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2026 Volume 11, Issue 4
Advances in Precision Medicine
ISSN: 2424-9106 (Online)
ISSN: 2424-8592 (Print)

The Correlation and Diagnostic Value of Serum MBL, MASP1, MASP2, and Coagulation Function Indexes in Children with Acute Leukemia Complicated by Infection

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Abstract: *Objective:* To explore the correlation and diagnostic value of serum mannose-binding lectin (MBL), mannose-binding lectin-associated serine protease 1 (MASP1), and MASP2 levels and coagulation function indexes in children with acute leukemia complicated by infection. *Methods:* A retrospective analysis was conducted on the clinical data of 149 pediatric patients with acute leukemia admitted to our hospital from January 1, 2023, to December 31, 2025. The patients were categorized into three groups based on their infection status: sepsis (38 cases), common infection (51 cases), and non-infection (60 cases). Serum levels of MBL, MASP1, and MASP2 were measured using ELISA. Coagulation function tests were performed to analyze D-dimer (D-D), prothrombin time (PT), activated partial thromboplastin time (APTT), and fibrinogen (FIB) levels. Pearson correlation analysis was used to explore the relationship between serum MBL, MASP1, MASP2, and coagulation function indicators. The diagnostic performance of each indicator, both individually and in combination, was evaluated using receiver operating characteristic (ROC) curves. *Results:* The serum levels of MBL (1889.18 ± 174.91 ng/mL), MASP1 (31.01 ± 3.60 ng/mL), MASP2 (555.81 ± 55.90 pg/mL), and coagulation function indicators (APTT, PT, FIB, D-D) in the sepsis group were significantly higher than those in the common infection group and the non-infection group ($P < 0.05$). Pearson correlation analysis showed a significant positive correlation between serum MBL, MASP1, MASP2, and coagulation function indicators ($P < 0.05$). ROC curve analysis showed that the AUC of MBL, MASP1, and MASP2 was 0.675, 0.782, and 0.745, respectively when detected alone, while the AUC of the three combined tests was 0.842, with a sensitivity of 86.0% and a specificity of 82.0%. The diagnostic performance was significantly better than that of the single test. *Conclusion:* The levels of serum MBL, MASP1, and MASP2, along with coagulation function indicators, were significantly elevated in children with acute leukemia complicated by infection. These three factors are closely related to coagulation function indicators. Jointly testing MBL, MASP1, and MASP2 provides a high diagnostic value for the condition, offering crucial evidence for early clinical diagnosis and disease assessment.

Keywords: Acute leukemia; MBL; MASP1; MASP2; Coagulation function

Online publication: April 26, 2026

1. Introduction

Acute leukemia (AL) is one of the most common malignant tumors in children, with a complex etiology that may involve genetic factors, environmental exposure, and abnormal immune function. Children with leukemia often have weakened immune defense. Chemotherapy can further suppress their immune system. Therefore, infection is a frequent complication during treatment and may seriously threaten their health and survival. Sepsis is the most serious type of infection. It is often accompanied by systemic inflammation and multiple organ dysfunction, and it may become life-threatening in children [1]. The complement system is an important part of innate immunity. Among its activation pathways, the mannose-binding lectin (MBL) pathway plays a key role in pathogen recognition. MBL binds to mannose structures on pathogen surfaces and then activates mannose-binding lectin-associated serine protease 1 (MASP1) and MASP2, which further triggers the complement cascade. Recent studies suggest that complement activation and coagulation dysfunction may affect each other during infection. Complement activation can promote coagulation abnormalities, while coagulation activation may further strengthen complement responses. This interaction may create a harmful cycle and worsen disease progression [2]. However, the diagnosis of infection in children with acute leukemia still mainly depends on clinical symptoms, pathogen detection, and inflammatory markers, such as C-reactive protein and procalcitonin. These markers are useful, but their specificity is limited. In our view, they may not be sufficient for early and accurate identification of infection type and severity. Coagulation dysfunction is also common in infected children. It is closely related to disease progression and prognosis. However, the relationship between complement activation and coagulation abnormalities in acute leukemia with infection remains unclear. Many previous studies have mainly used cell experiments or animal models, and clinical evidence remains relatively limited. Therefore, measuring serum MBL, MASP1, and MASP2 levels in children with acute leukemia and infection may be useful. Analyzing their associations with coagulation indicators may also help clarify the possible link between complement activation and coagulation dysfunction during infection. These findings may provide additional evidence for early diagnosis and treatment, although further clinical validation is still needed.

