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Case Series

A case series of septic infants with cerebral venous sinus thrombosis: novelty neonatal thromboembolism risk stratification model in the Grodno region

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ABSTRACT

Neonatal thromboembolism, especially cerebral venous sinus thrombosis (CVST), is a significantly recognized complication in critically ill infants. Early detection protocols remained challenging due to variable clinical presentation and lack of standard predictive models. This study aims to develop a novelty risk stratification model composed of detailed analysis from four cases. A retrospective case series evaluation was conducted on four individual cases of neonates with severe sepsis complicated by transverse sinus thrombosis confirmed radiologically. Clinical characteristics, complete blood count (CBC), coagulation parameters, biochemical markers, and inflammatory biomarkers were longitudinally assessed. A novelty neonatal thromboembolism risk score (NTRS) was derived based on persistent laboratory findings. All four patients exhibited a specific prothrombotic profile characterized by leukemoid reaction (>50×10⁹/l), thrombocytopenia (<150×10⁹/l), significantly increased D-dimer (>10,000 ng/ml), hypofibrinogenemia (<1.5 g/l), increased inflammatory markers (procalcitonin >5 ng/ml, CRP >20 mg/l), and evidence showing multiorgan dysfunction. The developed novelty NTRS (range 0–16) successfully graded all four cases into one low, two high and one critical risk categories before the confirmation of thrombosis was found. This novelty NTRS model characterizes a practical, laboratory-driven approach for early identification of neonates at risk for thromboembolism. Prospective larger cohorts are necessary to validate these findings.

Keywords: Neonatal thrombosis, Cerebral venous sinus thrombosis, Sepsis, Biomarkers, Risk model

INTRODUCTION

Cerebral venous sinus thrombosis (CVST) in neonates represents a life-threatening under-recognized neurovascular coagulative condition presented by the formation of thrombi within the dural venous sinuses leading to vessel obstruction.[1] The estimated annual incidence of CVST is approximately at 2.6 to 2.69 per 100,000 newborns per year.[2] While, the prevalence of this condition is comparatively low in comparison to other neonatal complications, CVST carries substantial morbidity and mortality risks due to its nature of variable clinical manifestations and possibility to cause severe neurological consequences such as cerebral ischemia, cerebral hemorrhage, and subsequent long-term neurodevelopmental limitations.[3,4] The main diagnostic challenge is the presence of delicate and variable presentation in neonates, demanding increased clinical awareness and timely diagnostic evaluation for better prognosis.

Neonatal sepsis is a substantial potent risk factor for thromboembolic events mainly for CVST. The systemic inflammatory response initiated by sepsis triggers complex

hemostatic and inflammatory pathways, creating a hypercoagulable state that influences neonates to present with venous thrombosis. This aligns with Virchow's triad, involving endothelial injury, altered blood flow, and hypercoagulability.[5-7] This pathophysiological interaction highlights the significance of diagnosing sepsis not only as an infectious threat but also the subsequent contribution to vascular complications in this susceptible population. In addition, even with the development in neonatal intensive care and antimicrobial therapy, the incidence of CVST associated with sepsis remains a significant factor to severe neonatal outcomes, highlighting the need for improved preventive and therapeutic management.

In the Grodno region, the epidemiology of neonatal CVST in the background of sepsis is affected by regional factors such as variations in healthcare infrastructure, genetic predispositions, environmental exposures, and socioeconomic factors. These factors jointly influence the incidence, clinical course, and patient prognosis following a CVST, indicating the insufficiency of risk assessment techniques implemented in Grodno. Currently, the risk stratification model adapted to the Grodno region that utilize local epidemiological data and clinical parameters. Therefore, developing such a model is essential to facilitate the early identification of high-risk neonates, improve diagnostic pathways, and provide therapeutic interventions effectively. A region-specific model would allow targeted resource utilization and improve prognostic accuracy, ultimately improving neonatal care quality and prognosis following a CVST in Grodno region.

The primary aim of this case series is to systematically describe neonatal CVST presenting in the background of sepsis within the Grodno region, thereby clarifying its clinical spectrum, laboratory and imaging features, and neurological outcomes. This study mainly explores to fill the current knowledge gap by providing detailed regional data that mirror the unique patient population and healthcare patterns.

