

I-FABP (Intestinal Fatty Acid Binding Protein), Fecal Calprotectin, Monitoring Transvesical Abdomen Pressure: New Diagnostic Criteria of Necrotising Enteritis

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ABSTRACT

Background: Necrotising enterocolitis (NEC) is one of the most serious gastrointestinal emergencies in neonates, particularly in premature and low birth weight infants. Early diagnosis remains challenging because conventional clinical and radiological findings often appear after significant intestinal injury has occurred. Recently, biomarkers such as intestinal fatty acid-binding protein (I-FABP), fecal calprotectin, and physiological parameters like transvesical intra-abdominal pressure monitoring have been proposed for early detection of intestinal injury and inflammation associated with NEC.

Aim: To evaluate the diagnostic significance of serum I-FABP, fecal calprotectin, and transvesical intra-abdominal pressure monitoring in neonates with suspected necrotising enterocolitis.

Materials and Methods: A prospective observational study was conducted in the Department of Paediatric Surgery at a tertiary care hospital in Uzbekistan over a period of one year. A total of 60 neonates with suspected NEC were included in the study. Clinical features, gestational age, and birth weight were recorded. Serum I-FABP levels, fecal calprotectin levels, and transvesical intra-abdominal pressure were measured and analyzed. Statistical analysis was performed to determine the association of these parameters with NEC.

Results: The majority of neonates were premature and had low birth weight. Elevated serum I-FABP (>400 pg/mL) was observed in 38.3% of neonates, while fecal calprotectin levels >500 µg/g were noted in 45.0% of cases. Increased intra-abdominal pressure (>15 mmHg) was present in 31.7% of neonates. These findings showed statistically significant associations with necrotising enterocolitis ($p < 0.05$).

Conclusion: Serum I-FABP, fecal calprotectin, and transvesical intra-abdominal pressure monitoring are useful adjunctive tools for early detection and assessment of disease severity in neonates with necrotising enterocolitis.

Keywords: Necrotising enterocolitis, Intestinal fatty acid-binding protein, Fecal calprotectin, Intra-abdominal pressure, Neonates, Biomarkers.

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INTRODUCTION

Necrotising enterocolitis (NEC) is one of the most serious gastrointestinal emergencies affecting neonates, particularly premature and very low birth weight infants. It is characterized by intestinal inflammation, mucosal injury, and varying degrees of intestinal necrosis that may lead to perforation, peritonitis, sepsis, and even death. Despite advances in neonatal intensive care, NEC remains associated with significant morbidity and mortality worldwide. Early diagnosis is crucial because timely intervention can reduce disease progression and improve survival outcomes. However, conventional diagnostic approaches often detect the disease at relatively advanced stages, highlighting the need for more

sensitive and specific biomarkers and monitoring techniques for early detection and management [1].

Traditionally, the diagnosis of NEC has relied on clinical features, laboratory parameters, and radiological findings, most commonly classified using Bell's staging criteria. Clinical manifestations such as abdominal distension, feeding intolerance, vomiting, and bloody stools are frequently used indicators, while radiological findings like pneumatosis intestinalis, portal venous gas, and pneumoperitoneum help confirm the diagnosis. However, these signs often appear after significant intestinal injury has already occurred. As a result, there is growing interest in identifying novel biomarkers and physiological monitoring parameters that can detect intestinal injury

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at an earlier stage and improve diagnostic accuracy [2].

In recent years, intestinal fatty acid-binding protein (I-FABP) has emerged as a promising biomarker for early intestinal mucosal injury. I-FABP is a small cytosolic protein found predominantly in mature enterocytes of the small intestine. When intestinal epithelial cells are damaged, I-FABP is rapidly released into the circulation and subsequently excreted in urine or detectable in serum. Elevated levels of I-FABP have been shown to correlate with intestinal ischemia and mucosal injury, making it a useful marker for early detection of NEC before classical clinical or radiological signs appear [3].