This study measured serum MBL, MASP1, MASP2, and coagulation indicators in children with acute leukemia and infection. It also explored the possible correlations among these markers and their diagnostic value. The analysis of MBL, MASP1, and MASP2 expression may provide some clinical evidence for the involvement of complement activation in infection. Second, exploring the association between complement-related molecules and coagulation indicators may help explain the interaction between inflammation and coagulation dysfunction. Although this study cannot fully prove a causal mechanism, it may provide a useful basis for further research and potential clinical intervention.

2. Research status

Natural immunity, also known as non-specific immunity, refers to the body's innate physiological defense mechanisms that can respond to various pathogenic microorganisms and foreign substances. This is the first line of defense against pathogens. Research has shown that complement activation plays a crucial role in natural immunity. There are N-acetylglucosamine and other sugar structures on the surface of pathogens through three pathways for complement activation: the classical pathway, the alternative pathway, and the lectin pathway. In the lectin pathway, MBL/fibronectin binds to MBL and mannoprotein, forming a C1-like complex with MASP1/2. The MBL recognizes and binds to mannos of the CRD. This triggers the subsequent activation of MASP2, which cleaves complement C4 and C2 to form C3 convertase C4b2a, thus initiating the complement activation of the lectin pathway. Complement activation triggers a cascade of enzymatic reactions, leading to the production of mediators responsible for clearing pathogens and triggering inflammation. These mediators recruit phagocytes to the infection site, accelerating the phagocytosis of pathogens, killing them, and causing cell lysis [3,4]. It has been found that MASP2, as a protease necessary for MBL and several (H, M, and L) fibrocollagen to activate complement, is a key protease in the agglutinin pathway and plays a crucial role in the agglutinin pathway [5,6].

The MASP2 molecule is a single-chain structure, consisting of six functional regions from the N to the C end: the CUB region (complement subcomponent CIr/CIs-like domain), the EGF region (epidermal growth factor-like domain), the CUB region, two CCP regions (complement control protein domain), and the SP region (serine protease) [7,8]. Research indicates that the functions of the N and C ends of the MASP protein are relatively independent: the three domains at the N end of MASP—two CUB regions and one EGF region—are the binding sites for MASP with MBL-CLR, interacting with MBL in a Ca^{2+} dependent manner to form the MBL-MASPs complex. The three domains at the C end—two CCP regions and one SP region—are the active centers of serine proteases, primarily responsible for activating complement through the SP region, which cleaves complement C4 and C2 to produce C3 convertase [9].

Changes in serum MASP2 levels are related to many diseases. These diseases include infectious diseases, tumors, and autoimmune disorders. The c.359A > G mutation in the *MASP2* gene changes the encoded amino acid from aspartic acid (Asp) to glycine (Gly). This mutation is located in the CUB1 domain. It may reduce serum MASP2 levels or even lead to MASP2 deficiency. This mutation may also reduce the binding of MASP2 to MBL and fibronectin. This can make the immune recognition complex less stable. As a result, the lectin pathway may not be activated normally. This change may increase the risk of pathogen infection or worsen existing infection [10]. MASP2 has also been studied in tumors. One study showed that serum MASP2 level was an independent marker for predicting recurrence and survival in colorectal cancer [11]. Other studies reported that MASP2 was associated with the progression and invasive behavior of esophageal squamous cell carcinoma [12]. In addition, *MASP2* gene polymorphisms may be related to susceptibility to HCV infection. Higher serum MASP2 concentrations have also been observed in patients with HCV infection [13].

