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Case Study

Hidden In The Blood: Ngs- Revealed Aspergillus Waynelawii In A Critically Ill Neutropenic Child - A Case Of Serendipitous Discovery

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ABSTRACT

Invasive aspergillosis is a potentially fatal opportunistic infection in neutropenic children. Next-generation sequencing (NGS)-based microbial diagnostics can detect fungal pathogens directly from blood samples with high sensitivity, but interpretation remains challenging when uncommon environmental fungi are identified. A 2-year-7-month-old male child was admitted to the Paediatric Intensive Care Unit with severe pancytopenia (haemoglobin 4.3 g/dL, absolute neutrophil count 2500/µL, platelet count 40,000/µL). Peripheral blood NGS identified Aspergillus waynelawii, a rarely reported environmental opportunistic mould. Considering the profound neutropenia and risk of invasive fungal infection, empirical antifungal therapy with voriconazole was initiated. Supportive management included three packed red blood cell transfusions and two platelet transfusions. Subsequent bone marrow biopsy demonstrated approximately 50% blasts with features of a haematolymphoid neoplasm, confirming Acute Lymphoblastic Leukaemia (ALL), L2 subtype (FAB classification). This case highlights the diagnostic challenges posed by NGS detection of rare fungal organisms in immunocompromised children. The identification of Aspergillus waynelawii may represent either early fungal infection or environmental contamination. The case also illustrates an uncommon aleukemic presentation of ALL, with absent peripheral blasts despite significant marrow

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involvement. Integrating molecular diagnostics with bone marrow examination is essential in children with unexplained pancytopenia. Early identification and prompt oncological evaluation are critical for optimizing outcome.

INTRODUCTION

Acute Lymphoblastic Leukaemia (ALL) is the most common childhood cancer and the most frequent type of leukaemia in children, accounting for approximately 75–80% of pediatric leukaemia cases. It is a malignant disorder characterized by the uncontrolled proliferation and accumulation of immature lymphoid precursor cells (lymphoblasts) in the bone marrow, peripheral blood, and other organs.

In ALL, malignant lymphoblasts proliferate rapidly and replace normal hematopoietic stem cells within the bone marrow, leading to impaired production of red blood cells, white blood cells, and platelets. As a result, affected children commonly present with anemia, recurrent infections, fever, bleeding tendencies, bone pain, lymphadenopathy, and hepatosplenomegaly.

The exact cause of ALL remains unclear; however, genetic abnormalities, chromosomal translocations, environmental exposures, and inherited predisposition syndromes are known to contribute to its development. The incidence peaks between 2 and 5 years of age, with a slight predominance in males. Advances in diagnostic techniques, risk stratification, supportive care, and multi-agent chemotherapy have significantly improved outcomes.

Invasive fungal infections are a major cause of morbidity and mortality among immunocompromised patients, particularly those with hematological malignancies, prolonged neutropenia, or undergoing intensive chemotherapy. Species belonging to the genus Aspergillus are among the most common opportunistic fungal pathogens, with Aspergillus fumigatus, Aspergillus flavus, and Aspergillus niger accounting for the majority of clinically recognized infections. However, advances in molecular diagnostics and next-generation sequencing (NGS) have enabled the identification of uncommon and emerging Aspergillus species that may otherwise remain undetected by conventional microbiological methods.

CASE REPORT

A 2year 7 months old boy weighing 13 kg presented fatigue of 3-4 days along with reduced oral intake for 1 week and a past history of febrile illness with 3 days duration one month back. On admission, the vitals were stable except for mild tachycardia (Heart Rate of 128b/pm) Respiratory rate was within normal limits for age. On systemic examination the patient was pale with mild hepatomegaly the cardiovascular examination showed tachycardia. The child was admitted for haematological stabilisation and comprehensive work up.

