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HYPERHOMOCYSTEINEMIA AND CHORIONIC VILLOUS CHANGES IN EARLY PREGNANCY LOSS: CLINICAL AND HISTOLOGICAL FINDINGS

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https://doi.org/10.5281/zenodo.23668694

Annotation

To describe homocysteine concentrations and qualitative chorionic-villus findings in women with spontaneous abortion or missed miscarriage. Mean homocysteine was 17.55±3.07 μmol/L and 18.05±2.64 μmol/L in the respective case groups, compared with 5.10±0.80 μmol/L in controls ($p<0.001$ for each case group versus controls). Histology described impaired villous vascularisation, stromal oedema and fibrosis, trophoblastic changes, fibrinoid deposition, and microthrombi or haemorrhage in some specimens. These observational findings do not establish causality or prospective prediction.

Keywords: hyperhomocysteinemia, spontaneous abortion, missed miscarriage, chorionic villi, histopathology

Introduction. Early pregnancy loss has multiple potential causes. Homocysteine, an intermediate in methionine metabolism, has been investigated in relation to endothelial function and placental development. Systematic reviews report associations between elevated homocysteine and selected pregnancy complications, although findings and clinical implications vary [1, 2]. Histology of products of conception can document structural changes in chorionic villi and decidua, but these observations are obtained after pregnancy loss and cannot independently predict an antenatal outcome.

Aim. To describe homocysteine concentrations across clinical groups and summarise qualitative histological findings in chorionic villi and decidual tissue from early reproductive losses.

Materials and methods. The clinical cohort included 50 women with spontaneous abortion before 12 weeks, 50 with missed miscarriage, and 30 clinical controls with physiologically progressing pregnancies. Venous blood was collected on admission. Homocysteine was measured biochemically; the laboratory reference interval was 4.4–13.5 μmol/L, with values above 13.5 μmol/L classified as hyperhomocysteinemia. Histology was performed on abortion specimens from the two case groups; tissue was not collected from healthy pregnancy controls. Samples were fixed in 10% phosphate-buffered formalin for 48 hours, dehydrated, embedded in paraffin, cut at 4–5 μm, stained with haematoxylin and eosin, and examined using a LEICA BIOMED light microscope. Morphology was assessed qualitatively; quantitative morphometry was not performed.

Results. Mean homocysteine was 17.55±3.07 μmol/L in spontaneous abortion and 18.05±2.64 μmol/L in missed miscarriage, compared with 5.10±0.80 μmol/L in controls. Each case group differed from controls at $p<0.001$; the case groups did not differ significantly from each other ($p=0.609$). Hyperhomocysteinemia was recorded in 46/50 (92.0%) spontaneous-abortion cases and 48/50 (96.0%) missed-miscarriage cases; no clinical control exceeded the stated threshold. The control group was younger, but age-adjusted analyses retained the differences versus controls. Histology described impaired villous vascularisation and avascular villi, stromal oedema and fibrosis,

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trophoblastic dystrophic/necrobiotic changes, fibrinoid deposition, and microthrombi or haemorrhage in some specimens. Individual findings were not quantified as prevalence percentages.

Discussion. The biochemical findings show higher homocysteine concentrations in both early-loss groups than in clinical controls in this cohort, consistent with prior studies of homocysteine and recurrent pregnancy loss [1, 2]. However, the observational design cannot establish that elevated homocysteine caused the pregnancy loss, and potential confounders beyond age were not comprehensively adjusted for. Histological findings are descriptive and were observed after the outcome; they cannot establish whether tissue changes preceded or caused the loss. Endothelial dysfunction and angiogenesis were not directly measured with specific biomarkers or experimental methods. Clinical evaluation should follow established pathways rather than relying on homocysteine or post-loss histology in isolation [3, 4].

Conclusion. Women with spontaneous abortion and missed miscarriage had higher mean homocysteine than clinical controls, with hyperhomocysteinemia recorded in 92.0% and 96.0% of the respective case groups. Abortion specimens showed qualitative vascular and stromal abnormalities, trophoblastic changes, and, in some specimens, microthrombi or haemorrhage. The data do not establish causality, and post-loss histology is not a prospective predictor. Further prospective studies with quantitative morphometry and adjustment for relevant confounders are needed.

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