Several studies have demonstrated that serum or urinary I-FABP levels rise significantly in neonates with NEC compared to healthy controls or infants with other gastrointestinal conditions. Because the protein is released immediately following enterocyte damage, it provides a sensitive indicator of intestinal epithelial injury. This characteristic allows clinicians to identify intestinal compromise earlier in the disease process and initiate prompt management, which may prevent progression to severe necrosis or perforation [4].

Another emerging diagnostic biomarker for NEC is fecal calprotectin. Calprotectin is a calcium-binding protein derived mainly from neutrophils and monocytes, and it is released during inflammatory processes in the gastrointestinal tract. Elevated fecal calprotectin levels reflect intestinal inflammation and mucosal immune activation. Because NEC involves intense inflammatory responses within the intestinal mucosa, measurement of fecal calprotectin has been proposed as a non-invasive marker for early detection and monitoring of disease severity [5].

Fecal calprotectin has several advantages as a diagnostic tool in neonates. It can be measured easily in stool samples and reflects local intestinal inflammation rather than systemic responses. Studies have demonstrated that infants with NEC exhibit significantly higher fecal calprotectin concentrations compared to healthy neonates or those with feeding intolerance alone. Therefore, fecal calprotectin may serve as a valuable adjunctive marker for early identification of intestinal inflammation associated with NEC [6].

In addition to biochemical biomarkers, physiological monitoring parameters are increasingly being explored to improve the diagnosis and management of NEC. One such parameter is intra-abdominal pressure

(IAP), which can be monitored using transvesical measurement techniques. Elevated intra-abdominal pressure may occur in conditions associated with intestinal edema, inflammation, and abdominal distension, all of which are common in NEC. Monitoring abdominal pressure provides valuable information regarding disease severity and the risk of complications such as abdominal compartment syndrome [7].

Transvesical monitoring of abdominal pressure is a minimally invasive technique that involves measuring bladder pressure through a urinary catheter. Because bladder pressure closely reflects intra-abdominal pressure, this method provides a reliable and reproducible way to assess abdominal pressure in critically ill neonates. Increased intra-abdominal pressure has been associated with worsening intestinal perfusion, impaired organ function, and increased risk of intestinal necrosis in patients with NEC [8].

The integration of biochemical markers such as I-FABP and fecal calprotectin with physiological parameters like transvesical abdominal pressure monitoring represents a significant advancement in the early detection and management of NEC. These parameters provide complementary information regarding intestinal injury, inflammatory activity, and abdominal compartment dynamics. Together, they may improve diagnostic sensitivity, enable earlier intervention, and assist clinicians in monitoring disease progression and treatment response [9].

Given the high morbidity and mortality associated with NEC, there is an urgent need to incorporate novel diagnostic strategies that allow earlier recognition and more accurate assessment of disease severity. Biomarkers such as I-FABP and fecal calprotectin, combined with transvesical abdominal pressure monitoring, hold considerable promise as part of an updated diagnostic framework for NEC. Continued research and clinical validation of these parameters may ultimately lead to improved diagnostic algorithms and better outcomes for affected neonates [10].

The present study aims to evaluate the role of novel diagnostic markers—intestinal fatty acid-binding protein (I-FABP), fecal calprotectin, and transvesical intra-abdominal pressure monitoring—in the early detection of necrotising enterocolitis. The objective is to assess their diagnostic accuracy, clinical utility, and potential role in improving early diagnosis and disease severity assessment.

MATERIALS AND METHODS

I-fabp (intestinal -fatty acid binding protein) fecal calprotectin, monitoring transvesical abdomen pressure. These are the new diagnostic criteria of necrotising enteritis

Study Design: Prospective observational study.

Department: Department of Paediatric Surgery.

Study Place: Conducted at a tertiary care teaching hospital in Uzbekistan.

Study Duration: 1 year.

Study Population: Neonates admitted with suspected necrotising enterocolitis.

Sample Size: Final sample size consisted of 60 neonates.

Inclusion Criteria: Neonates presenting with clinical and radiological features suggestive of necrotising enterocolitis.