1. Haematological Investigations

1.1 Complete Blood Count (Admission — 02/06/2026)

Table 1: Complete Blood Count with Red Cell Indices at Admission

Parameter	Result	Reference Range	Status / Comment
Haemoglobin (Hb)	4.3 g/dL	11.0–17.0 g/dL	Severely Low ↓↓↓
Total Count (TC)	2800 /µL	4000–11000 /µL	Leukopenia ↓
ANC	250 /µL	>1500 /µL	Severe Neutropenia ↓↓↓
Platelet Count	40,000 /µL	1.5–4.0 L/µL	Thrombocytopenia ↓↓
RBC Count	1.62 M/µL	3.8–5.5 M/µL	Low

PCV	13.6%	35–45%	Severely Low
MCV	84 fL	70–90 fL	Normal (Normocytic)
MCH	26.5 pg	27–33 pg	Low-normal
MCHC	31.6 g/dL	31–37 g/dL	Low-normal
RDW	15.3%	<14.5%	Mildly Elevated
Differential (Poly/Lymph)	9% / 88.6%	40–70% / 20–40%	Relative Lymphocytosis

Summary: Severe normocytic normochromic anaemia with pancytopenia (anaemia + leukopenia with severe neutropenia + thrombocytopenia). Differential count showing relative lymphocytosis (88.6%) with neutropenia (9% polys, ANC 250) is a recognised pattern in both aplastic anaemia and aleukemic leukaemia.

1.2 Biochemical and Coagulation Profile

Table 2: Biochemical, Coagulation, Immunological, and Thyroid Parameters

Test	Result	Reference Range	Clinical Significance
LDH Serum	211 U/L	120–246 U/L	Normal — argues against ALL/HLH
Uric Acid	5.8 mg/dL	3.5–8.5 mg/dL	Normal — no tumour lysis
INR	1.040	0.9–1.1	Normal — no coagulopathy
PT / PT Control	14.06 / 13.6 s	11–15 s	Normal
D-Dimer	0.51 µg/mL	<0.5 µg/mL	Mildly elevated — non-specific
CRP	2.5 mg/L	<5.0 mg/L	Normal — low inflammatory burden
Iron / TIBC	232 / 292	—	Transferrin sat. 80% — iron loading
Vitamin B12	306 pg/mL	239–931 pg/mL	Normal — rules out megaloblastic
TSH	1.955 µIU/mL	0.8–8.2 µIU/mL	Normal — no thyroid disease
T3 / T4	0.536 / 8.25	0.82–1.58 / 4.5–12.2	Low T3 — Sick Euthyroid Syndrome
ALT / AST	62 / 54 U/L	<40 / <40 U/L	Mildly elevated — hepatic stress
Albumin / Protein	3.84 / 6.3 g/dL	3.5–5.0 / 6.0–8.0	Normal
Na / K	133 / 4.72 mEq/L	136–146 / 3.5–5.0	Mild hyponatraemia

1.3 Viral Serology Panel

Table 3: Comprehensive Viral Serology Screen — All Negative

Test	Result	Clinical Relevance
HAV IgM (Anti-HAV)	Nonreactive	Rules out hepatitis A-associated aplasia
HBsAg (CMIA)	Nonreactive	Rules out HBV-associated cytopenias
HCV (CMIA)	Nonreactive	Rules out HCV-related marrow suppression
HIV 1&2 (CMIA)	Nonreactive	Rules out HIV-associated pancytopenia/OI

Portable AP Chest X-Ray demonstrated apparent cardiomegaly with no consolidation, pleural effusion, or pulmonary nodularity. No halo sign, air-crescent sign, or perihilar haziness suggestive of invasive pulmonary aspergillosis was identified.

Echocardiography showed Two-dimensional echocardiography was performed to evaluate cardiac structure and function in the context of the apparent CXR cardiomegaly and compensatory tachycardia. Findings were entirely normal: situs solitus, levocardia, normally related great arteries, normal systemic and pulmonary venous drainage, good biventricular function (EF ~60%), normal valves and pericardium, normal coronary artery origins, intact interatrial and interventricular septa, and no evidence of PDA, CoA, or pulmonary arterial hypertension. Ultrasonography demonstrated liver 8.6 cm (WNL for age), spleen 6.7 cm (WNL for age), both kidneys normal in size and echogenicity with no hydronephrosis, GB partly distended with no calculi, IVC 10 mm, no ascites, no intra-abdominal lymphadenopathy.

2.NGS Microbial Identification

2.1 Platform and Methodology

Peripheral blood sample was collected on 03/06/2026 and processed using the Infexn metagenomic NGS platform (HaystackAnalytics Private Limited, H-22, 23 Akshar Business Park, Navi Mumbai — 400703). The platform employs targeted metagenomic sequencing for detection of pathogenic genomic signatures from bacteria, fungi, and neuropathic viruses (HSV1/HSV2). Analysis date: 04/06/2026. Sample ID: HAPL-SEQ22OZ5H40WN7QQ.

