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# THE ENIGMA OF CHILDHOOD EPISTAXIS: A CASE SERIES

**Paediatrics**

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## ABSTRACT

Epistaxis is one of the frequently endured problems in pediatric outpatient and emergency departments. Childhood epistaxis is a compilation of a wide variety of local, systemic, and environmental factors. Most of the cases are benign and can be effortlessly diagnosed and treated. On the contrary, certain cases have rare presentations that are recognized by comprehensive clinical history taking and examination. Such elements should never be missed as they could signify serious disease patterns entailing prompt attention and thereby prevent fatality. We hereby proffer two such uncommon exhibits of childhood epistaxis. The first child presented to emergency department with severe epistaxis, diagnosed as Acute lymphoblastic leukemia (ALL) and the second case to the outpatient clinic with mild epistaxis and was diagnosed with Dengue Hemorrhagic Fever (DHF) with warning signs.

## KEYWORDS

Childhood epistaxis, systemic factors, benign, rare presentations, serious disease patterns, prompt attention, Acute lymphoblastic leukemia, Dengue Hemorrhagic Fever, warning signs.

## INTRODUCTION:

Epistaxis is one of the frequently endured problems in pediatric clinics. It affects 30% of the pediatric population aged under five, 56% of those aged between 6–10 and 64% of those aged between 11–15 years[1]. The mean age of presentation is usually 7-8 years of age [2] and is rare in children below 2 years age. While most of the cases are benign involving minimal intervention, few cases imply serious disease patterns that necessitate extensive workup.

Childhood epistaxis is a compilation of local, systemic, and environmental factors. While local factors generating epistaxis are trauma, infection, inflammation, and neoplasms; hypertension, coagulopathy, connective tissue disorders and hemorrhagic telangiectasias are included in differentials under systemic category. Temperature, humidity, and certain medications such as warfarin, aspirin, and nonsteroidal anti-inflammatory drugs constitute environmental elements of childhood epistaxis. In the current research from a rural health center, we spotlight two uncommon exhibits of childhood epistaxis with ALL and DHF with warning signs.

### Case-1:

An 8-year-old boy was accompanied by his mother to the pediatric emergency department with a chief complaint of severe bleeding from his right nostril for one hour, unresolving with manual pressure. He was immediately managed with anterior nasal packing and a hemostatic agent along with supportive therapy. (Figure-1)

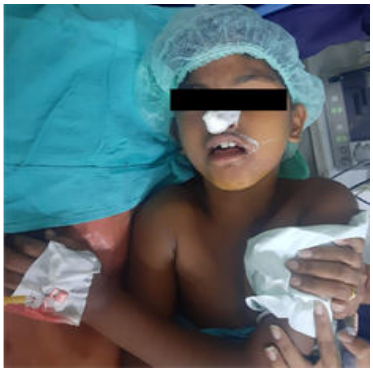


**Figure1:** Anterior nasal packing in an 8-year-old boy with epistaxis.

The boy was apparently normal 8 weeks ago. Initially, his mother noticed recurrent severe nasal bleeds associated with easy bruising and low-grade fevers. His mother further added that he appeared paler and got tired very easily with loss of weight and appetite. Over the course, he often woke up crying at night with severe bone pains in his legs. There was no history of trauma, nasal stuffiness, cough, or jaundice. He also had no history of night sweats, gum bleeding or any drug intake. The boy was treated for nasal bleed multiple times in the past, which required occasional nasal packing. He had no significant family or allergic history.

He was born out of a non-consanguineous marriage and was first in birth order. There were no significant elicitations in his antenatal, natal, and postnatal records. His development was normal in all domains and was immunized according to the national immunization schedule. He had a total of 400 calories and 13 grams protein deficit in his daily nutrition.

