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CASE REPORT

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Acute Lymphoblastic Leukaemia Presenting as Pleural and Pericardial Effusions: A Case Report

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Abstract
The most common presentations of Acute Lymphoblastic Leukaemia (ALL) are related to intramedullary symptoms, but extramedullary symptoms may also occur. ALL presenting as pleural effusion is very uncommon, and pericardial effusions are rarer. There is a need for a high index of suspicion in the presence of unusual symptoms to prevent delayed diagnosis and poorer outcomes. We report on the case of a nine-year old boy who presented with fever and cough of three weeks and had haemorrhagic bilateral pleural and pericardial effusions. Laboratory screenings for Lassa fever, tuberculosis, and sepsis were negative. Pericardiocentesis and closed thoracotomy continued to drain. Several full blood counts (CBC) at presentation and during admission were normal, but a repeat on day 23 of admission showed marked leucocytosis. ALL was confirmed with bone marrow aspirate cytology. Effusions resolved when chemotherapy was commenced. ALL can masquerade with features of common conditions, resulting in delayed diagnosis and poorer patient outcomes. A high index of suspicion is necessary when unusual findings are observed.

Keywords: Bone marrow failure, Chemotherapy, Dyspnoea, Myeloproliferative disorder, Serous effusions.

Introduction

Acute leukaemia is the commonest cancer among children, representing about 25% of all childhood cancers worldwide,[1 - 3] and acute lymphocytic leukaemia (ALL) accounts for approximately 75% of childhood acute leukaemia.[3]

Acute lymphoblastic leukaemia affects all organs and tissues of the body, but the most common symptoms are due to bone marrow failure and include leucopaenia, anaemia, and thrombocytopaenia.[1, 2, 4] Patients usually present with symptoms related to medullary involvement, including bleeding, petechiae, purpura, fatigue, anorexia, malaise, bone pain (usually long bones), pallor, and fever. [2, 4]

While leukaemia may manifest with extramedullary symptoms, it rarely causes malignant serous effusions as the first manifestation.[1] Pleural effusions are common in nearly all haematological malignancies during the course of the disease, and most frequently occur with worsening illness, particularly in Hodgkin's and non-Hodgkin's lymphomas,[1, 4,] but they are more likely to be benign than malignant.[2] Pleural effusion in leukaemia patients is often the result of underlying infections,

secondary malignancy, volume overload, or the toxicity of chemotherapy.[1]

Although pericardial effusion is rare in childhood acute leukaemia, when it occurs, it is more likely to be benign than malignant.[1, 2] Only a few studies have documented it as a presenting feature.[1, 2, 5] The objective of this report is to highlight some of the unusual presentations that may suggest an underlying malignancy in children.

Case presentation

A nine-year old boy presented with a three-week history of chest pain, cough, dyspnoea, and fever. He was lethargic and unable to sit without support. He was only able to eat small quantities of liquid foods at a time. He had no peripheral lymphadenopathy, but had dyspnoea and tachypnoea, with right sided tracheal deviation. Percussion notes were stony dull with reduced breath sounds on the left hemithorax, and crepitations on the mid and lower lung zones. He also had marked hepatosplenomegaly. The initial diagnostic considerations included Lassa fever, pneumonia complicated by pleural effusion, to exclude pulmonary tuberculosis.

Chest X-ray done at admission showed features of pneumonia with bilateral pleural effusion (Figure 1). A chest CT scan also showed a marked pericardial effusion with thickened pericardium (confirmed by an echocardiography), massive right pleural effusion with collapsed mid and lower lung zones, widespread infiltrates with ground glass appearance involving the left lung field, and an anterior mediastinum fullness with unenhanced soft tissue density. Blood culture was negative, and screening tests for Lassa fever disease and tuberculosis (using Mantoux test and Gene Xpert) were negative. A pericardial biopsy reported on the 22nd day of admission showed he had a low grade lymphoma (Figures 2 – 4), and although his initial full blood counts were within a normal range, the counts done on the 23rd day of admission showed hyperleukocytosis of 141,000/mm^3, with a lymphocyte predominance (83.3%) and neutropaenia of 10.5% (Table I). Other parameters included a platelet count of 241,000/mm^3 and a PCV of 31%. ALL was confirmed by a bone marrow aspiration cytology (Figures 5).

