Terminal Deoxynucleotidyl Transferase–Positive Lymphoid Cells in Reactive Lymph Nodes From Children With Malignant Tumors

Incidence, Distribution Pattern, and Immunophenotype in 26 Patients

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Key Words: TdT; Terminal deoxynucleotidyl transferase; Precursor B cell; Lymph node

Abstract

The presence of terminal deoxynucleotidyl transferase (TdT)-positive lymphoid precursors in benign lymph nodes from children has been characterized insufficiently. By using single- and double-labeling immunohistochemical analysis, we examined the frequency, distribution, morphologic features, and immunophenotype of TdT-positive cells in benign lymph nodes from 26 consecutive pediatric patients (4 boys, 22 girls; age, 10 weeks-17 years; median, 4.5 years), 23 of whom had a history of malignant neoplasm. We identified TdT-positive lymphoid cells in all 26 cases. These cells were found adjacent to medullary and cortical sinuses, with a frequency of 1 to 180 cells per high-power field (median, 20 cells), and were present singly and in small clusters. They were morphologically heterogeneous and showed a precursor B-cell immunophenotype including colocalization with CD34 by single-antibody immunohistochemical analysis and coexpression of variable levels of CD79a and CD10 and lack of CD3 expression by double immunostaining. These features should aid in the evaluation of pediatric lymph nodes for partial involvement by lymphoblastic lymphoma/leukemia.

Terminal deoxynucleotidyl transferase (TdT) is a nuclear enzyme whose expression is restricted, in normal tissues, to lymphoid precursors of B- and T-cell lineage. In vitro, TdT catalyzes the template-independent addition of deoxynucleotides to the 3’-hydroxyl terminus of oligonucleotide primers, hence its name.1 In vivo, it has a crucial role in insertion of N regions during immunoglobulin and T-cell receptor gene rearrangements at the D-J and V-DJ junction sites.2-4 This mechanism of junctional diversity is essential to the development of an adult-type immunoglobulin and T-cell receptor repertoire.4 TdT-positive lymphoid precursors are present mainly in the thymus and bone marrow, reflecting the physiologic role of these organs in lymphopoiesis. In addition, TdT-expressing lymphoid cells have been identified in peripheral blood5,6 and, recently, in tonsil.7,8 However, the presence, frequency, and localization of TdT-positive lymphoid cells in normal lymph nodes of children and adults have not been examined extensively.

TdT is expressed in malignant tumors of lymphoblastic lineage (including precursor-B and T lymphoblastic leukemia/lymphoma and lymphoid blast crisis of chronic myeloid leukemia) and in a subset of acute myeloid leukemias.9-11 Lymphoblastic tumors, manifesting as leukemia or lymphoma, are the most frequent malignant neoplasms of childhood and usually express TdT.12 Consequently, partial lymph node involvement by a lymphoblastic process is not infrequently included in the pathologist’s differential diagnosis when examining a lymph node biopsy specimen from a child with a history of a lymphoblastic malignant neoplasm. Thus, a rigorous characterization of the morphologic and immunophenotypic features of normal TdT-positive cells is of importance in the differential diagnosis with involvement by these neoplasms.
The purpose of the present study was to establish a normal baseline for interpretation of TdT staining in benign lymph nodes of children. By using immunohistochemical analysis, we assessed the incidence, distribution pattern, morphologic features, and immunophenotype of TdT-positive lymphoid cells in benign lymph nodes obtained from 26 pediatric patients with a variety of benign and malignant disorders. Our findings document the presence and characteristic distribution pattern of variable numbers of TdT-positive precursor B cells in reactive lymph nodes in this age group.

Materials and Methods

Patient Group

The pathology files at St Jude Children’s Research Hospital, Memphis, TN, from April 2000 to July 2001 were searched for lymph node excisional biopsy material for which the diagnosis of benign lymphoid hyperplasia had been given. The cases that had adequate material for additional immunohistochemical studies were included. Cases with a history of lymphoblastic lymphoma/leukemia or Burkitt lymphoma were excluded.