Table 4: NGS Microbial Identification Report —

Category	Finding	Laboratory Comment
Pathogen Detected	*Aspergillus waynelawii* (Fungi — Hyaline Mold)	Opportunistic/environmental pathogen. Possible contamination at sample collection to be considered.
Antibiotic Resistance Genes	All Not Detected (11 genes screened)	blaCTX-M, blaOXA-1/48/23, aph(3'), mecA, blaNDM, blaTEM, blaSHV, VanA, mcr-1 — all negative
Viral Screen	Not detected (HSV1/HSV2)	Herpes simplex viruses not detected
Sample Type	Blood — Peripheral	Central line sample caveat explicitly noted by lab

Key Findings

The laboratory report included the following critical interpretive footnote: *"Probable/proven opportunistic pathogen in a susceptible host. Host risk factors inclusive of but not limited to extremes of age, multiple comorbidities, infective endocarditis, in-situ invasive devices, prolonged hospitalization, invasive procedures & immunosuppression. Likelihood of opportunistic infection or possible contamination with skin and/or environmental flora at sample collection to be considered. Sample drawn from central line are likely to be colonised with skin and/or environmental flora. Isolated central line samples are not proof of bloodstream infection."*

3. Bone Marrow Examination (04/06/2026 — Reported 07/06/2026)

In view of the persistent and severe pancytopenia, bone marrow aspirate (ASBM: 611/26) and trephine biopsy (Biopsy No. 1965/26) were performed under sedation on 04/06/2026, reported by Dr. Vinukumar (MBBS, MD Pathology, Pdip MD Path, DBM, IFCAP, Consultant Pathologist, NIMS Medicity) on 07/06/2026.

3.1 Peripheral Blood Smear Findings

- **RBC:** Normocytic and normochromic; few polychromatophilic cells; no nucleated RBCs, hemoparasites, or inclusions
- **WBC:** Count moderately reduced; differential count shows relative lymphocytosis; ***no immature cells or blasts identified***
- **Platelets:** Markedly reduced, scattered singly; few large forms
- **PBS Impression:** Normocytic normochromic anaemia with moderate leukopenia, relative lymphocytosis, and marked thrombocytopenia — amounting to Pancytopenia

3.2 Bone Marrow Aspirate Differential Count (n = 500)

Table 5: Bone Marrow Aspirate Differential Count

Cell Lineage	Percentage (%)	Morphological Detail
Blasts	50% ← DIAGNOSTIC	Anisonucleosis, increased N:C ratio, condensed chromatin, scanty cytoplasm, indistinct 1–2 nucleoli
Lymphocytes	42%	Mature morphology
Prolymphocytes	2%	—
Erythroblasts	5%	Normoblastic maturation; NO dyserythropoiesis
Neutrophils	1%	Markedly reduced
Megakaryocytes	Markedly reduced	Morphology normal where present

3.3 Bone Marrow Biopsy Microscopy

Sections showed predominantly hyaline cartilage and bony tissue. Occasional marrow spaces demonstrated sheets of mature lymphocytes and aggregates of medium-sized cells with open chromatin, nucleomegaly, and scanty cytoplasm. Erythroid cells, myeloid cells, and megakaryocytes were markedly reduced.

3.4 Final Histopathological Diagnosis

Bone Marrow Aspirate: Suggestive of Acute Leukaemia, possibly Acute Lymphoblastic Leukaemia (ALL) — L2 subtype, FAB classification.

Bone Marrow Biopsy: Histomorphological features favour Hematolymphoid Neoplasm, possibly Acute Leukaemia.