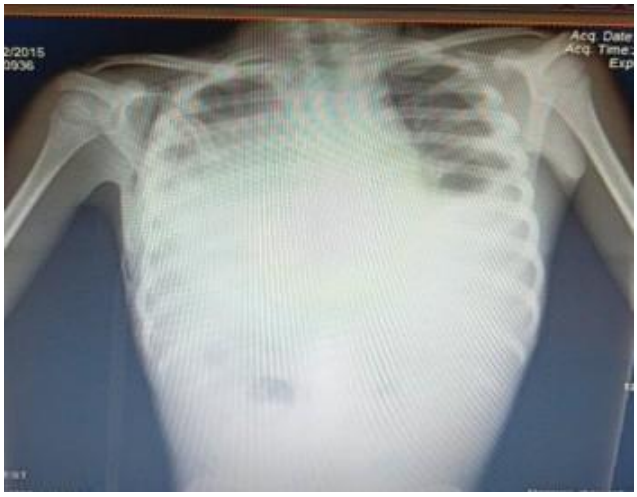

Figure 1: Chest radiograph at presentation, showing marked bilateral pleural effusion and pneumonic changes

There is a homogenous opacity with lateral meniscus in keeping with pleural effusion involving the lower and middle lung zones bilaterally, more marked on the left, obliterating the costophrenic and cardio-phrenic angles, as well as obscuring the hemidiaphragms and cardiac borders. Hence, the heart size cannot be objectively assessed. Contiguous inhomogeneous infiltrates are noted, more marked on the left. There is a chest tube with its tip towards the base of the left lung field. There is a lucency within the soft tissue suggestive of subcutaneous emphysema around the point of entry of the chest tube.

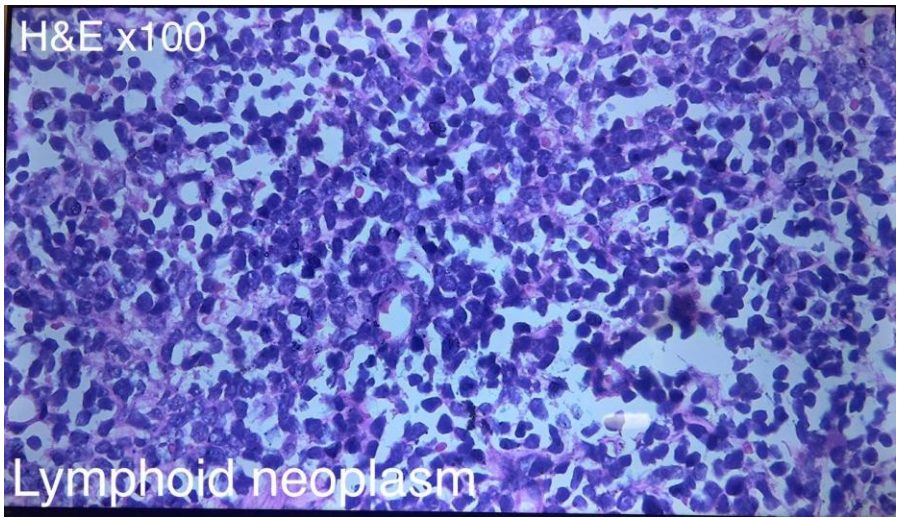

Figure 2: Pericardial tissue showing lymphoid hyperplasia

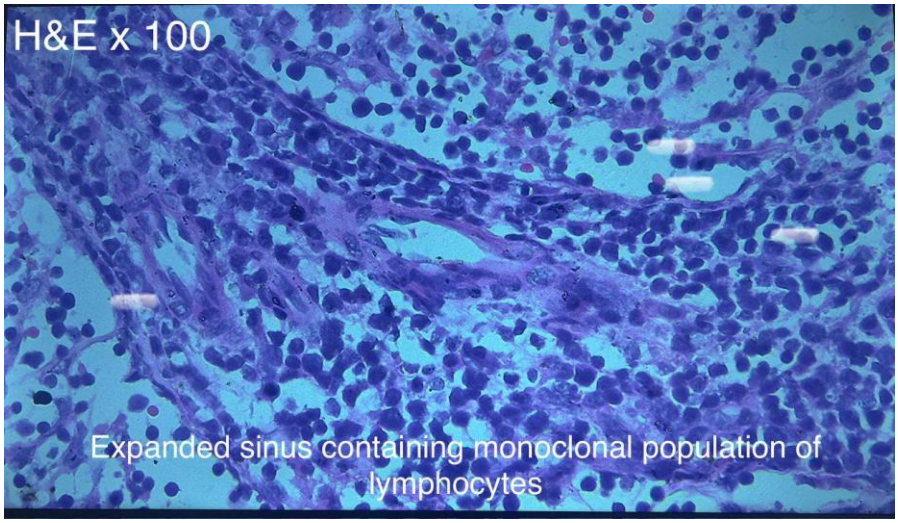

Figure 3: Pericardial tissue showing a monoclonal population of lymphocytes

Histologic sections from pericardial tissue show a neoplastic lymphoid lesion composed of diffuse proliferation of medium to large lymphocytes having round nuclei with scant cytoplasm. Foci of hyperplastic lymphoid follicles having reactive germinal centres are visualised. This nodular lesion is surrounded by desmoplastic connective tissue, which is markedly infiltrated by chronic inflammatory cells. Within the stroma, clusters of atypical cells are seen within blood vessels. These cells have hyperchromatic nuclei and scant cytoplasm. Histopathologic diagnosis: Low-grade lymphoid neoplasm with background para-cortical lymphoid hyperplasia.