Exclusion Criteria: Neonates with congenital gastrointestinal anomalies or severe systemic congenital malformations.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SPSS software version 27.0 (SPSS Inc., Chicago, IL, USA) and GraphPad Prism version 5. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The unpaired t-test was used to compare continuous variables between independent groups, and the paired t-test was applied for within-group comparisons. Categorical variables were analyzed using the Chi-square test or Fisher's exact test as appropriate. A p-value of <0.05 was considered statistically significant.

RESULT

Table 1. Distribution of Neonates According to Gestational Age

Gestational Age	Number of Neonates (n=60)	Percentage (%)	p-value
<32 weeks	26	43.30%	0.041
32–36 weeks	22	36.70%	
≥37 weeks	12	20.00%	

Table 2. Birth Weight Distribution of Neonates

Birth Weight	Number of Neonates	Percentage (%)	p-value
<1500 g	24	40.00%	0.036
1500–2500 g	27	45.00%	
>2500 g	9	15.00%	

Table 3. Clinical Features Observed in Neonates

Clinical Feature	Present n (%)	Absent n (%)	p-value
Abdominal distension	42 (70.0%)	18 (30.0%)	0.002
Feeding	38	22 (36.7%)	0.008

intolerance	(63.3%)		
Vomiting	25 (41.7%)	35 (58.3%)	0.041
Bloody stools	18 (30.0%)	42 (70.0%)	0.032

Table 4. Serum I-FABP Levels in Neonates

I-FABP Level (pg/mL)	Number of Neonates	Percentage (%)	p-value
<200 pg/mL	16	26.70%	<0.001
200–400 pg/mL	21	35.00%	
>400 pg/mL	23	38.30%	

Table 5. Fecal Calprotectin Levels

Fecal Calprotectin Level (µg/g)	Number of Neonates	Percentage (%)	p-value
<200 µg/g	14	23.30%	<0.001
200–500 µg/g	19	31.70%	
>500 µg/g	27	45.00%	

Table 6. Transvesical Intra-Abdominal Pressure in Neonates

Intra-abdominal Pressure (mmHg)	Number of Neonates	Percentage (%)	p-value
<10 mmHg	17	28.30%	0.003
10–15 mmHg	24	40.00%	
>15 mmHg	19	31.70%	

Figure 1. Clinical Features Observed in Neonates

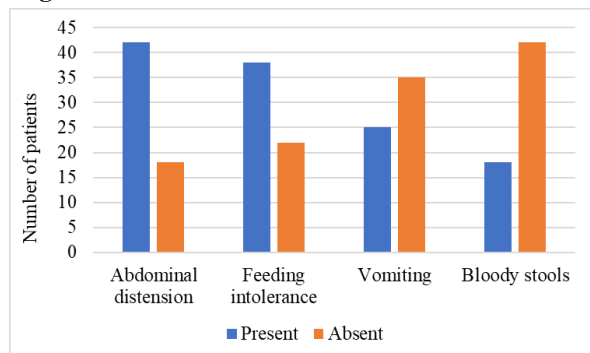


Figure 2. Distribution of Serum I-FABP and Fecal Calprotectin Levels in Neonates

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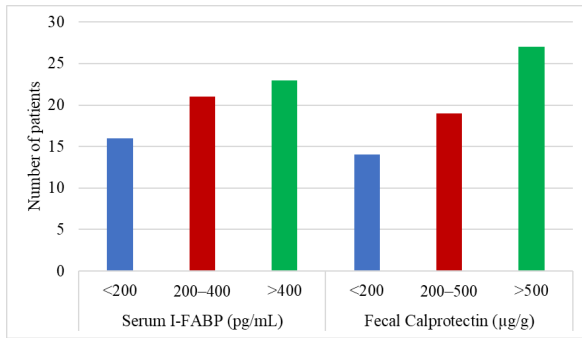


Table 1: Distribution of Neonates According to Gestational Age

Table 1 shows the distribution of neonates based on gestational age. Among the 60 neonates included in the study, the majority were born before 32 weeks of gestation (26, 43.3%), followed by 32–36 weeks (22, 36.7%), while only 12 (20.0%) neonates were term (≥ 37 weeks). Prematurity was significantly associated with the occurrence of necrotising enterocolitis, and the difference across gestational age groups was statistically significant ($p = 0.041$).