The clinical history (including patient sex, age at time of biopsy, and previous chemotherapy) and gross pathologic findings, including lymph node size, were extracted from the surgical pathology report.

Microscopic Examination

Three histologic sections were prepared for each case and stained with H&E. Slides were reviewed independently by 2 pathologists (M.O., R.B.L.). Morphologic findings were classified into commonly recognized patterns of reactive hyperplasia, including follicular hyperplasia, paracortical hyperplasia, and sinus histiocytosis. The relative extent of involvement of the lymph node by each pattern was quantified as the percentage of lymph node section, in 20% increments, from 0% to more than 80%.

Immunohistochemical Staining

Immunohistochemical staining was performed on consecutive formalin-fixed, paraffin-embedded tissue sections for each case, using the avidin-biotin peroxidase technique and hematoxylin counterstaining. Briefly, after paraffin sections were deparaffinized and rehydrated, antigen retrieval was performed by maintaining in citrate buffer (a 0.1-mol/L concentration of citric acid, pH 6.0) at 90°C to 96°C for 30 minutes, followed by a 30-minute cool-down in a steamer (model HS2776, Black and Decker, Towson, MD). Staining was performed on an autostainer (DAKO, Carpinteria, CA) using the enhanced peroxidase-diaminobenzidine (DAB) chromogen kit. A hematoxylin counterstain was used. Antibodies used along with their sources and titers are listed in Table 1. For each antibody used, negative controls (no primary antibody) and positive controls (lymphoblastic lymphoma for TdT and CD10 and tonsil for CD3, CD34, CD79a) were included.

Double-labeling immunohistochemical staining was performed on 5 cases with higher numbers of TdT-positive cells (cases 8, 16, 17, 21, 24) using antibodies for the following combinations of antigens: TdT/CD79a, TdT/CD10, and TdT/CD3. Antigens used, titers, and antigen retrieval were similar to those used for single-antibody immunohistochemical analysis. The enhanced DAB kit (DAKO) and enhanced alkaline phosphatase–blue (DAKO) were used, with a light green counterstain. In all combinations, following antigen retrieval as described, sections were incubated with anti-TdT antibody for 30 minutes at room temperature, followed by addition of peroxidase-DAB chromogen. Sections then were washed and incubated with CD3, CD10, or CD79a antibody for 30 minutes at room temperature, followed by the addition of alkaline phosphatase–blue chromogen.

Following immunohistochemical staining, each lymph node was examined for the presence of TdT-positive cells. When the latter were present, their cytologic features, pattern of distribution, and frequency were recorded. TdT-positive cells were enumerated in the area of maximum density and expressed as cells per high-power field (40× objective, 10× ocular, Olympus BX41 microscope, Melville, NY).

Results

Clinical Data

We identified 26 patients (4 boys and 22 girls), age 10 weeks to 17 years (median, 4.5 years). Their clinicopathologic findings are summarized in Table 2. Primary diagnoses included Wilms tumor (10 patients), neuroblastoma or ganglioneuroma (5 patients), rhabdomyosarcoma (3 patients), and rhabdoid tumor, desmoid tumor, vascular malformation, Castleman disease, colon adenocarcinoma, and Hodgkin disease (1 patient each). One patient had no
history of malignancy but a strong family history of non-Hodgkin lymphoma. One patient had a RET proto-oncogene mutation and underwent total thyroidectomy with cervical lymph node dissection for prophylaxis of medullary carcinoma. Seven patients had received chemotherapy for their malignant neoplasm before undergoing the lymph node biopsy. One patient had received local radiation therapy for desmoid tumor. None of the remaining 18 patients had received previous therapy.

**Gross and Histopathologic Findings**

Lymph node location included abdominal (periaortic, aortocaval, pericaval, renal hilar, and pericolonic) in 17 patients and peripheral (head and neck, inguinal, and axillary) in 9 patients. Lymph node size ranged from 0.4 to 3.0 cm in greatest dimension (median, 1.2 cm). The median size was similar in treated and nontreated patients (1.2 cm and 1.1 cm, respectively).