Recent studies on *MASP2* gene polymorphisms suggest that mutations at different sites may affect plasma MASP2 levels. These mutations may also change the function of the MASP2 protein. However, the effects of different mutation sites may not be completely consistent, and this still needs further clinical evidence. The c.359A > G mutation has been detected in Chinese, Danish, Caucasian, African, and American Indian populations. This mutation is only found in the Danish Caucasian population, with a frequency of 3.9%, while in the Chinese population, the CHNH156_159 point mutation has been identified and is associated with low plasma MASP2 levels, with a mutation frequency of 0.26% [14]. In Zambia, the average plasma MASP2 level is 196 ng/ml, compared to 262 ng/ml in Hong Kong, 290 ng/ml in Brazil, and 416 ng/ml in Danish Caucasians. Studies have reported a direct relationship between serum MASP2 concentration and patients with tumors, such as colorectal cancer, esophageal squamous cell carcinoma, liver cancer, and ovarian cancer, where serum MASP2 concentrations are significantly reduced. Additionally, in infectious diseases like pneumonia and fungal invasive infections following hematopoietic stem cell transplantation, MASP2 levels are also significantly reduced. In autoimmune diseases such as psoriasis, endemic pemphigus foliaceus, rheumatic fever, and hereditary angioedema, MASP2 levels are also significantly reduced. However, it remains unclear whether the deficiency of the MASP2 protein caused by the c.359A > G mutation is related to the susceptibility of leukemia patients after hematopoietic stem cell transplantation.

The literature reports that higher levels of MASP2 in serum are associated with improved survival in children with hematological malignancies, particularly lymphoma [15]. In children with leukemia undergoing chemotherapy, a deficiency in MASP2 increases the risk of febrile neutropenia, is linked to prolonged hospital stays, and is associated with intravenous antibiotic therapy [16]. Therefore, we hypothesize that MASP2 plays a crucial role in protecting children who have undergone hematopoietic stem cell transplantation (HSCT) from various microbial pathogens. A mutation at the c.359A > G site of the *MASP2* gene results in reduced expression of MASP2 protein in both serum and liver, which may lead to secondary infections in children who have undergone HSCT. Rapid detection of *MASP2* gene typing could help predict disease risk and provide an effective method for large-scale population screening.

3. Data and methods

3.1. General information

A retrospective analysis was conducted on the clinical data of 150 pediatric patients with acute leukemia admitted to our hospital from January 1, 2023, to December 31, 2025. The patients were categorized into three groups based on whether they exhibited infection symptoms: 49 cases of sepsis, 58 cases of common infections, and 42 cases of no infections. Among the sepsis group, there were 26 males and 24 females, ranging in age from 0.5 to 12 years (mean age 6.82 ± 3.15 years). In the common infection group, there were 32 males and 28 females, ranging in age from 0.6 to 12 years (mean age 7.05 ± 3.28 years). In the no infection group, there were 20 males and 20 females, ranging in age from 0.5 to 12 years (mean age 6.75 ± 3.10 years). There were no significant differences in gender, age, and other basic information among the three groups ($P > 0.05$), indicating that the groups were comparable.

Inclusion criteria: All participants in the observation group met the AL diagnostic criteria [7] and were clinically diagnosed with concurrent infection. Any of the following abnormalities in coagulation function was defined as abnormal: prothrombin time (TT) prolonged > 3s; activated partial thromboplastin time (APTT) prolonged > 10s; fibrinogen (FIB) < 2 g/L; D-dimer (D-D) level elevated > 0.3 mg/L; prothrombin time (PT) prolonged > 15s. The control group did not show significant proliferation of bone marrow primitive cells or immature cells, infections, bleeding, anemia, or extramedullary tissue infiltration through blood tests, bone marrow morphology, and histochemical staining. This study was approved by the hospital's medical ethics committee, and all participants signed informed consent forms. Exclusion criteria: Participants with organic lesions of vital organs such as liver and kidneys; participants with congenital heart disease, malformations due to rubella virus infection, congenital hip dislocation, or other congenital diseases; guardians with mental or behavioral abnormalities.