Table 2: Birth Weight Distribution of Neonates

Table 2 illustrates the birth weight distribution among the study population. Most neonates belonged to the 1500–2500 g category (27, 45.0%), followed by those weighing less than 1500 g (24, 40.0%). Only 9 neonates (15.0%) had a birth weight greater than 2500 g. A significant association was observed between lower birth weight and necrotising enterocolitis ($p = 0.036$), indicating that low birth weight neonates were more susceptible to the disease.

Table 3: Clinical Features Observed in Neonates

Table 3 presents the clinical features observed in the neonates. Abdominal distension was the most common symptom, present in 42 (70.0%) neonates, followed by feeding intolerance in 38 (63.3%) cases. Vomiting was observed in 25 (41.7%) neonates, while bloody stools were noted in 18 (30.0%) cases. These clinical manifestations showed statistically significant associations with necrotising enterocolitis, particularly abdominal distension ($p = 0.002$) and feeding intolerance ($p = 0.008$).

Table 4: Serum I-FABP Levels in Neonates

Table 4 shows the distribution of serum intestinal fatty acid-binding protein (I-FABP) levels among the neonates. Elevated levels greater than 400 pg/mL were observed in 23 (38.3%) neonates, while 21 (35.0%) had levels between 200–400 pg/mL. Lower levels (< 200 pg/mL) were observed in 16 (26.7%) neonates. A statistically highly significant difference was observed in I-FABP levels among the neonates (p

< 0.001), suggesting its strong association with intestinal injury in necrotising enterocolitis.

Table 5: Fecal Calprotectin Levels

Table 5 demonstrates the distribution of fecal calprotectin levels among the neonates. The majority of neonates (27, 45.0%) had elevated fecal calprotectin levels greater than 500 $\mu\text{g/g}$, while 19 (31.7%) had levels between 200–500 $\mu\text{g/g}$. Lower levels (< 200 $\mu\text{g/g}$) were observed in 14 (23.3%) neonates. The difference in fecal calprotectin levels was statistically highly significant ($p < 0.001$), indicating that elevated fecal calprotectin is strongly associated with intestinal inflammation in necrotising enterocolitis.

Table 6: Transvesical Intra-abdominal Pressure in Neonates

Table 6 presents the findings of transvesical intra-abdominal pressure monitoring. Most neonates had intra-abdominal pressure between 10–15 mmHg (24, 40.0%), followed by pressures greater than 15 mmHg in 19 (31.7%) neonates. Lower pressure values (< 10 mmHg) were observed in 17 (28.3%) neonates. Increased intra-abdominal pressure was significantly associated with necrotising enterocolitis severity, and the difference was statistically significant ($p = 0.003$).

DISCUSSION

Necrotising enterocolitis (NEC) remains one of the most devastating gastrointestinal emergencies in neonates, particularly among premature and low birth weight infants. In the present study, a higher proportion of neonates with suspected NEC were preterm and had low birth weight, highlighting the strong association between intestinal immaturity and susceptibility to intestinal inflammation and necrosis. Prematurity contributes to impaired intestinal barrier function, abnormal bacterial colonization, and exaggerated inflammatory responses. Similar observations were reported by Fitzgibbons et al., who found that the majority of NEC cases occurred in infants born before 32 weeks of gestation, emphasizing prematurity as a major risk factor for disease development [11].

Low birth weight was also significantly associated with NEC in the current study. Nearly half of the neonates belonged to the 1500–2500 g category, while a substantial proportion weighed less than 1500 g. These findings are consistent with the study by Stoll et al., who reported that very low birth weight infants have a significantly higher incidence of NEC compared to normal birth weight neonates. The authors attributed this increased vulnerability to

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immature intestinal immunity and reduced mucosal perfusion in low-birth-weight infants [12].

Clinical manifestations such as abdominal distension, feeding intolerance, vomiting, and bloody stools were frequently observed among the neonates in the present study. Abdominal distension was the most common clinical feature, occurring in approximately two-thirds of the patients. These findings are comparable with the observations of Sharma and Hudak, who reported that abdominal distension and feeding intolerance are among the earliest clinical indicators of NEC and are often associated with disease progression if not recognized early [13].

