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Case Report

Necrotizing enterocolitis in an extremely preterm infant born to a mother with type 1 diabetes mellitus: a case report with focused literature review

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ABSTRACT

Necrotizing enterocolitis is a major cause of morbidity and mortality in very preterm infants. Maternal type 1 diabetes mellitus is strongly associated with prematurity, but its independent association with necrotizing enterocolitis remains uncertain. A 27-year-old woman with pregestational type 1 diabetes mellitus developed threatened preterm labour and later delivered a male infant at 27 weeks and 4 days of gestation weighing 880 g. The infant required intubation and surfactant therapy for extreme prematurity. During the neonatal course, he developed abdominal distension, bilious aspirates, worsening respiratory status, and mixed acidosis consistent with Bell stage IIA necrotizing enterocolitis. Management included bowel rest, gastric decompression, intravenous antibiotics, parenteral nutrition, close monitoring, and early surgical review. Feeds were later reintroduced using expressed breast milk, and the infant recovered without surgery, was discharged home, and remained well at follow-up, with appropriate growth and no recurrent gastrointestinal symptoms. Relevant cohort studies, meta-analyses, and guideline-level evidence regarding maternal type 1 diabetes mellitus, prematurity, and necrotizing enterocolitis were reviewed. Current evidence supports a strong association between maternal type 1 diabetes mellitus and prematurity, while evidence for an independent association with necrotizing enterocolitis remains inconsistent. This case emphasizes the importance of antenatal optimization, early neonatal stabilization, prompt recognition of intestinal disease, careful reintroduction of human milk feeding, and structured post-discharge follow-up.

Keywords: Necrotizing enterocolitis, Prematurity, Type 1 diabetes mellitus, Infant of diabetic mother, Antenatal corticosteroids, Magnesium sulphate, Human milk

INTRODUCTION

Necrotizing enterocolitis is a severe inflammatory bowel disease of infancy that predominantly affects preterm neonates and remains a leading cause of neonatal morbidity and mortality.[1-3] Its pathogenesis is multifactorial and involves intestinal immaturity, abnormal microbial colonization, impaired mucosal perfusion, and an exaggerated inflammatory response.[2,3] Clinically, it presents with feeding intolerance, abdominal distension, bilious gastric aspirates, bloody stools, apnea, bradycardia, and systemic instability. Diagnosis rests on clinical findings supported by radiographic or sonographic evidence, and disease severity is commonly described

using Bell staging.[4,5] Management depends on severity and typically includes bowel rest, broad spectrum antibiotics, gastric decompression, parenteral nutrition, and surgical consultation when perforation or progressive deterioration is suspected.[6,7]

Maternal type 1 diabetes mellitus is not an established direct cause of necrotizing enterocolitis, but it is strongly associated with preterm birth, especially in pregnancies complicated by poor glycaemic control, microalbuminuria, nephropathy, and preeclampsia.[14-18] Because prematurity is the dominant risk factor for necrotizing enterocolitis, pregnancies complicated by type 1 diabetes mellitus may create a high risk neonatal context even when a direct diabetes to necrotizing enterocolitis link is uncertain.[14,15,21,22] This case report and focused literature review examine that interface and aim to place a clinically successful case within the best available evidence.

CASE REPORT

The timeline of this case report is presented in Table 1. A 27 year old woman with pregestational type 1 diabetes mellitus first presented to a peripheral district general hospital at approximately 24 weeks of gestation with uterine contractions. She received intramuscular dexamethasone and erythromycin, after which the contractions settled. At 27 weeks and 4 days of gestation, she developed recurrent contractions and hypertension. Nifedipine was commenced and she was transferred to a regional tertiary unit with specialist neonatal care. Where references are cited within this case description, they are provided to indicate that the corresponding antenatal or neonatal intervention reflects current evidence-based or guideline-recommended practice rather than an isolated clinical decision; the supporting evidence for these interventions is examined in detail in the literature review and discussion sections.