The child had a bilateral closed thoracotomy tube inserted on the day of presentation. At the same time, pericardiocentesis was done with a pericardial drainage bag *in-situ* on the fourth day (Day 4) of admission following chest CT scan confirmation of pericardial effusion. The chest CT scan was done on account of persistent tachypnoea and dyspnoea. The drainages remained active until a day after chemotherapy was commenced (Figure 6).

Other components of the management included oxygen therapy and intravenous antibiotics (meropenem and levofloxacin) at presentation. He was dependent on oxygen, remained dyspnoeic and tachypnoeic, and lethargic; he also continued to tolerate only liquid foods, but all symptoms resolved within 12 hours of commencement of chemotherapy. Although he had a packed cell volume (PCV) of 25%, he was commenced on chemotherapy on Day 26 as blood

was not immediately available for transfusion and he had worsening dyspnoea and hypoxia (oxygen saturation of 88%). The next day, the dyspnoea had resolved, but he developed bilateral pitting pedal oedema and severe anaemia with a PCV of 13%. He was transfused with packed red cells, and the oedema, thereafter, resolved completely. He did well on standard-risk induction-remission chemotherapy (oral prednisolone, intravenous vincristine, etoposide, L-asparaginase, intrathecal methotrexate), which lasted 28 days, and was discharged home after remission was confirmed by a repeat bone marrow aspiration cytology on Day 29 (Figures 7 and 8). With the mother's permission, intravenous doxorubicin was substituted with intravenous etoposide because of the risk of irreversible cardiac toxicity with doxorubicin.

Table I: FBC findings during hospital care

FBC Component	Day 2	Day 23	Day 28	Day 30	Day 37	Day 121
PCV %	36.3	31	27.9	24.8	28.4	31.4
Platelet count	504,000	241,000	166,000	17,600	125,000	197,000
TWBC (Cells/mm^3)	4,100	141,000	291,000	118,000	6,000	3,700
Neutrophil (%)	21.3	10.5	8.4	27.5	85.8	66.4
Neutrophil (Cells/mm^3)	873	14,805	24,452	32,450	5,148	2,457
Lymphocyte (%)	64.6	83.3	87.1	69.4	6.4	18.5

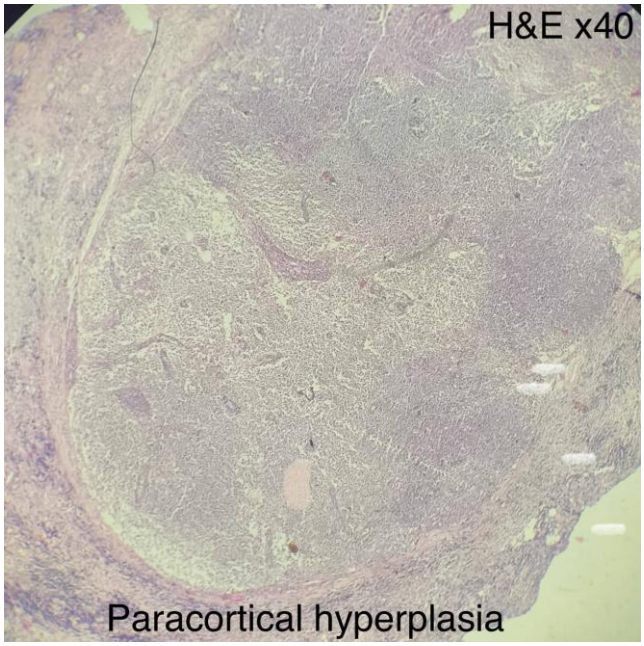

Figure 4: Pericardial tissue showing paracortical hyperplasia – the proliferation of lymphoid tissue, which may be reactive or malignant in nature