Histologic patterns of lymphoid hyperplasia included predominantly (80% or more) follicular hyperplasia (1 case), predominantly (80% or more) paracortical hyperplasia (4 cases), predominantly (80% or more) sinus histiocytosis (4 cases), or a combination of 2 or 3 of these patterns (each 20%-60%; 17 cases) **Image 1A**. In 2 cases (5 and 7), the overall pattern, combined with the presence of pigment-laden macrophages, was suggestive of dermatopathic lymphadenopathy. In 1 case (case 21) the lymph node contained scattered aggregates of epithelioid histiocytes and poorly formed noncaseating granulomas. None of the lymph nodes showed involvement by malignancy.

**Immunohistochemical Analysis for TdT-Positive Lymphoid Cells**

TdT-positive lymphoid cells were identified in all 26 lymph nodes by immunohistochemical analysis. These cells showed nuclear positivity for TdT of variable intensity, with most cells being strongly positive **Image 1B**. Morphologically, these cells had very scant cytoplasm and were heterogeneous, ranging from small lymphoid cells with smooth, round to ovoid nuclear contours, to medium-sized and large cells, with irregular nuclear contours and, occasionally, cleaved nuclei.

The pattern of distribution of TdT-positive cells was similar in all cases examined. They were identified immediately adjacent to sinuses and small vessels. Some cases showed variable numbers of TdT-positive cells within

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**Table**

**Correlation Between Clinicopathologic Features and Number of TdT-Positive Cells in Reactive Lymph Nodes From 26 Pediatric Patients**