Upon general examination, he was conscious and coherent. He had pallor with generalized lymphadenopathy. His head-to-toe assessment displayed no facial dysmorphology but several ecchymotic patches over his back and legs. He is febrile ($100.4^0$F) with a heart rate of 90 beats per minute, blood pressure of 94/62 mmHg and a respiratory rate of 20 per minute.

He maintained 97-98% saturation on room air. His weight was at 3rd percentile and height between 10-25th percentile with a BMI (body mass index) below 3rd percentile (underweight). Local examination of the nose revealed profuse bleeding from the right nostril, trickling into the oropharynx.

His systemic examination revealed bone tenderness in both lower limbs and mild hepato-splenomegaly.

Based on his clinical presentation, a provisional diagnosis of bleeding disorders and hematological malignancy were anticipated, and a workup is planned accordingly.

Results of the complete blood picture projected severe anemia and marked leukocytosis with lymphocytosis (Table-1). Owing to an unremarkable coagulation profile, bleeding disorders were ruled out and further investigations headed in the light of hematological malignancy.

**Table 1:** Results of complete blood picture.

| PARAMETER | VALUE | REFERENCE RANGE |
|---|---|---|
| Hemoglobin (g/dL) | 7.0 | 12-16 |
| RBC Count (106/µL) | 3 | 3.5-5 |
| Hematocrit (%) | 22 | 35-49 |
| WBC Count (103/µL) | 223.5 | 4-11 |
| Neutrophil % | 8 | 55-70% |
| Lymphocyte % | 76 | 20-35% |
| Eosinophil % | 1 | 1-3% |
| Monocyte % | 15 | 3-8% |
| Basophil % | 0 | 0-1% |
| Platelets (103/µL) | 10 | 150-450 |
| MCV | 80 | 80-100 |
| MCH | 27 | 26-32 |
| MCHC | 33 | 32-36 |
| RDW (%) | 17 | 11.5-14.5 |
| RBC: Red blood cell, WBC: White blood cell<br>MCV: Mean corpuscular volume<br>MCH: Mean corpuscular hemoglobin<br>MCHC: Mean corpuscular hemoglobin concentration<br>RDW: Red cell distribution width | | |

The peripheral blood smear pictured anisopoikilocytosis, normocytic normochromic anemia, with lymphoblasts (2-3 times the RBC size, with high N/C ratio, scant cytoplasm, hyperchromatic nucleus with few cells showing nucleoli) and thrombocytopenia, impressing ALL (Figure-2)

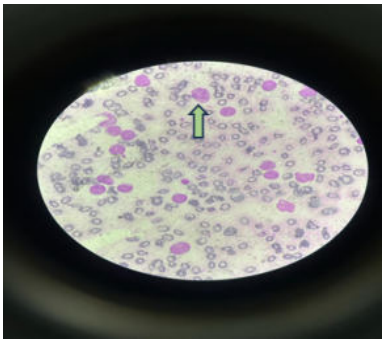


**Figure2**- Peripheral blood smear showing lymphoblasts (marked by green arrow).

The outcome of Bone marrow aspiration was a hypercellular marrow with reduced erythropoiesis, granulopoiesis, and megakaryopoiesis. The marrow represented an increase in lymphoid population with 60% lymphoblasts, findings affirming ALL.

The child was diagnosed with ALL and was referred to haemato-oncology for further management.

**Case-2:**

A previously healthy 11-year-old boy visited the pediatric outpatient clinic with a chief complaint of mild, intermittent nose bleeds for 5 days and high-grade fever ($103^0$F) associated with chills and rigors and generalized rash for 4 days. He had a history of mild arthralgias and retro orbital pain. The adolescent totted that he had mild epigastric pain with black colored stools and few episodes of non-bilious, non-projectile vomitings during the illness. He denied a history of altered consciousness, shortness of breath, gum bleeds, recent travel, animal exposure or allergies.