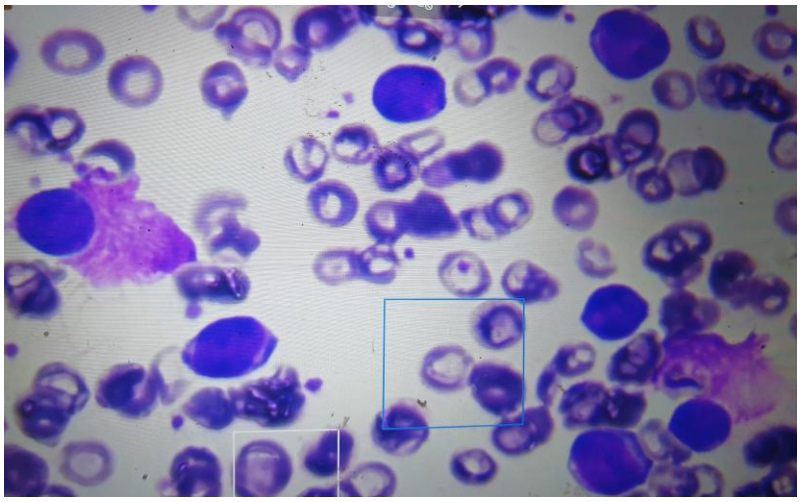

Figure 5: Peripheral Blood Film (PBF) on Day 23 (High power X100 Objective), showing numerous lymphoblasts.

These lymphoblasts are greater than 30% of all cells; they are medium-sized with open nuclear chromatin and high nuclear-cytoplasmic ratio. Also seen on this slide are hypochromic red blood cells and few platelets.

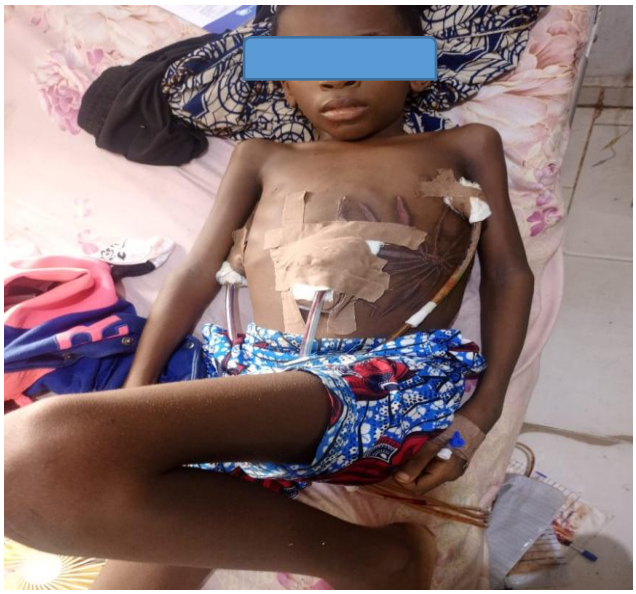

Figure 6: The child undergoing closed chest tube thoracostomy drainage and pericardiocentesis

Discussion

ALL typically presents with bone marrow failure symptoms, including bleeding, petechiae, purpura, fatigue, anorexia, malaise, bone pain, pallor, and fever. [2, 6] Extramedullary features from organ infiltrations may occur throughout the body and include painless lymphadenopathies, hepatosplenomegaly, and central nervous system (CNS), skin, and orbital involvement.[2,4,6] It also includes testicular swellings and musculoskeletal features.[1, 2]

Acute leukaemias rarely present with serous effusions, particularly as the initial presentation.[1] There has been only a few reported cases of pleuropericardial effusions in patients with acute leukaemias.[1] Pleural effusions are a common feature of nearly all haematological malignancies during the course of the disease. The effusions

mostly occur with worsening illness and only sporadically occur as the initial presentation.[4, 6] In addition, acute leukaemias rarely present initially with pleural effusion and bilateral massive leukaemic pleural effusion is particularly rare as an initial presentation.[1, 6 - 8] Indeed leukaemia-related pleural involvement is more frequently discovered at autopsies than *ante-mortem*. [4, 6] Malignant pleural effusions may suggest a poor prognosis, hence, a timely diagnosis and institution of appropriate management is of utmost importance.[7]

