

Sinusoidal pattern, the anemia's silent alarm: a case report

Padrão sinusoidal: um sinal silencioso de anemia – relato de caso

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Abstract

A sinusoidal fetal heart rate (SHR) pattern is a rare but severe indicator of fetal distress. We report a case of a 29-year-old primigravida at 35 weeks with reduced fetal movements and SHR on cardiotocography. An emergent cesarean section revealed a newborn with pallor, bruising, pancytopenia, and coagulopathy. Common causes were excluded. Maternal anti-HLA antibodies confirmed alloimmune thrombocytopenia and neutropenia, leading to fetal bone marrow suppression. This case highlights a rare mechanism of fetal anemia, expanding the differential diagnosis of SHR beyond fetomaternal hemorrhage to include alloimmune bone marrow suppression. Immediate recognition of SHR remains essential.

Keywords: Sinusoidal fetal heart rate; Fetal anemia; Neonatal alloimmune thrombocytopenia; Fetal distress; Case report.

Resumo

O padrão sinusoidal (SHR) é um sinal raro, mas grave, de sofrimento fetal. Apresentamos o caso de uma primigesta de 29 anos, 35 semanas de gestação, com diminuição dos movimentos fetais e CTG com padrão sinusoidal na cardiotocografia. Foi decidida cesariana emergente que revelou um recém-nascido com palidez, equimoses, pancitopenia e coagulopatia. A hemorragia fetomaternal e outras causas mais comuns foram excluídas. A presença de anticorpos maternos anti-HLA confirmou trombocitopenia e neutropenia aloimunes, com conseqüente supressão medular fetal. Este caso evidencia um mecanismo raro de anemia fetal, alargando o diagnóstico diferencial do SHR. O reconhecimento precoce do SHR é fundamental.

Palavras-chave: Padrão sinusoidal; Anemia fetal; Trombocitopenia neonatal aloimmune; Sofrimento fetal; Relato de caso.

BACKGROUND

A sinusoidal fetal heart rate pattern (SHR) manifests as a regular, smooth, undulating signal resembling a sine wave, with an amplitude of 5-15 bpm and a frequency of 3-5 cycles per minute¹. Although its precise pathophysiology remains unclear, it is strongly associated with severe fetal anemia^{1,2}. SHR is considered an

ominous sign of fetal jeopardy, often requiring immediate intervention². While fetomaternal hemorrhage is a well-established cause, other mechanisms, such as alloimmune processes remain underreported. This case highlights a rare etiology of SHR, expanding current knowledge on its implications.

CASE PRESENTATION

A 29-year-old healthy primigravida, A Rh D+, presented at 35 weeks of gestation due to reduced fetal

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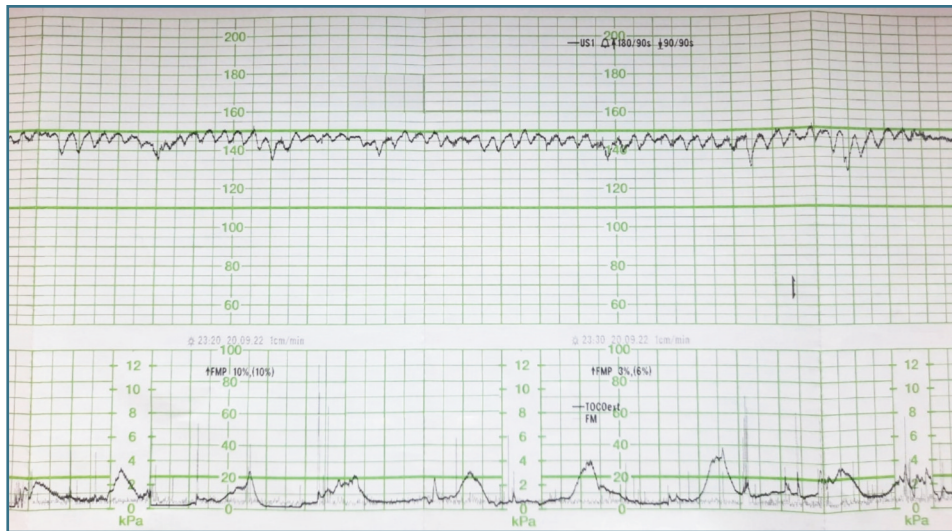


FIGURE 1. Cardiotocographic tracing showing sinusoidal pattern and regular uterine contractility.

movements. Her pregnancy had been uneventful, and an indirect Coombs test at 26 weeks was negative. She and her partner were not blood-related.