At the referral centre, magnesium sulphate was administered for fetal neuroprotection in the setting of threatened very preterm birth.[10] Delivery occurred at 27 weeks and 4 days of gestation. A male infant weighing 880 g was born and required immediate neonatal stabilization. Because of extreme prematurity and high risk of respiratory distress syndrome, he was intubated after birth and received surfactant therapy in keeping with current neonatal respiratory practice.[13]

During the neonatal course, the infant developed abdominal distension, green bilious nasogastric aspirates, increased oxygen requirement, and mixed acidosis on blood gas analysis. These findings were consistent with Bell stage IIA necrotizing enterocolitis Figure 1, Figure 2. Management included bowel rest, gastric decompression, intravenous broad-spectrum antibiotics, parenteral nutrition, serial abdominal assessment, laboratory surveillance, and prompt paediatric surgical review. Following clinical stabilization, enteral feeds were reintroduced gradually using expressed breast milk. Feed tolerance improved, abdominal findings resolved, and the infant recovered without operative intervention. He was subsequently discharged home after an overall uncomplicated recovery.

Following discharge, the infant was enrolled in the routine neonatal follow-up programme for extremely preterm infants. At subsequent follow-up visits, he continued to tolerate full enteral feeds of expressed breast milk with appropriate interval weight gain, and no recurrence of abdominal distension, feeding intolerance, or other signs suggestive of a late intestinal stricture or recurrent necrotizing enterocolitis was observed. Growth was tracked against corrected gestational age, and developmental surveillance was planned in line with standard follow-up protocols for extremely preterm infants, given their recognised long-term risk of growth restriction and neurodevelopmental impairment.

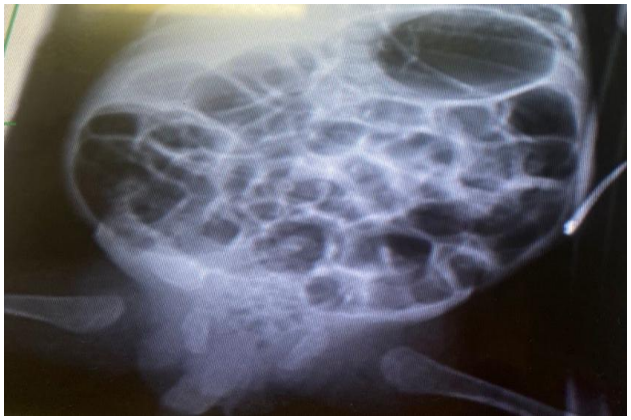

Figure 1: Plain abdominal radiograph (supine view).
*Multiple dilated, gas-filled bowel loops with a bubbly, soap-bubble-like pattern, in keeping with the necrotizing enterocolitis findings described in this case.

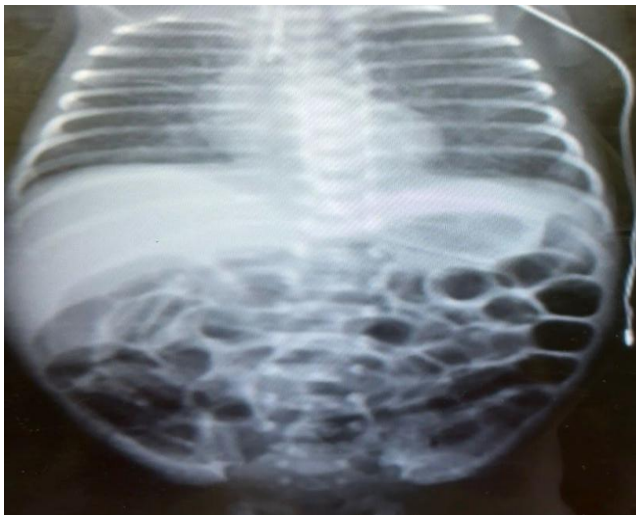

Figure 2: Supine chest and abdominal radiograph.
*Diffusely dilated, gas-filled bowel loops throughout the abdomen with a similar bubbly pattern, consistent with the necrotizing enterocolitis findings described in this case.