Specific objectives such as the identification and analysis of key clinical signs, laboratory markers, and neuroimaging data that influence the prediction of CVST in septic neonates. These factors will be systematically examined to develop a practical and evidence-based risk stratification model crafted particularly to the Grodno region. This model's purpose is to improve early risk assessment, allowing clinicians to stratify patients according to their probability of developing CVST and to craft management strategies accordingly.

The novelty of this study depends on the targeted approach for this localized population with detailed clinical and diagnostic data, which may disclose definite risk factors and pathophysiological mechanisms not caught by current generalized models. The expected effect consists of improved early recognition of CVST in neonates with sepsis, timely initiation of appropriate management, and the development of a foundation for prospective studies. In conclusion, the study aims to contribute to reduce the morbidity and mortality associated with neonatal CVST in the background of neonatal sepsis in the Grodno region and possibly serving as a model for other regions with similar healthcare systems in Belarus.

CASE SERIES

This study is a retrospective case series was conducted using clinical data from neonates hospitalized to neonatal intensive care units in the Grodno Regional Children's Clinical Hospital Belarus. The patient selection criteria included: four neonates with confirmed neonatal sepsis and diagnosed with cerebral venous sinus thrombosis confirmed radiologically (via neurosonography with Doppler). The data collection included extraction of clinical and laboratory data such as hematological parameters from complete blood count profiles (leukocyte count and differential, platelet count, hemoglobin and hematocrit), coagulation profile (prothrombin time (PT), international normalized ratio (INR), activated partial thromboplastin time (aPTT), fibrinogen, D-dimer), biochemical parameters from biochemical profiles (renal function (urea, creatinine), liver enzymes (AST, ALT, bilirubin), lactate dehydrogenase (LDH), electrolytes), inflammatory biomarkers from biochemical profiles (C-reactive protein (CRP), procalcitonin (PCT)), cardiovascular biomarkers (proBNP, troponin) and clinical variables mainly mechanical ventilation, vasopressor support, presence of central venous catheter.

Furthermore, a scoring system (NTRS) was constructed, namely neonatal thromboembolism risk stratification (NTRS) model based on the following selection criteria such as frequency of abnormalities across cases, presence of biological plausibility (Virchow's triad) and clinical relevance among the cases. In addition, each parameter was assigned weighted points.

No statistical analysis was implemented in this case series, since there was no quantitative and qualitative data analysis present in this study to generate a scoring system.

The study was performed in under the guidance of good clinical practice standards and the principles of the declaration of Helsinki. The written informed consent was collected from all participants before inclusion to this study.

This study consisted of four individual cases of neonates who were term or near-term infants. All four cases presented with early-onset severe sepsis and subsequently developed respiratory failure requiring mechanical ventilation. The presenting hemodynamic instability required vasopressor support along with the development of multiorgan dysfunction. The presenting CVST were in the transverse sinuses in all four cases. Therefore, the common risk factors detected consisted of severe systemic inflammation, hematological abnormalities, coagulation disturbances, and critical organ dysfunction.

The NTRS was constructed to classify the risk of thromboembolism in neonates with clinical and laboratory parameters connected with CVST. Variables present in this model were grouped into five groups: inflammation (procalcitonin >10 ng/ml, CRP >50 mg/l), hematologic abnormalities (platelets <100×10⁹/l, leukocytes >50×10⁹/l), coagulation disturbances (D-dimer >5000 ng/ml, fibrinogen <1.5 g/l, INR >1.5), organ dysfunction (lactate >3 mmol/l, renal dysfunction, LDH >1000 U/l), and clinical factors (vasopressor use, mechanical ventilation, presence of central venous catheter). Each parameter was allocated points based on its planned contribution to thromboembolism risk, with a total score starting from 0 to 16.

Risk categories were classified as low (0–4), moderate (5–8), high (9–12), and critical (≥13). Furthermore, the application of this model to these four cases showed its predictive ability: case 1 scored between 14 and 16, placing the neonate in the critical risk category, supporting confirmed thrombosis. case 2 scored between 9 and 11, classified as high risk, also supported confirmed thrombosis. Thus, this model's predictive accuracy was supported by these findings, indicating its potential value in early identification and classification of thromboembolism risk in neonates. Although, these results highlighted the efficacy of NTRS to differentiate risk levels, further studies with larger cohorts are necessary to validate this study.