3.2. Experimental materials

The human mannose-binding lectin (MBP; MBL) quantitative detection kit (item number: RX105914H), the human mannan-binding lectin-associated serine protease 1 (MASP1) quantitative detection kit (item number: RX106945H), and the human mannan-binding lectin serine peptidase-2 (MASP-2) quantitative detection kit (item number: RX103076H) were all purchased from RuiXin Biotechnology Co., Ltd. The HBS-1096A microplate reader was purchased from Nanjing Defei Biotechnology Co., Ltd.

3.3. Methodology

3.3.1. Experimental methods for serum samples

All subjects were collected 4 mL of venous blood on an empty stomach from 6:00 to 8:00 a.m., placed in the procoagulation tube for 30 min, and then placed in the ordinary centrifuge at a speed of 2,500 r/min (centrifugal radius 8 cm) for 10 min. The serum samples were collected and stored in a -80°C refrigerator for testing.

3.3.2. Detection methods of serum MBL, MASP1, and MASP2

Set up the sample wells (add 100 µL of standard) and the double-standard wells (add 100 µL of the sample). Gently shake to mix, then cover with a plate holder. Incubate at 37°C for 2 hours. Discard the liquid and shake dry. Add 100 µL of biotin-labeled antibody working solution to each well, cover with a new plate holder, and incubate at 37°C for 1 hour. Discard the liquid, shake dry, and wash the plates three times, soaking for 2 minutes each time, then shaking dry. Add 100 µL of horseradish peroxidase-labeled avidin working solution to each well, cover with a new plate holder, and incubate at 37°C for 1 hour. Discard the liquid, shake dry, and wash the plates five times, soaking for 2 minutes each time, then shaking dry. Add 90 µL of substrate solution to each well in sequence, and incubate in the dark at 37°C for 30 minutes. Add 50 µL of stop solution to each well in sequence to terminate the reaction. Detect the reaction using a microplate reader within 5 minutes after the reaction is stopped, and set the wavelength to 450 nm.

3.3.3. Detection methods of coagulation function indicators (D-dimer, PT, APTT, FIB)

All subjects had their venous blood samples collected at 6:00 to 8:00 AM on an empty stomach, with a volume of 2 mL. The blood was placed in a 3.2% sodium citrate anticoagulant tube (product number: CSB-E08847h, purchased from Wuhan Huamei Biotechnology Co., Ltd.) and gently mixed 5 to 6 times to ensure adequate anticoagulation. The tubes were then placed in a centrifuge and centrifuged at 3,000 r/min (radius 8 cm) for 10 minutes to separate the plasma. The upper layer of plasma was used for testing. After turning on the instrument, calibration was performed according to the manual, using calibrators for D-D, PT, APTT, and FIB to ensure the accuracy of the test results. During testing, 50 μL of plasma was added to the test wells, along with the appropriate reagents (D-dimer antibody, prothrombin, partial thromboplastin, or thrombin working solution). After mixing, the samples were incubated at 37°C for the specified time (10 min for D-D, 2 min for PT, 3 min for APTT, and 5 min for FIB). Once the reaction was initiated, the clotting time was recorded, or the absorbance value was read, and the concentrations of each indicator were calculated based on the standard curve.

3.4. Statistical analysis

SPSS 18.0 statistical software was used for data analysis. Measurement data were presented as mean ± standard deviation (SD). ANOVA was used to compare the three groups, and independent samples t-tests were used for pairwise comparisons between groups. Count data were expressed as rates, and χ^2 tests were employed. Pearson correlation analysis was conducted to examine the relationships between serum MBL, MASP1, MASP2, and coagulation function indicators (D-D, PT, APTT, FIB). The analysis of influencing factors involved a multivariate logistic regression, with the area under the ROC curve, critical value, sensitivity, and specificity calculated using ROC curves. $P < 0.05$ was considered statistically significant.

