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Original Case Report

Maternal Malperfusion and Placental Necrosis in Sudden Maternal Death: A Forensic and Histopathological Case Analysis of Severe Preeclampsia

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Abstract

Background: Sudden maternal death associated with hypertensive disorders of pregnancy can be difficult to interpret when clinical and autopsy documentation is incomplete. **Objectives:** To describe a sudden maternal death in a woman with clinical and placental findings suggestive of severe preeclampsia and to emphasize the diagnostic value and limitations of placental histopathology. **Methods:** A forensic case of a 29-year-old gravida 3 para 2 woman at 32 weeks of gestation who was found dead at home was reviewed using available clinical records, laboratory findings, toxicology, placental gross examination, and histopathology. **Results:** Two weeks before death, headache, blurred vision, epigastric pain, hypertension, and proteinuria were documented. Laboratory findings included proteinuria +3, hematuria +1, and serum creatinine 2.1 mg/dL. Toxicological screening was negative. The placenta showed pale, firm infarcted areas. Histopathology demonstrated focal villous necrosis, ghost villi, and fibrinoid necrosis of maternal spiral arteries, consistent with maternal vascular malperfusion. Complete maternal organ findings and full exclusion of other major causes of sudden maternal death were unavailable. **Conclusions:** The findings strongly support severe preeclampsia as the underlying maternal condition associated with death; however, the immediate mechanism of death remains undetermined. Placental histopathology is valuable when integrated with complete clinical and forensic evaluation.

Keywords: preeclampsia; sudden maternal death; placental necrosis; forensic pathology; maternal vascular malperfusion

Introduction

Sudden maternal death remains a major global health problem and requires careful clinical and forensic investigation. Hypertensive disorders of pregnancy are among the important direct causes of maternal death worldwide[1]. Preeclampsia is a pregnancy specific hypertensive disorder that can progress rapidly and can involve renal, hepatic, neurological, hematological, cardiopulmonary, and uteroplacental dysfunction. Contemporary international guidance emphasizes that diagnosis and severity assessment should be based on documented blood pressure and organ dysfunction rather than on a single pathological finding[2,3].

The biological basis of preeclampsia involves abnormal placentation, impaired uteroplacental perfusion, placental stress, endothelial dysfunction, and an imbalance of maternal vascular responses[4,5]. A widely used conceptual model describes a placental stage

followed by a maternal clinical stage, while recognizing that the disorder is heterogeneous and influenced by maternal susceptibility[6]. Failure of physiological spiral artery transformation and related decidual vascular lesions can contribute to reduced placental perfusion and ischemic injury[7,8].

Placental examination is therefore particularly valuable in severe disease and in maternal death investigations. Maternal vascular malperfusion is a pattern that may include placental infarction, accelerated villous maturation, distal villous hypoplasia, and decidual arteriopathy, but no single lesion is specific for preeclampsia. In Central Sulawesi, published HTJ research has documented the continuing clinical relevance of preeclampsia and its risk factors[9]. Other HTJ evidence also supports the importance of maternal knowledge and early recognition of hypertension in pregnancy[10].

The urgency of the present case lies in the need to distinguish pathological evidence that supports an underlying hypertensive disorder from evidence that proves the immediate mechanism of sudden death. The submitted record includes a history of hypertension and proteinuria, renal laboratory abnormality, negative toxicology, placental infarction, villous necrosis, and spiral artery fibrinoid change, but does not document a complete maternal organ examination or all major severe feature laboratory values. Antenatal education and recognition of danger signs remain important preventive components of maternal care[11].

This report addresses the question of how placental maternal vascular malperfusion should be interpreted in a sudden maternal death when the available forensic record is incomplete. The objectives are to describe the documented clinical timeline, laboratory findings, toxicology, gross and microscopic placental findings, and relevant differential diagnoses, while separating the underlying maternal condition from the immediate mechanism of death and acknowledging evidence that was not available in the source record.