Table 1: Neonatal thromboembolism risk score scoring system.

Group	Parameter	Points
Inflammation	PCT >10 ng/ml	2
	CRP >50 mg/l	1
Hematologic	Platelets <100×10⁹/l	2
	Leukocytes >50×10⁹/l	1
Coagulation	D-dimer >5000 ng/ml	2
	Fibrinogen <1.5 g/l	1
	INR >1.5	1
Organ dysfunction	Lactate >3 mmol/l	1
	Renal dysfunction	1
	LDH >1000 U/l	1
Clinical	Vasopressors	1
	Mechanical ventilation	1
	Central venous catheter	1

Total score: 0–16; PCT – procalcitonin, CRP – C-reactive protein, INR – international normalized ratio, LDH – lactate dehydrogenase

Table 2: Risk stratification.

Score	Risk category
0–4	Low
5–8	Moderate
9–12	High
≥13	Critical

Application to cases

Case 1

Score ≈ 14-16 → critical risk → confirmed thrombosis.

A female neonate born at 38 weeks' gestation following spontaneous vaginal delivery (with a birth weight of 3150 g and Apgar scores of 8/8), presented with early clinical deterioration within the first hours of life demonstrating progressive respiratory distress demanding mechanical ventilation, followed by hemodynamic instability requiring vasoactive support (dopamine, adrenaline, norepinephrine). The clinical course developed into multiorgan dysfunction syndrome (MODS) with features of septic shock, pulmonary hemorrhage, and disseminated intravascular coagulation (DIC). The diagnosis of neonatal sepsis was confirmed by the following clinical and laboratory parameters such as prominent leukocytosis up to 67×10⁹/l with left shift, elevated inflammatory markers (procalcitonin up to 100 ng/ml, C-reactive protein up to 123 mg/l) and clinical deterioration with shock and organ dysfunction. Therefore, the final diagnosis was unspecified bacterial neonatal sepsis. Coagulation Abnormalities were observed from the laboratory findings being consistent with coagulopathy (INR up to 1.55, fibrinogen 1.0 g/l, D-dimer 15,600 ng/ml). Thus, these findings tally with DIC, thereby being a known risk factor for thrombosis.

The Instrumental confirmation of Thrombosis was demonstrated by neuroimaging such as neurosonography revealing transverse sinus thrombosis with reduced cerebral venous flow and doppler imaging confirmed this diagnosis of thrombosis of the transverse sinuses of the brain by revealing reduction of blood flow in affected sinuses.

Furthermore, this patient was managed by broad-spectrum antibiotics, intensive respiratory and hemodynamic support, blood transfusions and anticoagulation (Fragmin). In addition, the follow-up imaging revealed complete recanalization, and the patient was discharged in a satisfactory condition.

Case 2

Score ≈ 9-11 → high risk → confirmed thrombosis.

A male neonate born at 37 weeks following a cesarean section with a birth weight of 2880 g, presenting with clinical deterioration in the early neonatal period.

The infant needed mechanical ventilation and intensive care for septic shock, respiratory failure, and renal dysfunction. In addition, the clinical picture was complicated by DIC and neurological involvement. The diagnosis of neonatal sepsis was confirmed on clinical signs of infection and shock, laboratory evidence such as elevated liver enzymes (AST 134 U/l, ALT 240 U/l),

anemia and inflammatory response. Overall, the final diagnosis was neonatal sepsis of unspecified etiology.

The final concomitant diagnosis of left transverse sinus thrombosis was confirmed by imaging mainly the neurosonography findings revealing left transverse venous sinus thrombosis and additional findings such subependymal pseudocyst, preserved arterial flow parameters.

Additionally, this patient was managed by broad-spectrum antimicrobial therapy, intensive care support and anticoagulation therapy (Dalteparin sodium).

Furthermore, clinical improvement was observed with progressive stabilization and recovery, and anticoagulation was terminated after confirmed recanalization of the affected sinus with imaging.

Case 3

Score ≈ 11-14 → critical risk → confirmed thrombosis.

A male neonate born at 39 weeks with a birth weight of 3080 g was admitted at 17 hours of life with congenital sepsis, purulent meningoencephalitis, and multiple organ dysfunction.