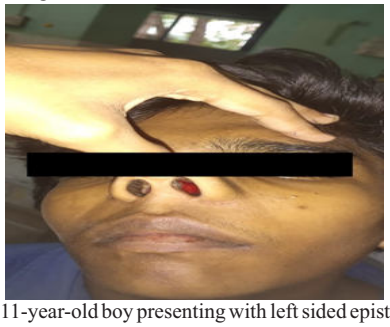


**Figure 3-** A 11-year-old boy presenting with left sided epistaxis.

There was no history of similar illness in the past. He was immunized as per the schedule. His growth and development were normal with alignment of secondary sexual characters to Tanner stage 1.

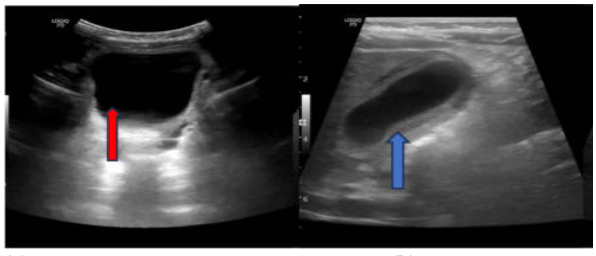
On general examination, he was conscious, coherent and oriented to time, place and person. He had mild pallor. His vitals demonstrated a temperature of $102^0$F, heart rate of 120 beats per minute, blood pressure of 100/60 mm Hg and respiratory rate of 18 per minute. His room air oxygen saturation was 98-99%.

There was mild bleeding in his left nostril. There was a generalized, erythematous rash on his body with several petechiae in cubital fossa (after tourniquet test), purpura in both lower limbs with warm peripheries and three large ecchymosis on his back. He had epigastric tenderness with mild hepatomegaly on his abdominal examination. Rest of his systemic examination was normal.

The boy's clinical profile is conspicuous with viral hemorrhagic fevers and therefore we delved appropriately.

His hemogram manifested mild anemia (Hb-10g/dL), leukopenia ($3x10^9$/L), thrombocytopenia (25,000/µL) and a low hematocrit (30). The liver function panel notified mild elevation of transaminases (AST 380 U/I and ALT 300 U/I). In addition, serology was positive for NS1 (Non-structural protein) and IgM, specific for Dengue, confirming DHF.

Ultrasound (USG) abdomen was performed, illustrating mild-moderate ascites and gallbladder wall oedema, unfolding DHF with warning signs. (Figure-4)



**(a)** **(b)**

**Figure-4**- USG abdomen in an 11-year-old boy with DHF illustrating (a) mild to moderate ascites (pointed by red arrow) and (b) gallbladder wall oedema (pointed by blue arrow).

The boy was managed with acetaminophen (15mg/kg/dose) on as needed basis with normal saline maintenance.

On day 2 of admission, his platelet count dropped to 15,000/µL with hematocrit increasing to 35. In view of DHF with warning signs, he was immediately transferred to PICU and was administered 3 units of platelets along with ringer lactate fluid therapy. He was put on daily check of platelets, hematocrit, and bleeding manifestations. (Table-2)

**Table-2:** Platelets and hematocrit during the hospital course

| DAY OF ADMISSION | PLATELETS (/µL) | HEMATOCRIT (%) |
|---|---|---|
| DAY 1 | 25,000 | 30 |
| DAY 2 | 15,000 | 35 |
| DAY 3 | 10,000 | 40 |
| DAY 4 | 25,000 | 38 |
| DAY 5 | 50,000 | 37 |
| DAY 6 | 90,000 | 36 |
| DAY 7 | 1,20,000 | 34 |

However, on day 3 of admission, his platelet counts further dropped to 10,000/µL with further rise in hematocrit to 40. He was again repeated 3 units of platelets and continual fluid therapy, monitoring blood counts and bleeding manifestations.

The patient began to show clinical improvement from day 4. His fever spikes reduced with a gradual incline in his platelet counts and hematocrit decline. His fluids were gradually tapered. On day 8 of admission, he had no bleeding manifestations, and his blood counts were normalized. He was then discharged and put on follow up.