Materials and Methods

Research Design

This manuscript is a descriptive forensic case report based on the clinical history, available laboratory data, toxicology results, gross post mortem findings, and placental histopathology recorded in the source material. The reporting approach was revised using principles from the CARE case report guideline to improve chronological clarity, diagnostic transparency, and explicit reporting of limitations[12]. No analytic comparison group or inferential research design was used.

Sample

The sample consisted of one deceased pregnant woman, gravida 3 para 2, at 32 weeks of gestation, together with the fetus and placenta examined during the forensic investigation. A sample size calculation was not applicable because this is a single case report. The case was included because the source record contained a clinical history, renal laboratory results, toxicology screening, gross placental findings, and placental histopathology relevant to a suspected hypertensive disorder of pregnancy.

Data Collection Techniques

Data were obtained from the available clinical history, laboratory record, external and post mortem examination documentation, toxicology screening, gross placental examination, and microscopic placental examination. The documented variables included gestational age, obstetric history, symptoms reported two weeks before death, a recorded diagnosis of hypertension and proteinuria, urinalysis findings, serum

creatinine, toxicology results, fetal measurements, gross placental abnormalities, villous injury, and maternal spiral artery changes. Accepted placental pathology terminology was used as a framework for interpretation[13].

The source manuscript did not provide the actual blood pressure values, platelet count, liver enzymes, lactate dehydrogenase, placental weight or dimensions, cord and membrane findings, estimated percentage of infarction, placental sampling protocol, histological stain, objective magnification, calibrated scale bars, or complete gross and microscopic findings for the maternal brain, lungs, heart, liver, and kidneys. These details were not reconstructed or invented. Their absence is stated explicitly because they limit classification of disease severity, standardized placental reporting, and cause of death assessment.

Data Analysis Techniques

Analysis was descriptive and clinicopathological. The documented placental lesions were interpreted within the maternal vascular malperfusion framework and were compared with recognized placental pathology terminology. The clinical timeline, laboratory findings, toxicology, and pathology were then integrated to assess the strength of evidence for severe preeclampsia as the underlying maternal condition. Major competing causes of sudden maternal death were considered according to whether the source record contained sufficient evidence to support or exclude them. No inferential statistical analysis was performed.

Etical Consideration

The source manuscript does not provide an ethics committee approval number, a formal ethics waiver, documentation of the legal authority for the forensic autopsy, or documentation of consent for publication from the next of kin. Ethical feasibility for publication of this post mortem case therefore depends on confirmation by the authors that the forensic examination was performed under the applicable legal authority and that publication is covered by institutional approval, exemption, waiver, or next of kin consent as required by local regulations and journal policy. Confidentiality was protected in the revised text by limiting personal information to details necessary for clinical interpretation. No ethics number, waiver, or consent statement has been fabricated.

Case Description

A 29 year old woman, gravida 3 para 2, was 32 weeks pregnant at the time of death. Approximately two weeks before death, the available clinical history recorded headache, blurred vision, and epigastric pain, together with a diagnosis of hypertension and proteinuria. The actual blood pressure value from that encounter was not available in the submitted record. The source manuscript reported no history of trauma or known comorbidity. She was later found dead at home. No symptoms immediately preceding the terminal collapse were documented in the available materials.

This chronology is compatible with a hypertensive disorder of pregnancy and possible severe features, but the absence of the original blood pressure values and several laboratory parameters limits retrospective severity classification before the post mortem findings are considered.

Result

External examination documented abdominal enlargement with a fundal height of 30 cm, livor mortis, and rigor mortis without reported signs of violence. A male fetus weighed 2000 g and measured 50 cm, with maceration and generalized meconium staining. Gross placental examination showed multiple pale, firm areas interpreted as infarction, as illustrated in Figure 1A. Figure 1B shows fetal

fingernail discoloration; this is a nonspecific post mortem observation and was not used as evidence for the maternal cause of death.