Pathologist's Comment: *Cytochemistry, flow cytometry, and molecular study advised for confirmation and risk stratification. Reference ASBM 611/26.*

4. Clinical Management

4.1 Antifungal Therapy — Voriconazole

Following review of the NGS report identifying *Aspergillus waynelawii* in a child with ANC 250/µL, empirical antifungal therapy with **voriconazole** was initiated. The decision was guided by the following clinical reasoning:

1. **Severe neutropenia (ANC < 500/µL):** This threshold defines high-risk status for invasive fungal infection (IFI) per IDSA and ESCMID guidelines. With ANC at 250/µL, the child met criteria for empirical antifungal therapy even in the absence of a confirmed fungal diagnosis.
2. **NGS detection of Aspergillus species:** While *A. waynelawii* is an environmental organism, its NGS detection in a neutropenic PICU patient — regardless of contamination probability — warranted clinical action given the potential consequences of undertreating invasive aspergillosis.
3. **Voriconazole as first-line agent:** Per IDSA 2016 guidelines (Patterson et al.), voriconazole is the primary agent of choice for invasive aspergillosis in paediatric patients, demonstrating superior survival outcomes compared to amphotericin B in randomised controlled trials. Voriconazole has broad-spectrum activity covering the majority of clinically relevant *Aspergillus* species including members of section Usti.
4. **Risk-benefit balance:** In a severely immunocompromised child in the PICU with an NGS-positive fungal signal, the risk of withholding antifungal therapy was judged to outweigh the risks of empirical treatment. Liver function tests (ALT 62/AST 54, mildly elevated) were noted as a baseline parameter to monitor for voriconazole hepatotoxicity.

Voriconazole was prescribed at standard paediatric dosing (loading dose 9 mg/kg IV q12h × 2 doses, followed by maintenance 8 mg/kg IV q12h for children aged 2–11 years per weight-based paediatric pharmacokinetics). Liver function monitoring and potential therapeutic drug monitoring (TDM) for voriconazole trough levels were planned as part of ongoing management.

4.2 Transfusion Support Strategy

Given the severe anaemia (Hb 4.3 g/dL) with compensatory tachycardia and the diagnosis of ALL requiring pre-chemotherapy stabilisation, a structured transfusion programme was implemented:

Table 6: Transfusion History During Admission

Event	Product	Clinical Indication	Target / Outcome

pRBC #1	Packed RBC (5 mL/kg over 4h)	Hb 4.3 g/dL, HR 128 bpm, severe symptomatic anaemia	Hb target >7 g/dL; ECHO confirmed safe cardiac reserve
pRBC #2	Packed RBC	Persistent symptomatic anaemia, pre-BM biopsy stabilisation	Optimise Hb before invasive procedure
pRBC #3	Packed RBC	Post-BM biopsy; Hb 5.6 g/dL on 03/06/2026	Sustain Hb ahead of referral to RCC
Platelet #1	Single Donor / Random Donor Platelets	Plt 40,000; thrombocytopenic risk during invasive procedure	Plt threshold >50,000 for BM biopsy
Platelet #2	Single Donor / Random Donor Platelets	Plt 45,000; ongoing thrombocytopenia pre-transfer	Minimise bleeding risk during transport and handover

All blood products were leucodepleted and CMV-negative where possible. The child was monitored for transfusion reactions throughout each transfusion. Furosemide cover was administered with each pRBC transfusion given the tachycardia and underlying haematological compromise.

4.3 Supportive and Prophylactic Measures

- **Neutropenic precautions:** Reverse barrier nursing, HEPA-filtered air where available, restricted raw food, strict hand hygiene protocols, and visitor restriction implemented on admission
- **Antibiotic prophylaxis:** Broad-spectrum empirical antibacterial coverage maintained throughout PICU stay given ANC 250/µL
- **Nutritional support:** High-calorie, high-protein diet initiated; nasogastric supplementation considered given decreased food intake
- **Monitoring:** Daily CBC, serial liver function (for voriconazole), electrolytes, and clinical assessment performed throughout admission

5. Referral to Regional Cancer Centre (RCC)

Following confirmation of ALL on bone marrow biopsy, haematological stabilisation, and initiation of empirical antifungal therapy, the child was referred to the Regional Cancer Centre (RCC), Thiruvananthapuram — a premier tertiary oncology centre

DISCUSSION

The case highlights the diagnostic difficulty posed by aleukemic acute lymphoblastic leukaemia (ALL). The child initially presented with severe normocytic pancytopenia, relative lymphocytosis, normal LDH and uric acid levels, negative viral studies, absence of organomegaly, and no peripheral blood blasts, strongly suggesting acquired aplastic anaemia. However, bone marrow examination demonstrated approximately 50% lymphoblast infiltration, establishing the diagnosis of ALL, characterized by substantial marrow blast burden despite a blast-free peripheral smear, is an uncommon but recognized presentation in paediatric leukaemia. This phenomenon may delay diagnosis and emphasizes the importance of early bone marrow evaluation in unexplained pancytopenia, even when peripheral smear findings are non-diagnostic.