**DISCUSSION:**

The current research from a community health center portrayed two uncommon exhibits of childhood epistaxis. In the former case, the boy

demonstrated bleeding manifestations and systemic symptoms for 8 weeks, which appeared unusual, warranting further evaluation for potential bleeding disorders and hematological malignancy. However, the compressive analysis of case study reflected ALL.

ALL accounts for 80% of childhood leukemias and is one of the commonest childhood malignancies under 15 years [3]. The clinical presentation in children with ALL include bleeding manifestations (in our case epistaxis and skin bruising), bone pains, fever, infections due to cytopenias, hepatosplenomegaly, central nervous system and testicular infiltration, easy fatigability, weight loss and lymphadenopathies [4,5]. Defects in bone marrow can lead to inadequate production of cell lineages and abnormal cancer cell infiltration that accounts for wide-spread symptomatology [4,5]. Anemia in ALL could be a consequence of inadequate production of erythrocytes, or due to bleeding secondary to thrombocytopenia [6,7].

The presence of 20% or more lymphoblasts in the bone marrow or peripheral blood confirms the diagnosis of ALL [8], which in our case, showed 60% of blast cells in the marrow. Moreover, his hemogram showed, Hb 7.0 g/dL, WBC count 2,23,500/µL with lymphocytosis (76%) and platelets 10,000/µL. In total, by meticulous clinical history, examination and lab work, a diagnosis as ALL was accomplished from an uncommon manifestation. Later, the child was allocated to the hemato-oncology wing for further management.

In contrast, the latter case presented with epistaxis, fever, generalized rash, arthralgias and retro orbital pain for 5 days. Considering the likelihood of viral hemorrhagic fevers as a causation for epistaxis, the diagnostic progress depicted DHF with warning signs.

Dengue is a self-limiting disease in most children requiring minimal supportive intervention. However, in less than 1%, symptoms of severe dengue include plasma leak, fluid accumulation and dengue shock syndrome (DSS), causing death if left untreated.

In this regard, 2009 WHO classification intends physicians for classifying dengue cases based on presentations (Table 3 and 4), early identification of warning signs [9] and thereby halt progression to severe dengue and hence mortality.

**Table-3:** Dengue fever with warning signs

| S.No | WARNING SIGNS* |
|---|---|
| 1. | Abdominal Pain and tenderness |
| 2. | Persistent Vomitings |
| 3. | Clinical fluid accumulation |
| 4. | Mucosal bleeds |
| 5. | Lethargy and restlessness |
| 6. | Increase in hematocrit, concurrent with rapid decrease in platelet count |
| * | Require strict observation and medical intervention |

**Table-4:** Criteria for severe dengue

| S.No | CRITERIA FOR SEVERE DENGUE |
|---|---|
| 1. | Severe Plasma leakage (leading to DSS and fluid accumulation with respiratory distress) |
| 2. | Severe bleeding |
| 3. | Severe organ involvement evidenced by:<br>Liver AST or ALT ≥ 1000<br>CNS: impaired consciousness<br>Heart and other organs |

A diagnosis of DSS is labeled in patients with signs of hypotension or narrow pulse pressure (less than 20 mm Hg).

In our case, the adolescent had 5 out of 6 listed warning signs that required strict attention in the PICU. Nevertheless, early identification of these warning signs by careful history and examination aided in early intervention, that breached the progression into DSS and thereby mortality.

## CONCLUSION:

In the current research, careful history and clinical examination clinched a crucial lead in early recognition of serious disease patterns of childhood epistaxis. This favored early therapeutic intervention and thereby prevented possible complications. This study hence concludes that even though the majority of cases with childhood epistaxis have benign elements, rare presentations recognized in clinical history taking and examination, should never be missed as they could signify serious disease that needs prompt attention, thereby averting fatality.

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