Table 1: Case timeline.

Time points	Clinical event	Management	Comment
About 24 weeks	First episode of threatened preterm labour at peripheral hospital	Dexamethasone and erythromycin	Contractions settled
27 weeks and 4 days before delivery	Recurrent contractions and hypertension	Nifedipine and transfer to tertiary centre	High risk delivery context
Before delivery	Threatened very preterm birth	Magnesium sulphate	Fetal neuroprotection
Birth	Male infant, 880 g, 27 weeks and 4 days	Intubation and surfactant	Extreme prematurity and respiratory risk
Neonatal course	Abdominal distension, bilious aspirates, increasing oxygen need, mixed acidosis	Evaluation for Bell stage IIA necrotizing enterocolitis	Prompt recognition of intestinal disease
After NEC diagnosis	Medical NEC management	Bowel rest, gastric decompression, antibiotics, parenteral nutrition, monitoring, surgical review	No operative intervention needed
Recovery phase	Improving abdominal signs and feed tolerance	Gradual reintroduction of expressed breast milk	Good clinical response
Discharge	Clinically stable infant	Home discharge	Overall uncomplicated recovery
Follow-up	No recurrence of feeding intolerance or abdominal signs; appropriate interval weight gain	Enrolment in neonatal follow-up programme; growth and developmental surveillance	Favourable short-term outcome

DISCUSSION

Literature review methods

A structured narrative review was conducted to contextualize the present case within the existing evidence. Studies were selected based on relevance to three predefined domains: maternal type 1 diabetes mellitus and prematurity, maternal diabetes and necrotizing enterocolitis risk, and placental and antenatal mechanisms potentially linking maternal disease to neonatal intestinal injury. Priority was given to meta-analyses, multicenter cohort studies, national datasets, and guideline-level evidence. Additional references were included to support key aspects of necrotizing enterocolitis diagnosis, management, and prevention. The aim was to provide an evidence-based interpretive framework rather than a formal pooled analysis.

Evidence synthesis

Maternal type 1 diabetes mellitus and prematurity

Across multiple high-quality cohort studies, maternal type 1 diabetes mellitus is consistently associated with an increased risk of preterm delivery. This association includes both spontaneous and indicated preterm birth and is observed in comparison with both uncomplicated pregnancies and other high-risk conditions.[14,15] The risk is further amplified in the presence of diabetic microvascular complications such as microalbuminuria and nephropathy, as well as with suboptimal glycaemic control and the development of preeclampsia.[16-18] Collectively, these findings establish maternal type 1 diabetes mellitus as a high-risk obstetric condition in which prematurity represents the most robust and reproducible adverse outcome pathway.

Maternal diabetes and necrotizing enterocolitis risk

In contrast to the strong and consistent association with prematurity, evidence linking maternal diabetes mellitus directly to necrotizing enterocolitis in preterm infants is heterogeneous. Some cohort studies have reported an increased risk of moderate to severe necrotizing enterocolitis among infants born to diabetic mothers, particularly in extremely preterm populations or in association with pregestational insulin use.[19,20] However, larger international cohort analyses, matched case-control studies, and meta-analyses have not demonstrated a consistent independent association after adjustment for confounding factors.[21-24] Taken together, the available evidence does not support maternal diabetes mellitus as a reliable independent causal factor for necrotizing enterocolitis, and any observed association appears to be context-dependent and potentially mediated by other variables.

Placental and antenatal mechanisms as a biological bridge

A more coherent explanatory framework emerges when placental and antenatal factors are considered. Maternal type 1 diabetes mellitus has been associated with placental features consistent with uteroplacental malperfusion.[25]

Independent studies of necrotizing enterocolitis have demonstrated associations with fetal vascular obstructive lesions, maternal vascular malperfusion, abnormal antenatal Doppler flow patterns, and inflammatory conditions such as chorioamnionitis.[26-29] These findings support a biologically plausible pathway in which diabetic pregnancy contributes to an adverse intrauterine environment characterized by vascular and inflammatory stress. In the context of extreme prematurity, these antenatal exposures may increase susceptibility of the immature intestine to postnatal injury and dysregulated inflammation, thereby facilitating the development of necrotizing enterocolitis in selected infants.

