complaint of pain in the abdomen. She had no history of any other comorbidities. Her recent laboratory reports were within normal limits. Her last ultrasound scan, done at 32 weeks, revealed a single live intrauterine gestation with parameters of a 30-week foetus weighing approximately 1.7 kg with adequate liquor and grade 3 placental maturity with no Doppler velocimetry abnormalities. On examination, her Blood Pressure (BP) was 140/90 mmHg. Trial of normal labour was given with continuous intrapartum maternal and foetal well-being assessment. Due to foetal distress, the decision for a caesarean section was taken. A male child of 1.72 kg was delivered, cried immediately after birth, with an APGAR score of 8 at 1 min. Neonatal Intensive Care Unit (NICU) admission for further observation in view of low birth weight was advised.

Following delivery, the placenta was examined in the labour room. Gross inspection revealed a small, firm organ, which is a finding consistent with chronic uteroplacental insufficiency. The most notable feature was the presence of calcifications, evidenced by irregular white deposits distributed along the maternal surface of the placenta [Table/Fig-1].



[Table/Fig-1]: Gross image of placenta showing calcification suggestive of accelerated ageing (white arrows).

The gross pathological findings in this case provide a clear and compelling example of the sequelae of pregnancy-induced hypertension on placental morphology. The observed calcification is not an isolated event but a visible manifestation of a complex pathological cascade [1]. The pathogenesis of Hypertensive Disorders of Pregnancy (HDPs) is initiated by a failure of normal trophoblast invasion into the maternal spiral arteries, leading to reduced uteroplacental perfusion [2]. The gross calcification observed in this case represents dystrophic mineral deposition in areas of cellular damage and necrosis caused by this chronic ischaemic injury [3].

The pathological processes observed in the placenta have long-term implications for the health of both mother and child. HDPs are known to increase the long-term risk of cardiovascular, metabolic, and kidney diseases for both, highlighting the need for ongoing maternal health monitoring and a deeper understanding of the origins of foetal programming [4]. Molecular research into placental dysfunction in preeclampsia is beginning to identify specific pathways, such as the activation of JNK and the release of Senescence-Associated Secretory Phenotype (SASP) proteins, which may represent promising targets for future therapeutic strategies [3,5].

This case serves as a compelling clinical illustration of the direct association between pregnancy-induced hypertension and accelerated placental senescence, which manifests as gross calcification.

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