She reported no additional complaints and denied blood or amniotic fluid loss. An ultrasound examination showed a fetal heart rate of 137 bpm and an amniotic fluid index of 27 cm, but no active fetal movements were observed.

Cardiotocography (CTG) revealed a low variability tracing with a sinusoidal pattern and regular uterine contractility (Figure 1). Suspecting fetal distress, an urgent cesarean section was performed. A male newborn, weighing 3030 g, scored 7/5/4 on the Apgar score and exhibited marked pallor and scattered bruises at birth. Umbilical cord blood gas analysis 50 min after birth revealed pH 6.99, HCO_3^- 8.0 mmol/L, base excess 23.4 mmol/L, pCO_2 33 mmHg, pO_2 53 mmHg, lactate <15 mmol/L and hematocrit <15%, consistent with metabolic acidosis.

Posterior blood analysis revealed severe pancytopenia (hemoglobin 2.1 g/dL, leukocytes 1900/ μL , platelets $5 \times 10^3/\mu\text{L}$) and coagulopathy (prothrombin time 30.8 s, activated partial thromboplastin time 58.4s, INR 2.54, fibrinogen 241 mg/dL). The neonate was transferred to the neonatal intensive care unit and received transfusions of plasma, red blood cells, and platelets.

Flow cytometry ruled out fetomaternal hemorrhage (anti-hemoglobin F <0.01%). Placental histopathology

showed no anomalies. Further investigations aimed to determine the cause of pancytopenia.

No malformations were detected, and infectious, immunological, and metabolic studies yielded negative results. Bone marrow aspiration revealed reduced megakaryocytes and erythroid cells. A genetic panel excluded congenital medullary failure syndromes, mitochondrial DNA analysis showed no deletions, and chromosomal breakage tests ruled out Fanconi anemia. MPL gene analysis was normal.

The human platelet antigen (HPA) test detected maternal anti-HLA class I alloantibodies, confirming neonatal alloimmune thrombocytopenia and neutropenia. The resulting bone marrow suppression contributed to fetal anemia and distress.

The newborn required multiple platelet transfusions in the first month of life. At nine months, the baby exhibited normal development with no need for further transfusions.

CONCLUSIONS

This case reinforces the critical nature of SHR as a marker of fetal jeopardy requiring urgent intervention³. SHR is widely documented in association with fetal anemia, most often linked to fetomaternal hemorrhage². The pathophysiological mechanism linking

sinusoidal fetal heart rate patterns to anemia is thought to involve a profound reduction in oxygen transport capacity, leading to chronic hypoxemia and disruption of autonomic regulation of fetal heart rate variability⁴. In our case, alloimmune-mediated bone marrow suppression produced severe anemia, likely triggering this mechanism.

SHR identification remains challenging and necessitates trained cardiotocographic analysis. While fetomaternal hemorrhage is the most recognized cause, clinicians should consider alloimmune mechanisms in cases where traditional causes are absent^{5,6}.

This case underscores the importance of maternal-fetal immunological interactions in perinatal care and broadens the spectrum of conditions associated with SHR.

The main strength of this report lies in its contribution to the understanding of rare causes of fetal anemia presenting as SHR. However, as a single case report, the findings cannot be generalized, and immunological confirmation was limited by the availability of specific laboratory tests. Further studies are needed to better characterize these alloimmune interactions.

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DECLARATIONS

Ethics approval and consent to participate. Ethical approval was waived as this is a retrospective case report with no interventions beyond standard clinical care. Informed consent for participation and publication was obtained from the patient.

CONSENT FOR PUBLICATION

Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

COMPETING INTERESTS

The authors declare that they have no competing interests.

AUTHORS' CONTRIBUTIONS

Fabiana Castro conceptualized and designed the study, collected and analyzed the data, and wrote the manuscript. António de Pinho assisted in data collection and contributed to manuscript writing. Andreia Mota Sousa assisted in data collection and contributed to manuscript writing. Cristina Oliveira contributed to data collection and interpretation and provided critical revisions to the manuscript. Cristina Carrapatoso contributed to manuscript revision.

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