4. Results

4.1. Baseline data

There was no significant difference in the baseline data of the three groups ($P > 0.05$), and they were comparable, as shown in **Table 1**.

Table 1. Comparison of general data of three groups of children [mean ± SD, cases (%)]'

Group	Cases	Age (years)	BMI (kg/m²)	Acute lymphoblastic leukemia	Acute myeloid leukemia
Sepsis group	50	6.82 ± 3.15	16.85 ± 2.34	42(84.00)	8(16.00)
Common infection group	60	7.05 ± 3.28	16.96 ± 2.17	48(80.00)	12(20.00)
Non-infection group	40	6.75 ± 3.10	16.50 ± 2.20	30(75.00)	10(25.00)
χ^2/F		0.208	0.543	1.385	1.385
P		0.813	0.582	0.501	0.501

4.2. Comparison of serum MBP, MASP1, and MASP2 levels in three groups of children

The serum levels of MBP, MASP1, and MASP2 in children with sepsis were significantly higher than those in the common infection group and the non-infection group, and the serum levels of MBP, MASP1, and MASP2 in the normal infection group were significantly higher than those in the non-infection group, all of which were statistically significant ($P < 0.05$), as shown in **Table 2**.

Table 2. Comparison of serum MBP, MASP1, and MASP2 levels in three groups of children (mean ± SD)

Group	Cases	MBP (ng/mL)	MASP1 (ng/mL)	MASP2 (pg/mL)
Sepsis group	50	1889.18 ± 174.91	31.01 ± 3.60	555.81 ± 55.90
Common infection group	60	1250.10 ± 139.45[a]	20.32 ± 2.14[a]	320.34 ± 41.25[a]
Non-infection group	40	750.42 ± 126.28[ab]	9.06 ± 1.38[ab]	150.75 ± 25.19[ab]
F		198.456	312.809	925.609
P		0.001	0.001	0.001

4.3. Comparison of coagulation function indexes among the three groups of children

The levels of APTT, PT, FIB, and D-D in children with sepsis were significantly longer or higher than those in the common infection group and the non-infection group, and the levels of APTT, PT, FIB, and D-D in children with the common infection group were significantly longer or higher than those in the non-infection group, all of which were statistically significant ($P < 0.05$), as shown in **Table 3**.

Table 3. Comparison of coagulation function indexes in three groups of children (mean ± SD)

Group	Cases	APTT (s)	PT (s)	FIB (g/L)	D-D (mg/L)
Sepsis group	50	41.04 ± 2.78	15.95 ± 1.45	6.12 ± 0.68	5.62 ± 0.77
Common infection group	60	35.63 ± 2.04[a]	14.10 ± 1.09[a]	4.20 ± 0.45[a]	2.15 ± 0.31[a]
Non-infection group	40	27.17 ± 2.31[ab]	12.97 ± 0.65[ab]	3.12 ± 0.61[ab]	0.42 ± 0.05[ab]
F		285.456	112.809	198.630	1,245.609
P		0.001	0.001	0.001	0.001

Note: Compared with the sepsis group, [a]$P < 0.05$; compared with the general infection group, [b]$P < 0.05$.

4.4. Correlation between serum MBP, MASP1, MASP2, and coagulation function

The results of Pearson correlation analysis showed that serum MBP, MASP1, MASP2, and coagulation function indexes (D-D, PT, APTT, FIB) were significantly positively correlated ($P < 0.05$), as shown in **Table 4**.

Table 4. Correlation between serum MBP, MASP1, MASP2 and coagulation function

Metric	MBL		MASP1		MASP2	
	r	***P***	***r***	***P***	***r***	***P***
APTT	0.287	0.001	0.256	0.004	0.298	0.002
PT	0.215	0.018	0.198	0.027	0.241	0.007
FIB	0.263	0.003	0.205	0.015	0.278	0.001
D - D	0.304	0.000	0.267	0.002	0.356	0.001

4.5. Analysis of the diagnostic value of serum MBP, MASP1, and MASP2 in children with acute leukemia complicated by infection

When MBP, MASP1, and MASP2 are used individually or in combination, the ROC-AUC (95% CI) values are 0.675 (0.372,0.945), 0.782 (0.612,0.928), 0.745 (0.482,0.978), and 0.842 (0.736,0.941), respectively. MBP has a relatively low diagnostic performance, while MASP1 and MASP2 show some diagnostic effectiveness. When all three are used together, the diagnostic performance is significantly improved, with AUC, sensitivity, specificity, and accuracy all showing significant improvements compared to when each indicator is used alone (**Table 5** and **Figure 1**).