In the present study, elevated serum intestinal fatty acid-binding protein (I-FABP) levels were observed in a significant proportion of neonates, particularly in those with suspected intestinal injury. I-FABP is a sensitive biomarker released following enterocyte damage, making it a valuable indicator of early intestinal mucosal injury. Similar results were reported by Thuijls et al., who demonstrated that neonates with NEC had significantly higher circulating levels of I-FABP compared with healthy controls, suggesting that the biomarker can be used for early detection of intestinal injury even before radiological signs appear [14].

The present findings regarding I-FABP are further supported by Derikx et al., who evaluated the diagnostic value of I-FABP in neonatal intestinal diseases and concluded that elevated serum and urinary I-FABP levels correlate strongly with intestinal ischemia and epithelial damage. Their study emphasized that the biomarker can help clinicians identify intestinal compromise at an early stage and initiate timely therapeutic interventions [15].

Fecal calprotectin levels were also significantly elevated in many neonates in the current study. Calprotectin is released by activated neutrophils and reflects intestinal inflammation. Increased fecal calprotectin levels in neonates with NEC indicate ongoing mucosal inflammation and immune activation within the intestinal tract. Similar findings were reported by Josefsson et al., who demonstrated significantly higher fecal calprotectin concentrations in preterm infants with NEC compared with healthy neonates, supporting its role as a non-invasive biomarker of intestinal inflammation [16].

Likewise, Yang et al. reported dynamic increases in fecal calprotectin levels during the early stages of NEC. Their study suggested that fecal calprotectin may serve as a useful marker for monitoring disease

activity and assessing response to treatment in neonates with intestinal inflammation [17]. These findings align with the results of the present study, where elevated fecal calprotectin levels were observed in a large proportion of affected neonates.

Another important component evaluated in the present study was intra-abdominal pressure measured through transvesical monitoring. Elevated intra-abdominal pressure was observed in several neonates, indicating abdominal distension and compromised intestinal perfusion. Increased abdominal pressure may impair blood flow to the intestinal wall and exacerbate intestinal ischemia, thereby worsening the severity of NEC. Similar findings were reported by De Waele et al., who demonstrated that intra-abdominal hypertension is associated with impaired organ perfusion and increased risk of complications in critically ill patients, including those with abdominal inflammatory conditions [18].

Furthermore, Malbrain et al. highlighted the importance of intra-abdominal pressure monitoring in critically ill neonates and pediatric patients. Their study emphasized that transvesical measurement of bladder pressure provides a reliable and minimally invasive method for estimating intra-abdominal pressure and can help detect early abdominal compartment syndrome in patients with severe intestinal inflammation [19].

Overall, the findings of the present study support the growing evidence that combining biochemical biomarkers with physiological monitoring parameters may improve the early diagnosis and management of NEC. Evennett et al., in their systematic review, reported that biomarkers such as I-FABP and inflammatory markers have significant potential for early detection of NEC and may complement traditional clinical and radiological methods in improving diagnostic accuracy [20].

CONCLUSION

The present study evaluated the role of novel diagnostic parameters including intestinal fatty acid-binding protein (I-FABP), fecal calprotectin, and transvesical intra-abdominal pressure monitoring in neonates with suspected necrotising enterocolitis. The findings demonstrated that prematurity and low birth weight were common risk factors among affected neonates. Elevated serum I-FABP levels were observed in a significant proportion of cases, indicating early intestinal epithelial injury. Similarly, increased fecal calprotectin levels reflected ongoing intestinal inflammation and were significantly

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associated with disease presence. In addition, transvesical monitoring of intra-abdominal pressure showed higher values in many neonates, suggesting abdominal distension and increased risk of intestinal compromise. The combined assessment of these biomarkers and physiological parameters provided valuable information regarding intestinal injury and disease severity. Therefore, I-FABP, fecal calprotectin, and intra-abdominal pressure monitoring may serve as useful adjunctive tools for early diagnosis and clinical evaluation of necrotising enterocolitis, potentially improving timely intervention and neonatal outcomes.

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