Clinical management and protective factors

The literature also provides strong support for the key management strategies applied in this case. Necrotizing enterocolitis is understood as a multifactorial disease involving intestinal immaturity, microbial dysbiosis, impaired perfusion, and exaggerated inflammatory responses.[2,3] Diagnosis relies on clinical presentation supported by imaging modalities, including the increasing use of bowel ultrasound.[4,5] Standard medical management includes bowel rest, broad-spectrum antibiotics, parenteral nutrition, and close monitoring, with surgical consultation when disease progression/complications are suspected.[6,7]

Preventive and supportive strategies are equally important. Human milk feeding has consistently been shown to reduce the incidence and severity of necrotizing enterocolitis in very low birth weight infants.[8,9] In addition, antenatal interventions such as corticosteroids and magnesium sulphate, along with evidence-based neonatal respiratory support including surfactant administration, contribute to improved outcomes in very preterm infants.[10-13]

The structured evidence supporting these interpretations is summarized in Table 2.

Table 2: Selected evidence linking maternal type 1 diabetes mellitus, prematurity, and necrotizing enterocolitis.

Themes	Study	Design and population	Main findings
Prematurity in pregestational diabetes	Sibai et al 2000[14]	Prospective comparison of women with pregestational diabetes, chronic hypertension, and uncomplicated pregnancies	Pregestational diabetes was associated with higher overall, spontaneous, and indicated preterm delivery rates
Pregnancy complications in type 1 diabetes	Evers et al 2004[15]	Nationwide prospective study of pregnancies in women with type 1 diabetes in the Netherlands	Prematurity and preeclampsia were markedly more common than in the background population
Microalbuminuria and prematurity	Ekbom et al 2001[16]	Prospective cohort of pregnant women with type 1 diabetes	Microalbuminuria and nephropathy were strongly associated with preterm delivery and preeclampsia
HbA1c, preeclampsia, and preterm birth	Jensen et al 2009[17]	Prospective nationwide Danish cohort of women with type 1 diabetes	Microalbuminuria predicted preeclampsia and higher third trimester HbA1c predicted delivery before 34 weeks
Factors linked to preterm delivery in type 1 diabetes	Lepercq et al 2004[18]	Cohort study of pregnancies in women with type 1 diabetes	Poor glycaemic control, nephropathy progression, and preeclampsia were linked to preterm delivery
Maternal diabetes and NEC signal	Grandi et al 2015[19]	Multicentre Latin American cohort of very low birth weight infants	Maternal diabetes was independently associated with Bell stage 2 to 3 NEC
Maternal insulin use before pregnancy and NEC	Boghossian et al 2016[20]	Prospective cohort of infants born at 22 to 28 weeks	Higher NEC risk was found in infants of mothers using insulin before pregnancy
Large adjusted cohort with null result	Persson et al 2018[21]	International cohort of infants born at 24 to 31 weeks and less than 1500 g	Maternal diabetes was not independently associated with NEC after adjustment
Meta-analysis of preterm infant outcomes	Razak and Faden 2020[22]	Systematic review and meta-analysis	No significant overall association between maternal diabetes and NEC in preterm infants
Matched NEC study with null diabetes result	March et al 2015[23]]	Retrospective matched case-control study	Diabetes was not significantly associated with NEC; intrauterine growth restriction was more prominent
National case-control NEC study	Ahle et al 2018[24]	Swedish national matched case-control study	Maternal diabetes was not independently associated with NEC
Diabetic placental pathology	Huynh et al 2015[25]	Placental pathology study across diabetes types	Type 1 diabetes was associated with uteroplacental malperfusion features
Placental vascular lesions and NEC	Dix et al 2010[26]	Placental histopathology study in preterm infants with and without NEC	Fetal vascular obstructive lesions were markedly more common in infants who later developed NEC

Continued.