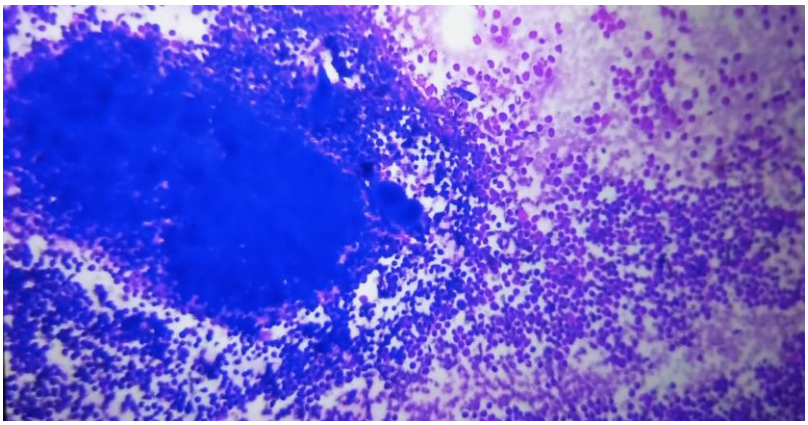

Figure 7: Bone Marrow Cytology on Day 23 (Low power X4 Objective)

This slide shows infiltration of the bone marrow by numerous lymphoblasts, greater than 40% of the total bone marrow nucleated cells. The diagnosis of ALL is made when bone marrow blasts are greater than 20%.

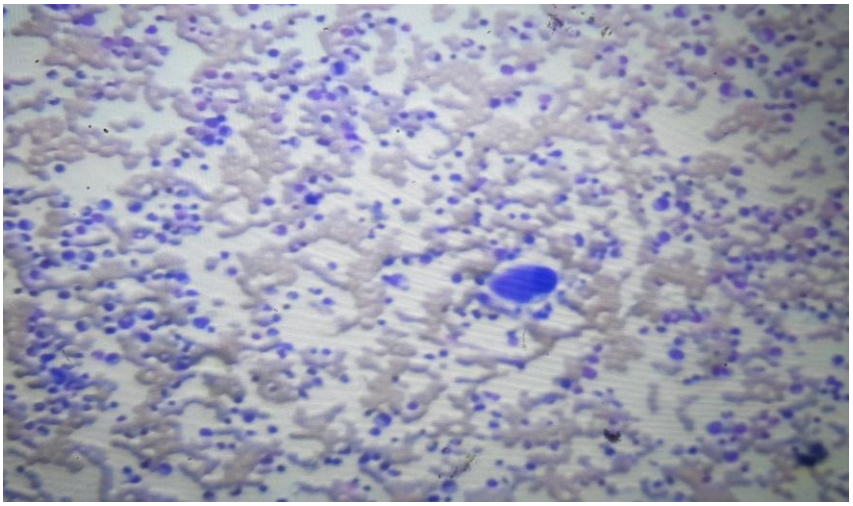

Figure 8: Bone Marrow Cytology following disease remission (Low power X4 Objective)

The slide shows evidence of megakaryocytes, sequential maturation of myeloid cells and mature-looking lymphocytes.

Pericardial effusion is not very common in childhood acute leukaemia, and it is often discovered after the diagnosis of leukaemia.[1, 7] Occasionally, it may be an initial indication of the disease.[9] Acute cardiac presentations are extremely rare,[9] with few studies reporting pericardial involvement as the initial manifestation of ALL.[1, 2, 5, 9] Massive pericardial effusion or cardiac tamponade is more common as the first manifestation in T-ALL than B-ALL.[1] Literature had previously reported a case of T-ALL presenting with pleuropericardial effusion.[1]

The diagnosis of ALL in the index patient was delayed due to the unusual presentations, the initial normal full blood count and the need to rule out other conditions (particularly complicated pneumonia, tuberculosis and Lassa fever disease) because of the presence of pleural and cardiac effusions. The pleuropericardial effusion resolved following treatment. Although not histologically confirmed in the index patient, the diagnosis of ALL may be made through a pericardial biopsy.[10] In the pathogenesis of ALL, an infection has been suggested to lead to the development of the myeloproliferative disorder due to the second hit phenomenon.[11] Also, an earlier report of a similar white blood cell count pattern had postulated that leukaemic cells may have produced granulocyte colony stimulating factor (G-CSF) like stimulants to drive the initial leucocytosis.[12] The initial normal WBC counts may have resulted from the time interval between the proliferation of immature lymphoid precursors, the replacement of the normal cells of the bone marrow and the time blast cells spill into the peripheral circulation.[13] The rapid drop in haemoglobin levels following the commencement of induction chemotherapy may have resulted from chemotherapy-induced haemolytic anaemia, and cancer related microangiopathic anaemia, considering the subsequent associated thrombocytopenia. [14, 15]

Conclusion

There is a need to consider rare conditions when clinical findings are unusual. This is necessary to prevent delayed diagnosis and enhance better patient outcome.

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Ethical considerations: Consent was obtained from the parents of the child and ethical approval was obtained from the Ethics committee of Irrua Specialist Teaching Hospital.

Conflicts of Interest: None declared.

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