Microscopic examination of placental tissue showed focal villous necrosis and villi with loss of nuclear detail while basic villous outlines remained recognizable. These so called ghost villi were interpreted as morphological evidence of advanced ischemic or infarcted tissue rather than as a lesion specific to preeclampsia. Neutrophils were described near the periphery of an infarct in the available figure material. The stain, objective magnification, and scale calibration were not documented in the submitted record.

Fibrinoid necrosis was reported in maternal spiral arteries. This finding is compatible with a component of decidual arteriopathy, but the source record did not document the complete spectrum of vascular changes required for a more detailed classification. Perivascular lymphohistiocytic infiltration was also described. This inflammatory finding was treated separately and was not classified as a defining feature of maternal vascular malperfusion.

Taken together, the placental findings were compatible with maternal vascular malperfusion and ischemic placental injury. The source record did not provide placental weight, dimensions, cord or membrane findings, the proportion and distribution of infarction, or a standardized sampling description; therefore, the severity and completeness of the placental lesion pattern could not be quantified.

Available ante mortem laboratory data showed proteinuria of +3, hematuria of +1, and serum creatinine of 2.1 mg/dL, supporting renal involvement. Toxicological screening after death was reported as negative for alcohol, narcotics, and other tested toxic agents. The manuscript did not provide actual blood pressure values, platelet count, liver enzymes, lactate dehydrogenase, or other severe feature measurements. It also did not provide sufficient gross or microscopic findings from the brain, lungs, heart, liver, and kidneys to verify the multiorgan injury statement used in the earlier abstract. Accordingly, the revised report does not claim specific maternal organ lesions that were not documented.

The available documentation was also insufficient to demonstrate complete exclusion of pulmonary embolism, amniotic fluid embolism, intracranial hemorrhage, peripartum cardiomyopathy, eclampsia, sepsis, or another acute event. No witnessed convulsion was reported, but absence of a witnessed seizure does not exclude eclampsia. Because the immediate terminal mechanism was not documented, the most defensible interpretation is that the available clinical and placental findings strongly support severe preeclampsia as the underlying maternal condition associated with death, while the immediate mechanism remains undetermined.

Discussion

The central interpretive issue in this case is the distinction between evidence for an underlying hypertensive disorder and proof of the immediate cause of death. The placental pattern is consistent with maternal vascular malperfusion, a recognized manifestation of abnormal uteroplacental blood flow, but maternal vascular malperfusion is not unique to preeclampsia and should be interpreted in clinical context[14,15]. In this case, the combination of a recent history of hypertension and proteinuria, renal dysfunction, and ischemic placental injury substantially strengthens the association with preeclampsia.

Placental ischemic injury develops when maternal blood flow to the intervillous space is reduced or abnormal. Severe malperfusion can be accompanied by infarction, villous injury, altered maturation, and reduced functional exchange surface. Similar pathways are

described in placental disorders associated with fetal growth restriction[16]. The fetal findings in the submitted record, however, were not accompanied by a complete growth assessment or placental weight, so the revised manuscript does not infer fetal growth restriction from the available measurements alone.

The reported fibrinoid necrosis of maternal spiral arteries is relevant because decidual arteriopathy is associated with hypertensive disorders of pregnancy. Current pathological literature emphasizes that vascular changes must be described precisely and separated from villous lesions and inflammatory lesions[17]. The earlier manuscript treated perivascular lymphohistiocytic inflammation as an inflammatory dimension of maternal vascular malperfusion. That wording has been moderated because chronic inflammatory infiltrates are not interchangeable with the standard maternal vascular malperfusion lesion set.

The clinical evidence also requires careful qualification. Modern diagnostic criteria for preeclampsia rely on documented hypertension after 20 weeks of gestation together with proteinuria or other maternal organ or uteroplacental dysfunction. Renal impairment is a recognized severe feature, but interpretation depends on the full clinical record[18]. The creatinine value of 2.1 mg/dL and proteinuria of +3 are concerning, yet the actual blood pressure, platelet count, liver enzymes, and lactate dehydrogenase were not available. The revised report therefore avoids recreating a severity profile that the source data cannot support.