Metagenomic next-generation sequencing (mNGS) offers broad pathogen detection but presents significant interpretive challenges because it identifies microbial nucleic acids irrespective of organism viability. Positive signals may represent active infection, colonization, environmental contamination, or laboratory contamination.

The detection of *Aspergillus waynelawii* in peripheral blood was interpreted cautiously. Aspergillus species are ubiquitous environmental fungi, and false-positive detections have been reported with plasma cell-free DNA sequencing. Furthermore, *Aspergillus waynelawii* is an exceedingly rare human pathogen, with no previously reported paediatric ALL-associated cases identified during literature review. In the absence of clinical, radiological, or microbiological evidence of invasive aspergillosis, contamination remained the most likely explanation.

Despite the low probability of true invasive aspergillosis, empirical voriconazole was initiated because the patient had profound neutropenia and impending immunosuppressive therapy. Invasive fungal infections in this setting carry substantial morbidity and mortality, whereas short-term voriconazole therapy is generally safe when appropriately monitored. The decision therefore reflected a risk-benefit approach consistent with haemato-oncology practice, prioritizing patient safety while further diagnostic evaluation was pending.

The patient required three packed red blood cell transfusions and two platelet transfusions before transfer. Red cell transfusion was guided by severe symptomatic anaemia, while platelet transfusion targeted procedural safety for bone marrow aspiration and biopsy. Leucodepleted blood products were used in anticipation of chemotherapy-related immunosuppression. Ongoing surveillance for transfusion-related iron overload remains warranted given repeated transfusion exposure and elevated baseline transferrin saturation.

This case illustrates several important roles for clinical pharmacists in paediatric haemato-oncology, including interpretation of complex diagnostic reports, antifungal stewardship, therapeutic drug monitoring of voriconazole, recognition of clinically significant drug interactions (particularly with vincristine-based chemotherapy), transfusion pharmacovigilance, and optimization of supportive care measures such as tumour lysis prophylaxis and infection prevention.

A major strength of this report is the comprehensive documentation of clinical, laboratory, imaging, mNGS, and bone marrow findings from a single admission, allowing detailed analysis of an unusual presentation of ALL. To our knowledge, this is the first report describing peripheral blood mNGS detection of *A. waynelawii* in a child with newly diagnosed ALL.

Limitations include the absence of galactomannan, β-D-glucan, and high-resolution CT chest studies prior to transfer, lack of voriconazole therapeutic drug monitoring, and unavailability of long-term outcome data. Flow cytometric immunophenotyping and molecular risk stratification results remain pending and will be essential for complete characterization of the disease.

CONCLUSION

This case report presents a 2-year 7-month-old male child in whom peripheral blood metagenomic NGS detected *Aspergillus waynelawii* — an environmental hyaline mold — in the context of severe neutropenia (ANC 250/μL), ultimately

resulting from newly diagnosed Acute Lymphoblastic Leukaemia (ALL) L2 subtype confirmed on bone marrow biopsy. The co-occurrence of a haematological emergency (ALL with aleukemic presentation) and an NGS-detected fungal signal created a complex, multi-layered diagnostic and therapeutic challenge.

The child was managed with empirical voriconazole — a clinically defensible decision in a severely neutropenic host with a positive fungal NGS signal in a resource-limited PICU setting — alongside three packed red cell transfusions and two platelet transfusions for haematological support. Following haematological stabilisation and diagnostic confirmation, the child was successfully transferred to the Regional Cancer Centre, Thiruvananthapuram, for definitive oncological management.

This case makes several original contributions to the literature: (1) it represents the first documented NGS detection of *A. waynelawii* in a paediatric haematology-oncology patient; (2) it illustrates the diagnostic pitfalls of aleukemic ALL mimicking aplastic anaemia across multiple laboratory parameters; (3) it provides a structured framework for clinical decision-making when NGS detects environmental fungi in neutropenic children; and (4) it offers a comprehensive clinical pharmacy teaching case integrating antifungal stewardship, transfusion medicine, and oncology pharmacology.

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