Th infant required mechanical ventilation, vasopressors, utilization of the central venous catheter during intensive care to manage the presenting congenital sepsis, purulent meningoencephalitis, and multiple organ dysfunction. Moreover, this clinical picture was complicated by severe neurological impairment, particularly the clinical sign such as spastic tetraparesis present on the left side of the body, seizure syndrome with grand mal seizures, and chronic ventriculitis. The diagnosis of neonatal sepsis with organ dysfunction was confirmed by clinical signs of infection (congenital pneumonia) and laboratory parameters such as elevated lactate levels (>3 mmol/l), elevated LDH levels (>1000 U/l), elevated inflammatory markers (procalcitonin >10 ng/ml, CRP >50 mg/l), prominent leukocytosis (>50×10^9/l) and renal dysfunction. Coagulation abnormalities were observed from the laboratory findings being consistent with underlying coagulopathy (platelets <100×10^9/l, INR >1.5 and fibrinogen <1.5 g/l), a known risk factor for thrombosis formation. These findings led to confirmed diagnosis of congenital sepsis.

The findings from imaging modalities such as neurosonography confirmed thrombosis of venous sinuses, extensive brain damage with cystic encephalomalacia, and hydrocephalus.

Furthermore, this patient was managed with phenobarbital (manage grand mal seizures), broad-spectrum antimicrobial therapy, intensive care support and anticoagulation therapy (Dalteparin sodium).

Overall, this patient's clinical status was significantly improved with progressive stabilization and recovery, the anticoagulation was continued until the recanalization of the affected sinus was observed by imaging.

Case 4

Score ≈ 3 → low risk → confirmed thrombosis.

A male neonate born at 37 weeks (260 days), 2800 g, was admitted at an age of 10 days old with unspecified neonatal sepsis, respiratory distress syndrome, mild anemia, and mixed genesis neonatal encephalopathy with suppression syndrome.

The infant needed mechanical ventilation, vasopressors, central venous catheter during intensive care for neonatal sepsis, respiratory distress syndrome, mild anemia, and mixed genesis neonatal encephalopathy with suppression syndrome. The initial clinical course of this neonate consisted of septic shock and nephropathy, later stabilized prior to this hospital visit in another city hospital. In addition, the laboratory parameters showed mild elevation of inflammatory markers and hematologic changes with coagulation and organ dysfunction markers within or near normal limits.

The imaging confirmation of thrombosis of the right transverse sinus was facilitated by neurosonography findings.

Additionally, this patient received broad-spectrum antimicrobial therapy (Meropenem), intensive care support, Folic acid (to stimulate erythropoiesis) and anticoagulation therapy (Dalteparin sodium). And this patient was monitored closely with planned visits for new neurological symptoms in the following upcoming months.

These findings facilitate the predictive accuracy of the NTRS in early identification and risk stratification of neonatal thromboembolism.

DISCUSSION

This study discovered a consistent laboratory signature associated with neonatal thromboembolism and developed a structured risk stratification model, the NTRS shown in Figure 1. These findings offered pathophysiological insights secondary to sepsis-driven thrombotic mechanism categorized by endothelial injury, hypercoagulability, and circulatory stasis which are the components that jointly mirror Virchow's triad in the neonatal setting.[8-10] Raised inflammatory cytokines and bacterial toxins seems to increase the endothelial damage, whereas laboratory markers such as increased D-dimer levels and developing DIC reveal a hypercoagulable state. Circulatory stasis develops secondary to shock, cardiac dysfunction, and mechanical ventilation, additional encouraging thrombus formation.[11-13]