<table>
<thead>
<tr>
<th>Case No./Sex/Age</th>
<th>Diagnosis</th>
<th>Previous Chemotherapy</th>
<th>Lymph Node Location</th>
<th>Lymph Node Pattern of Hyperplasia</th>
<th>Lymph Node Size (cm)</th>
<th>TdT+ Cells/HPF</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/F/3 y</td>
<td>Neuroblastoma</td>
<td>Yes</td>
<td>Abdominal</td>
<td>Sinus histiocytosis</td>
<td>0.5</td>
<td>16</td>
</tr>
<tr>
<td>2/F/14 y</td>
<td>Colon adenocarcinoma</td>
<td>Yes</td>
<td>Abdominal</td>
<td>Follicular</td>
<td>0.6</td>
<td>1</td>
</tr>
<tr>
<td>3/F/4 y</td>
<td>Neuroblastoma</td>
<td>Yes</td>
<td>Abdominal</td>
<td>Paracortical</td>
<td>0.6</td>
<td>3</td>
</tr>
<tr>
<td>4/M/7 y</td>
<td>Neuroblastoma</td>
<td>Yes</td>
<td>Abdominal</td>
<td>Paracortical</td>
<td>1.2</td>
<td>1</td>
</tr>
<tr>
<td>5/M/9 y</td>
<td>Rhabdomyosarcoma</td>
<td>Yes</td>
<td>Inguinal</td>
<td>Paracortical</td>
<td>1.2</td>
<td>25</td>
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<tr>
<td>6/F/17 y</td>
<td>Hodgkin disease</td>
<td>Yes</td>
<td>Cervical</td>
<td>Mixed</td>
<td>2.0</td>
<td>5</td>
</tr>
<tr>
<td>7/F/12 y</td>
<td>Desmoid tumor</td>
<td>No (local RT)</td>
<td>Axillary</td>
<td>Mixed</td>
<td>2.0</td>
<td>10</td>
</tr>
<tr>
<td>8/F/7 y</td>
<td>Rhabdomyosarcoma</td>
<td>Yes</td>
<td>Inguinal</td>
<td>Mixed</td>
<td>3.0</td>
<td>65</td>
</tr>
<tr>
<td>9/M/3 mo</td>
<td>Neuroblastoma</td>
<td>No</td>
<td>Abdominal</td>
<td>Mixed</td>
<td>0.4</td>
<td>22</td>
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<tr>
<td>10/F/10 y</td>
<td>RET gene mutation</td>
<td>No</td>
<td>Central neck</td>
<td>Mixed</td>
<td>0.5</td>
<td>6</td>
</tr>
<tr>
<td>11/F/3 y</td>
<td>Vascular malformation</td>
<td>No</td>
<td>Cervical</td>
<td>Paracortical</td>
<td>0.5</td>
<td>1</td>
</tr>
<tr>
<td>12/F/5 y</td>
<td>Ganglioneuroma</td>
<td>No</td>
<td>Abdominal</td>
<td>Mixed</td>
<td>0.5</td>
<td>5</td>
</tr>
<tr>
<td>13/F/4 y</td>
<td>Wilms tumor</td>
<td>No</td>
<td>Abdominal</td>
<td>Mixed</td>
<td>0.7</td>
<td>5</td>
</tr>
<tr>
<td>14/F/16 mo</td>
<td>Wilms tumor</td>
<td>No</td>
<td>Abdominal</td>
<td>Paracortical</td>
<td>1.0</td>
<td>24</td>
</tr>
<tr>
<td>15/F/2 y</td>
<td>Wilms tumor</td>
<td>No</td>
<td>Abdominal</td>
<td>Sinus histiocytosis</td>
<td>1.0</td>
<td>50</td>
</tr>
<tr>
<td>16/F/5 y</td>
<td>Reactive lymphadenopathy</td>
<td>No</td>
<td>Cervical</td>
<td>Mixed</td>
<td>1.1</td>
<td>80</td>
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<tr>
<td>17/F/7 y</td>
<td>Rhabdomyosarcoma</td>
<td>No</td>
<td>Diaphragmatic</td>
<td>Mixed</td>
<td>1.1</td>
<td>52</td>
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<tr>
<td>18/F/10 wk</td>
<td>Rhabdoid tumor</td>
<td>No</td>
<td>Abdominal</td>
<td>Mixed</td>
<td>1.3</td>
<td>18</td>
</tr>
<tr>
<td>19/F/16 mo</td>
<td>Wilms tumor</td>
<td>No</td>
<td>Abdominal</td>
<td>Mixed</td>
<td>1.3</td>
<td>37</td>
</tr>
<tr>
<td>20/F/3 y</td>
<td>Wilms tumor</td>
<td>No</td>
<td>Abdominal</td>
<td>Mixed</td>
<td>1.3</td>
<td>31</td>
</tr>
<tr>
<td>21/F/16 y</td>
<td>Castleman disease</td>
<td>No</td>
<td>Postauricular</td>
<td>Sinus histiocytosis</td>
<td>1.4</td>
<td>32</td>
</tr>
<tr>
<td>22/F/6 y</td>
<td>Wilms tumor</td>
<td>No</td>
<td>Abdominal</td>
<td>Mixed</td>
<td>1.4</td>
<td>180</td>
</tr>
<tr>
<td>23/F/11 mo</td>
<td>Wilms tumor</td>
<td>No</td>
<td>Abdominal</td>
<td>Mixed</td>
<td>1.5</td>
<td>25</td>
</tr>
<tr>
<td>24/F/18 mo</td>
<td>Wilms tumor</td>
<td>No</td>
<td>Abdominal</td>
<td>Mixed</td>
<td>1.5</td>
<td>125</td>
</tr>
<tr>
<td>25/F/14 mo</td>
<td>Wilms tumor</td>
<td>No</td>
<td>Abdominal</td>
<td>Sinus histiocytosis</td>
<td>1.6</td>
<td>7</td>
</tr>
<tr>
<td>26/M/5 y</td>
<td>Wilms tumor</td>
<td>No</td>
<td>Abdominal</td>
<td>Sinus histiocytosis</td>
<td>2.0</td>
<td>2</td>
</tr>
</tbody>
</table>

HPF, high-power field; RT, radiation therapy.

* Follicular is follicular hyperplasia in ≥80% of lymph node; paracortical, paracortical hyperplasia in ≥80% of lymph node; sinus histiocytosis, sinus histiocytosis in ≥80% of lymph node; mixed, mixture of any 2 or 3 of the previous patterns (each 20%-60% of lymph node).