Forensic attribution of sudden maternal death requires consideration of other major pregnancy related causes. Population based mortality analyses show that cardiovascular disease, hemorrhage, hypertensive disorders, infection, thromboembolism, and other conditions can contribute to pregnancy related death[19]. Pulmonary embolism is an important differential diagnosis in sudden collapse during pregnancy and requires appropriate anatomic or imaging evidence for exclusion[20]. The submitted manuscript did not provide a documented pulmonary vascular examination sufficient to state that pulmonary embolism had been excluded.

Amniotic fluid embolism is another cause of sudden maternal cardiorespiratory collapse, although it is primarily a clinical diagnosis and cannot be established or excluded by a single nonspecific pathological finding[21]. The available record does not describe the circumstances of the terminal collapse, coagulopathy, cardiopulmonary findings, or targeted ancillary investigation needed for a reasoned assessment of this differential. The revised discussion therefore identifies it as unassessed rather than excluded.

Peripartum cardiomyopathy and other cardiac disorders must also be considered in sudden maternal death. Peripartum cardiomyopathy may present with heart failure, arrhythmia, thromboembolism, or sudden deterioration, and diagnosis requires cardiac assessment and exclusion of other causes[22]. No complete gross or microscopic cardiac findings were provided in the source manuscript. Consequently, the present report cannot use the placenta alone to exclude a concurrent or alternative cardiac mechanism.

The pathophysiology of preeclampsia provides a coherent explanation for the clinical and placental observations. Placental stress can promote a systemic maternal inflammatory response, endothelial dysfunction, and widespread vascular consequences[23]. Antiangiogenic signaling is also central to the maternal syndrome and can contribute to hypertension, renal endothelial injury, and other organ dysfunction[24,25]. These mechanisms make severe preeclampsia biologically plausible in the present case, but

mechanistic plausibility does not replace direct documentation of the terminal event.

The renal findings are among the strongest maternal clinical data that were actually available. Proteinuria of +3 and creatinine of 2.1 mg/dL support significant renal involvement in a woman with a recent diagnosis of hypertension and proteinuria. Nevertheless, the absence of serial measurements, baseline renal function, and complete severe feature laboratory testing restricts the certainty with which the clinical stage can be reconstructed. This limitation is particularly important in a forensic report, where the level of diagnostic certainty should be proportional to the completeness of the record.

The placental findings also require standardized reporting. The Amsterdam consensus recommends systematic gross examination, defined sampling, and precise terminology for maternal vascular malperfusion, fetal vascular malperfusion, inflammatory lesions, and villous maturation abnormalities[13]. The submitted material did not provide placental weight, dimensions, cord insertion and vessel number, membrane description, infarct percentage, sampling protocol, stain, or scale information. These omissions should be corrected from the original pathology record if those data are available before publication.

The microscopic image demonstrates infarcted or ischemic placental tissue with villous nuclear loss and inflammatory cells near the infarct margin. These morphological findings support tissue injury but are not individually specific for preeclampsia. The figure caption has therefore been revised to identify what the arrows show without treating ghost villi or neutrophils as pathognomonic. Likewise, the fetal fingernail discoloration in Figure 1B is explicitly described as nonspecific and is not used in the cause of death reasoning.

The distinction between underlying disease and immediate mechanism is crucial. Based on the available record, severe preeclampsia is strongly supported as the underlying maternal condition because the clinical history, renal abnormality, and placental malperfusion pattern converge. The immediate mechanism could have involved neurological, cardiopulmonary, thrombotic, or other complications, but the source manuscript does not provide enough evidence to select one. This cautious formulation is more appropriate than stating that placental lesions alone definitively establish the primary cause of death.