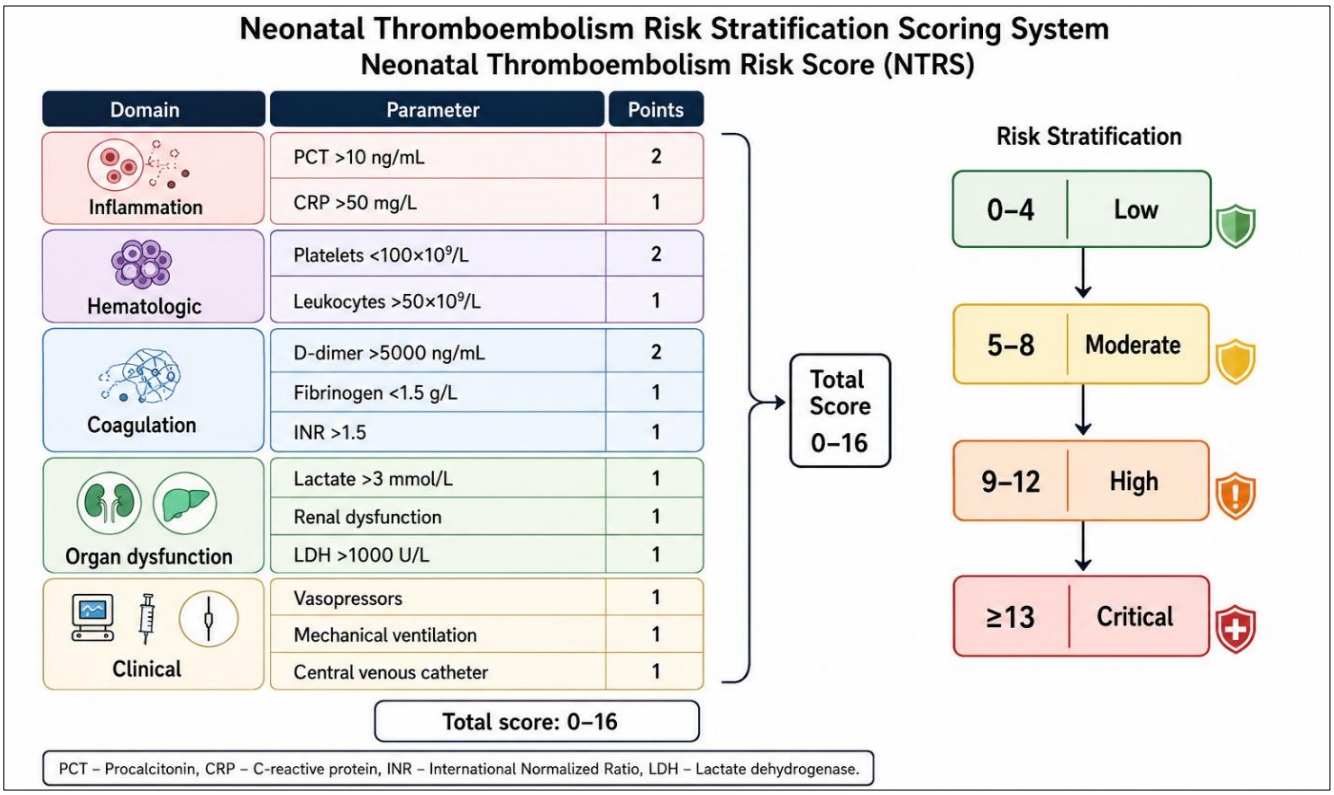

Figure 1: The NTRS model.

In comparison to previous studies that have principally focus on isolated risk factors such as central venous catheters, sepsis, or congenital heart disease, this study reports a critical gap by utilizing multiple routinely existing laboratory parameters into a unified scoring system. On the contrary with past research, which lacks a widely accepted, complete neonatal laboratory risk model, the NTRS offers a real-world tool for early risk stratification. This combination elevates the clinical utility by simplifying timely diagnosis of neonates at high risk for thromboembolism, thereby controlling neuroimaging and anticoagulation choices. Proactive management has the possibility to progress clinical outcomes by plummeting diagnostic delays and improving therapeutic interventions.

However, the study's limitations must be recognized. The small sample size (n=4) limits the generalizability of findings, and the retrospective design may present selection and information partialities. The absence of genetic thrombophilia testing confines the understanding of inherited risk association, and absence of external validation permits attention before prevalent clinical implementation. These limitations underscore the requirement for prospective, multicenter studies to confirm the NTRS and evaluate its predictive accuracy among diverse neonatal populations.

CONCLUSION

Neonatal thromboembolism in the background of sepsis is predicted by a measurable laboratory profile. The proposed NTRS offers a practical framework for early risk stratification and may simplify timely diagnostic and therapeutic interventions.

Recommendations

Future research must focus on combining advanced coagulation assays such as Protein C and Antithrombin III levels and using viscoelastic testing modalities like TEG and ROTEM to perfect the model's sensitivity. Additionally, implementing machine-learning approaches can boost predictive capabilities by detecting multipart interactions among clinical and laboratory variables. Such advances would reinforce the risk stratification outline and simplify tailored management strategies.

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