Research Article

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A Male Child's Coexistence of Lupus Erythematosus and Acute Lymphoblastic Leukemia: An Extremely Rare Association.

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ABSTRACT

Seldom is there research for the pediatric age range describing the correlation between acute lymphoblastic leukemia (ALL) and systemic lupus erythematosus (SLE). Typically, lymphoproliferative illness develops after SLE, although The cancer may develop sooner or even at the same time. We present the case of a 15-year-old boy youngster who was diagnosed with SLE 12 years ago and was unintentionally diagnosed with ALL during a regular investigation.

SLE is an inflammatory illness that affects multiple systems and is characterized by inflammation of the connective tissue and blood vessels. There is great variation in the clinical symptoms. 10% to 20% of patients are thought to have been diagnosed with SLE before reaching adulthood. Even in the prepubescent age range, girls are more likely than boys to experience childhood SLE (8:1). Typically, childhood SLE is more severe.

Compared to adult SLE, SLE is typically more severe and has a worse prognosis. Antinuclear antibodies are a defining feature of sickle cell disease (SLE). There are very few cases of juvenile children with SLE and acute lymphoblastic leukemia (ALL) documented in the literature. Although the neoplasia can develop earlier or even concurrently with lymphoproliferative illness, SLE typically occurs before the latter.

Keywords
Systemic lupus erythematosus; Acute lymphoblastic leukemia; Anti-double stranded DNA.

INTRODUCTION

A complete blood count revealed elevated TLC in a 15-year-old male boy who was a known case of lupus nephritis during a routine follow-up. Acute lymphoblastic leukemia is suggested by peripheral blood film analysis (Figure 1. 60% of the blasts identified by flow cytometry analysis were positive for CD79a+, CD45+, CD34+, HLADR+, CD20+, CD38+, CD19+, CD10+, and CD22 HTG +, whereas CD13,33,3, and MPO were negative. Fluocytometry points to acute lymphoblastic leukemia in B cells. The child's karyotype was 46XY, and no hypo- nor hyperdiploidy was observed (Figure 2). In all the metaphases, the Philadelphia chromosome was not visible. No indication of t(15:17), t(8:21), or inversion 16 was found. At five years old, the sibling was likewise diagnosed with ALL. A youngster developed twelve years ago.

The results of the clinical assessment were negative. Following a kidney biopsy, the glomeruli exhibit mesenchymal growth along with an increase in segmental mesenchymal cellularity. thickening of the basement membrane. Medially, blood vessels exhibit a slight thickening. Focal space inflammatory infiltration is visible in the interstitial.

The glomeruli did not exhibit crescent development, tuft necrosis, segmental or widespread proliferation. The histology that has been discussed points to lupus nephitis (WHO class II). Both anti-double stranded DNA (anti-dsDNA) and anti-nuclear antibody (ANA) tested positive. The patient had been taking prednisone for SLE from the beginning, but six months ago the medication was discontinued due to illness considerations.

DISCUSSION

SLE, or systemic lupus erythematosus, is an autoimmune unclear place of origin. Inadvertently attacking its own tissues, the immune system leads to inflammation (swelling) of the skin, kidneys, lungs, nervous system, and other organs [1]. Even though it happens occasionally SLE can occur at any age, however it becomes more common at the age of five and after the first ten years of life [2]. Children are affected by SLE in the same manner as adults are. The primary distinction is in how adults and children are cared for, as a child's growth and development can be significantly impacted by their medical and psychological needs. While late damage, such as infections, atherosclerosis, and cancers, is typically connected to problems

of long-term disease, early damage is mostly associated to illness had a 1.7-fold increased risk of leukemia [4]. Among a global cohort of 9,547 SLE patients, Bernatsky et al. found an elevated risk of all hematologic malignancies. Just a small percentage of people with Concurrent SLE and coexisting chronic lymphatic leukemia/chronic myeloid leukemia have been documented. Following the patients for eight years, the leukemia standardized incidence ratio was determined to be 1.89 (95% CI 0.76–3.88) [5]. Immunosuppressive medication exposure may raise the risk for hematological malignancies, especially five years after therapy ends.

Irreversible renal impairment is the primary cause of death in cases of SLE renal involvement, a feared consequence [6]. One of the best medications for reducing inflammation in lupus is prednisone, which must be taken continuously.

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