Theme	Study	Design and population	Main finding
Prenatal origins of NEC	Watson and McElroy 2021[27]	Narrative review of prenatal NEC risk factors	Prenatal vascular and inflammatory exposures may contribute to NEC pathogenesis
Antenatal and placental predictors of NEC	Duci et al 2019[28]	Case-control study	Abnormal antenatal umbilical artery flow, chorioamnionitis, and preeclampsia patterns were linked to NEC severity or risk
Maternal vascular malperfusion and NEC severity	Garg et al 2022[29]	Matched case-control study in preterm infants	Multiple placental lesions and maternal vascular malperfusion were associated with NEC
General NEC pathogenesis	Niño et al 2016[2]	Review	NEC arises from intestinal immaturity, microbial, perfusion, and inflammatory mechanisms
NEC diagnosis	D'Angelo et al 2018[4]	Review	Diagnosis combines clinical, laboratory, and imaging features
Bowel ultrasound and imaging practice	Alexander et al 2021[5]	Practice implementation study	Ultrasound can improve NEC diagnosis and management
Medical NEC management	Hu et al 2024[6]	Review	Bowel rest, antibiotics, supportive care, and tailored escalation remain core therapy
Surgical NEC management	Thakkar and Lakhoo 2016[7]	Review	Surgical consultation is needed for perforation or failure of medical therapy
Human milk and NEC prevention	Herrmann and Carroll 2014[8]	Review	Exclusive human milk feeding reduces NEC risk
NEC reduction bundle in VLBW infants	Chandran et al 2021[9]	Quality improvement study	Evidence based feeding and care practices reduced NEC and improved nutrition outcomes
Magnesium sulphate	Shepherd et al 2024[10]	Cochrane review	Magnesium sulphate before very preterm birth improves neuroprotection
Repeat antenatal corticosteroids	Walters et al 2022[11]	Cochrane review	Repeat doses can reduce respiratory morbidity in selected women still at risk of preterm birth
Antenatal corticosteroid meta-analysis	Crowther et al 2019[12]	Individual participant meta-analysis	Repeat prenatal corticosteroids improved some neonatal respiratory outcomes
Neonatal RDS guidelines	Sweet et al 2023[13]	Guideline update	Early respiratory support and surfactant remain standard for very preterm infants at risk of RDS

Necrotizing enterocolitis is a multifactorial disease of prematurity characterized by intestinal immaturity, dysregulated microbial colonization, impaired perfusion, and an exaggerated inflammatory response.[2,3] In this context, understanding how maternal conditions contribute to neonatal risk requires careful distinction between direct causation and indirect risk pathways.

The most consistent and well-established association in the literature is between maternal type 1 diabetes mellitus and preterm birth. Multiple cohort studies have demonstrated that pregnancies complicated by type 1 diabetes are associated with substantially higher rates of both spontaneous and indicated preterm delivery.[14,15] This risk is further amplified in the presence of microvascular complications such as microalbuminuria and nephropathy, as well as with suboptimal glycaemic control and the development of preeclampsia.[16-18] These findings support the interpretation that maternal type 1 diabetes mellitus primarily contributes to neonatal morbidity by creating a high-risk obstetric environment in which extreme prematurity is more likely to occur. In the present case, delivery at 27 weeks and 4 days of gestation placed the infant within a population already highly vulnerable to intestinal injury.

By contrast, evidence supporting maternal diabetes as an independent risk factor for necrotizing enterocolitis remains inconsistent. While some cohort studies have reported increased rates of moderate to severe necrotizing enterocolitis among infants of diabetic mothers, particularly in extremely preterm populations or in association with pregestational insulin use.[19,20] However, larger adjusted analyses, matched case-control studies, and meta-analyses have not demonstrated a consistent independent association.[21-24]

This heterogeneity suggests that maternal diabetes alone is unlikely to represent a direct causal factor. Instead, any observed relationship is likely context-dependent as well as mediated by the other variables, most notably prematurity.