Table 5. Analysis of the diagnostic value of serum MBP, MASP1, and MASP2 levels in children with acute leukemia complicated by infection

Metric	AUC (0.95CI)	Sensitivity	Specificity	Youden index	Accuracy
MBP	0.675 (0.372, 0.945)	0.650	0.660	0.310	0.655
MASP1	0.782 (0.612, 0.928)	0.770	0.750	0.520	0.760
MASP2	0.745 (0.482, 0.978)	0.720	0.730	0.450	0.725
Joint application	0.842 (0.736, 0.941)	0.860	0.820	0.680	0.840

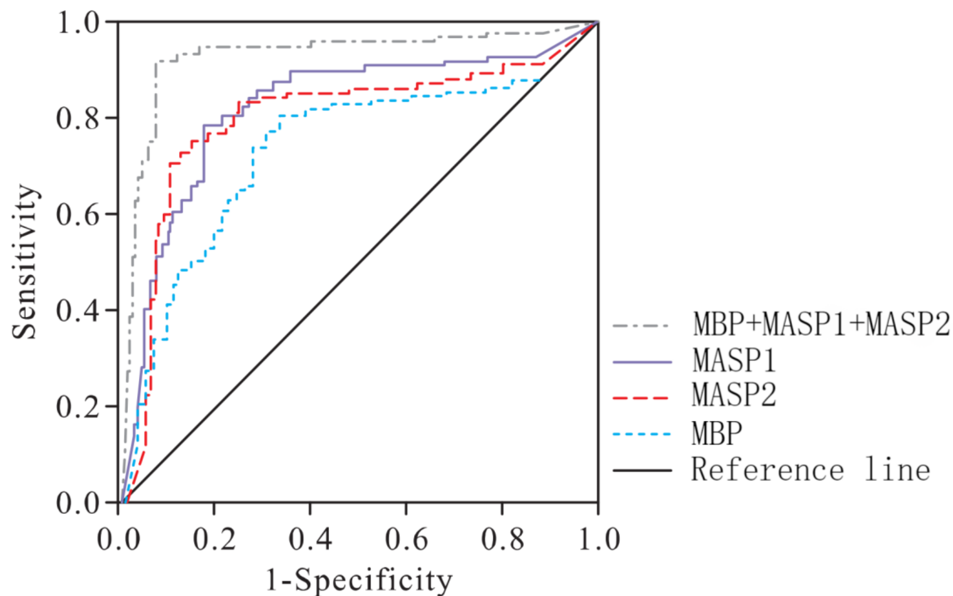

Figure 1. ROC curve of serum MBL, MASP1, and MASP2 for the diagnosis of children with acute leukemia complicated by infection

5. Discussion

Acute leukemia is one of the most common malignant tumors in children. Its incidence has increased in recent years. Leukemia cells grow rapidly in the bone marrow and suppress normal blood cell production. This can reduce the number and function of white blood cells, especially neutrophils [17–20]. As a result, the natural immune defense of children becomes weaker.

Chemotherapy can further damage immune function. It may inhibit lymphocyte activity and reduce immunoglobulin levels [21,22]. Therefore, children with acute leukemia are prone to infection during treatment. Common symptoms include fever, cough, sputum, abdominal pain, diarrhea, frequent urination, and urgency [23]. Among these symptoms, fever is the most common. Severe infection may progress to sepsis or infectious shock. Some children may show fatigue, pale skin, cold limbs, and low blood pressure. If treatment is delayed, the condition may rapidly develop into multiple organ failure [24,25].