The case also has implications beyond the forensic diagnosis. Preeclampsia is associated with later maternal cardiovascular risk, and systematic evidence supports long term cardiovascular surveillance for survivors of hypertensive pregnancy disorders[26]. More recent evidence also shows that adverse maternal and perinatal outcomes in preeclampsia remain heterogeneous and difficult to predict accurately, reinforcing the importance of comprehensive clinical assessment rather than reliance on one marker[27]. Although the present patient died, these broader data underscore why early recognition and structured follow up are important in routine obstetric practice.

From a prevention perspective, antenatal surveillance should emphasize recognition of headache, visual symptoms, epigastric or right upper quadrant pain, hypertension, proteinuria, and signs of maternal organ dysfunction. Standard obstetric references emphasize prompt assessment of severe symptoms and appropriate management when preeclampsia is suspected[28]. The local HTJ literature cited earlier also supports strengthening maternal knowledge, risk recognition, and antenatal education[9,10,11].

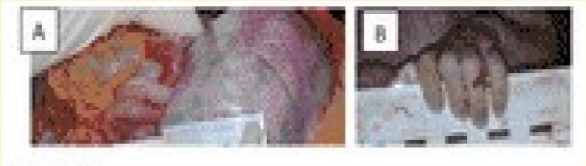

Figure 1. Gross Macroscopic Findings in Autopsy. (A) Placenta showing pale, firm regions interpreted grossly as infarcted tissue. (B) Fetal fingernail discoloration. Panel B is a nonspecific post mortem observation and was not used to determine the maternal cause of death.

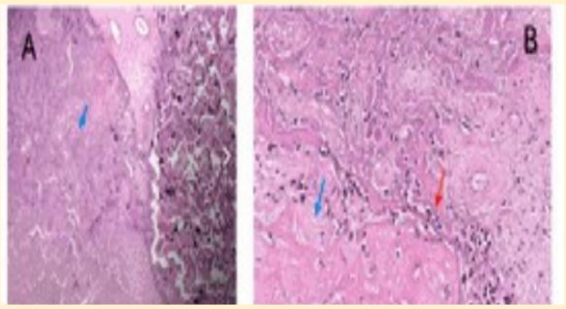

Figure 2. Microscopic Findings of Placenta. (A) Focal villous necrosis indicated by the blue arrow. (B) Inflammatory cells, including neutrophils, at the margin of infarcted tissue indicated by the red arrow, with villi showing loss of nuclear detail indicated by the blue arrow. These changes are morphological features of ischemic and infarcted tissue. The histological stain, objective magnification, and calibrated scale bars were not documented in the submitted source material and should be confirmed from the pathology record before publication.

The principal strengths of this report are the documented prodromal symptom history, renal laboratory abnormalities, negative toxicology, gross placental infarction, and placental histopathology. Its main limitations are the absence of the actual blood pressure values, incomplete severe feature laboratory data, incomplete maternal organ autopsy reporting, incomplete standardized placental measurements and sampling information, and absent documentation of ethics approval or waiver and publication consent. These limitations preclude a definitive statement about the immediate mechanism of death and should remain visible to the reader rather than being concealed by stronger language.

Conclusion

The available clinical, laboratory, toxicological, gross placental, and histopathological findings strongly support severe preeclampsia as the underlying maternal condition associated with this sudden death. Maternal vascular malperfusion, placental infarction, villous necrosis, and fibrinoid necrosis of maternal spiral arteries provide important clinicopathological support when interpreted together with the documented hypertension, proteinuria, and renal impairment. However, the immediate mechanism of death cannot be established from the submitted record because complete maternal organ findings and exclusion of major competing causes were not documented. Placental lesions should therefore be used as contributory forensic evidence rather than as isolated proof of the terminal mechanism.

The case supports strengthening antenatal surveillance, timely recognition of maternal danger signs, and complete documentation of severe features. For future forensic reports, standardized placental examination, full maternal organ assessment, clear differential diagnosis, complete figure documentation, and explicit ethics and publication consent information are essential for a defensible cause of death interpretation.

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