A more coherent explanation emerges when placental and antenatal factors are considered. Maternal type 1 diabetes mellitus has been associated with placental features consistent with uteroplacental malperfusion.[25] Independent studies of necrotizing enterocolitis have identified links with fetal vascular obstructive lesions, maternal vascular malperfusion, abnormal antenatal Doppler flow patterns, and inflammatory conditions such as chorioamnionitis.[26-29] Together, these findings support a biologically plausible pathway in which diabetic pregnancy contributes to an adverse intrauterine environment characterized by vascular and inflammatory stress. In the setting of extreme prematurity, these antenatal exposures may increase the susceptibility of the immature intestine to postnatal injury and dysregulated inflammation, thereby facilitating the development of necrotizing enterocolitis in selected infants rather than acting as a direct causal mechanism.

The clinical course and management of present case were consistent with current evidence-based practice. Early recognition of abdominal signs and systemic deterioration allowed prompt identification of Bell stage IIA necrotizing enterocolitis. Standard medical management-including bowel rest, gastric decompression, intravenous antibiotics, parenteral nutrition, and close monitoring-was appropriately implemented, with early surgical consultation.[6,7] Subsequent cautious reintroduction of enteral feeding using expressed breast milk is particularly relevant, as human milk has consistently been shown to reduce incidence and severity of necrotizing enterocolitis in very low birth weight infants.[8,9]

In addition, antenatal and immediate neonatal interventions aligned with best practice recommendations. The use of antenatal corticosteroids and magnesium sulphate in the setting of threatened very preterm birth, together with early respiratory stabilization and surfactant therapy after delivery, is well supported by the literature and contributes to improved neonatal outcomes.[10-13] These measures form part of a broader continuum of care that modifies risk even in high-risk pregnancies.

Follow-up is an important component of the overall management of extremely preterm infants who recover from necrotizing enterocolitis. In this case, the infant's enteral feeding tolerance and growth were monitored after discharge, with no recurrence of abdominal distension, feeding intolerance, or other signs suggestive of a late intestinal stricture. Continued surveillance of growth and neurodevelopment in the context of routine preterm follow-up programmes remains important, given the recognised long-term risks associated with extreme prematurity and with necrotizing enterocolitis itself.

Taken together, this case supports a balanced interpretation of the relationship between maternal type 1 diabetes mellitus and necrotizing enterocolitis. The strongest evidence links maternal diabetes to prematurity, while the evidence for a direct independent association with necrotizing enterocolitis remains inconclusive.[14-24] A more plausible unifying framework is that maternal diabetes contributes indirectly to necrotizing enterocolitis risk through increased rates of very preterm birth and, in some pregnancies, through placental vascular or inflammatory dysfunction that affects the developing intestine.[25-29]

This case is useful because it links a familiar neonatal emergency to a maternal background that is clinically important but often overstated in the literature. It supports a balanced interpretation. Maternal type 1 diabetes mellitus clearly matters for prematurity risk. Prematurity remains the dominant substrate for necrotizing enterocolitis. Any additional independent diabetes effect on necrotizing enterocolitis is uncertain. Placental vascular and inflammatory pathology may help explain why some diabetic pregnancies are followed by intestinal morbidity in the neonate.

CONCLUSION

This case illustrates the successful medical management of Bell stage IIA necrotizing enterocolitis in an extremely preterm infant born to a mother with type 1 diabetes mellitus. The literature strongly supports an association between maternal type 1 diabetes mellitus and prematurity, but only mixed evidence for an independent association with necrotizing enterocolitis. The most defensible interpretation is that maternal T1DM contributes to necrotizing enterocolitis risk chiefly by increasing the likelihood of very preterm birth and, in some pregnancies, placental vascular or inflammatory dysfunction. Timely antenatal management, specialized neonatal stabilization, early recognition of intestinal disease, and careful use of human milk feeding, together with structured follow-up, remain central to good outcomes.

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