Infection in children with acute leukemia may be related to several factors. Leukemia cells can suppress normal hematopoiesis and reduce neutrophil levels, which weakens pathogen clearance. Chemotherapy may further damage cellular and humoral immunity [26,27]. At the same time, leukemia cell infiltration can impair mucosal barriers and make pathogen invasion easier [28]. Long hospital stays may also increase exposure to hospital pathogens and drug-resistant bacteria. In addition, long-term use of broad-spectrum antibiotics may disturb the microbial balance and increase the risk of opportunistic infection [29].

MASP1 is an important enzyme in the MBL-MASP complex. It can cleave complement components and may promote activation of the lectin pathway. MASP1 may also take part in coagulation and fibrinolysis by activating prekallikrein and factor XII. Therefore, changes in MASP1 activity may be associated with both infection response and coagulation abnormalities. MASP2 is another key enzyme in the lectin pathway. It mainly cleaves C4 and C2 and helps form C3 convertase. Compared with MASP1, MASP2 may be more directly involved in complement activation. In children with acute leukemia and infection, abnormal MASP2 levels may lead to weak or excessive complement activation. This may affect pathogen clearance and may also contribute to inflammatory tissue injury.

Coagulation indicators can also reflect disease severity in infected children [24]. APTT mainly reflects the intrinsic coagulation pathway. PT reflects the extrinsic coagulation pathway. Prolonged APTT or PT may suggest coagulation factor consumption, liver dysfunction, or disseminated intravascular coagulation. FIB is an important coagulation protein. Its increase may reflect inflammation or stress, while its decrease may indicate severe coagulation consumption. D-dimer reflects fibrin formation and degradation. Increased D-dimer often suggests coagulation activation or secondary fibrinolysis.

In this study, serum MBL, MASP1, and MASP2 levels were higher in children with sepsis than in those with common infection or no infection. Their levels were also higher in children with common infections than in uninfected children. This suggests that activation of the MBL pathway may be related to infection severity in children with acute leukemia. At the same time, APTT, PT, FIB, and D-dimer also increased or prolonged with infection severity. These changes indicate that infection may activate the coagulation system and disturb the coagulation balance. Correlation analysis further showed that serum MBL, MASP1, and MASP2 were positively related to D-dimer, PT, APTT, and FIB. This result suggests a possible link between complement activation and coagulation dysfunction. During infection, MBL may activate MASP1 and MASP2 and then promote the production of complement fragments such as C3a and C5a. These fragments may enhance inflammation, activate endothelial cells, and promote coagulation to some extent. At the same time, coagulation-related molecules, such as thrombin, may further influence complement activation. This possible interaction may contribute to a cycle of inflammation and coagulation dysfunction, especially in children with sepsis [30].

This study also evaluated the diagnostic value of MBL, MASP1, and MASP2. The results showed that MBL alone had relatively limited diagnostic value, while MASP1 and MASP2 showed better performance. The combined analysis of these three markers further improved the diagnostic efficiency. This may be because MBL levels are affected by several factors, such as genetic background and nutritional status. In contrast, MASP1 and MASP2 may reflect activation of the lectin complement pathway more directly. Therefore, combined detection of MBL, MASP1, and MASP2 may provide more useful information for identifying infection in children with acute leukemia and may serve as a supplementary reference for clinical evaluation.

6. Conclusion

In summary, the levels of serum MBL, MASP1, and MASP2, along with coagulation function indicators (D-D, PT, APTT, FIB), are significantly elevated in children with acute leukemia complicated by infection. These three factors show a clear positive correlation with coagulation function indicators, suggesting a close interaction between the complement system and the coagulation system during infections. Furthermore, the combined detection of serum MBL, MASP1, and MASP2 has a high diagnostic efficacy for acute leukemia complicated by infection. The AUC, sensitivity, specificity, and accuracy are all significantly better than those of single tests, providing an important reference for early clinical diagnosis and condition assessment.

Disclosure statement

The author declares no conflict of interest.

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