† Assessed in the area of maximum density.
Morphologic and immunophenotypic features of terminal deoxynucleotidyl transferase (TdT)-positive cells in a benign reactive renal hilar lymph node from an 18-month-old girl with Wilms tumor. 

A. Follicular hyperplasia and mild sinus histiocytosis (H&E, ×20). 
B. Numerous TdT-positive cells are present adjacent to medullary sinuses (frequency of TdT-positive cells in the area of maximal density, 125 per high-power field) (TdT, ×20). 
C. TdT/CD79a double immunostaining. A subset of TdT-positive cells expressed variable levels of CD79a (TdT/alkaline phosphatase–blue, CD79a/diaminobenzidine [DAB], ×60). 
D. TdT/CD10 double immunostaining. Many of the TdT-positive cells coexpressed CD10 with variable intensity (TdT/alkaline phosphatase–blue, CD10/DAB, ×60). 
E. TdT/CD3 double immunostaining. There is no coexpression of TdT and CD3 (TdT/alkaline phosphatase–blue, CD3/DAB, ×60).
sinuses or transitioning between the lymph node pulp and sinus lumen. No TdT-positive cells were detected within lymphoid follicles. The overall distribution was patchy. Within the area of maximal density, they ranged from 1 to 180 per high-power field (median, 20 cells). In cases containing more than 20 cells per high-power field, clusters of 2 to 5 TdT-positive cells were detected. However, no large aggregates or sheets of TdT-positive cells were seen in any of the cases. Of note, owing to the patchy distribution, even in cases with the highest density of TdT-positive cells, the overall frequency of these cells (as a percentage of the entire lymph node cell population) was less than 1%.

In all cases, single-antibody immunohistochemical staining for CD10 and TdT performed on consecutive levels showed colocalization of cells positive for these markers. In most cases, areas containing these CD10- and TdT-expressing cells overlapped with areas containing numerous B cells positive for CD79a. Immunohistochemical analysis for TdT and CD34 colocalization was more difficult to interpret owing to the high density of endothelial cells strongly positive for CD34 that were present in the same areas. However, in 19 cases, small lymphoid cells that were positive for CD34 were identified in areas where TdT-positive cells were present. These cells were less frequent than the TdT-positive cells. By double immunohistochemical staining, there was coexpression of TdT with CD79a and CD10 in a variable percentage of cells in all 5 cases examined (Image 1C and Image 1D). No cells coexpressing TdT and CD3 were seen in any of the cases. Image 1E. The intensity of CD79a and CD10 expression was variable and did not correlate with the level of TdT expression. The pattern of expression of these 2 markers (ie, cytoplasmic vs membranous) was difficult to discern owing to the scant amount of cytoplasm of these cells.

In lymph nodes from patients who had not received chemotherapy, the frequency of TdT-positive lymphoid cells seemed to correlate with the overall size of the lymph node. (Table 2). Lymph nodes larger than 1 cm in greatest dimension generally contained more frequent TdT-positive cells (2-180 cells; median, 32 cells) than those smaller than 1 cm (1-22 cells; median, 5 cells). However, there was overlap between these 2 categories (Table 2, cases 9, 25, and 26). This correlation was even less consistent in patients who had received previous chemotherapy or local radiation therapy (Table 2). The frequency and pattern of distribution of the TdT-positive cells were similar in the treated and untreated patient groups. Likewise, the frequency of TdT-positive lymphoid cells did not seem to correlate with patient age, previous or coexisting disease, lymph node location, or pattern of lymphoid hyperplasia, although the number of cases in each category is too small to allow statistically significant conclusions.

Discussion

In B-lymphoid precursors, TdT expression is limited to the early stages of maturation (pro-B and early pre-B), during which immunoglobulin heavy chain gene rearrangements occur. TdT expression is promptly down-regulated following the onset of immunoglobulin heavy chain (mu) synthesis in the cell and is, therefore, absent during rearrangement of the immunoglobulin light chain genes, which accounts for the absence of N insertions in the latter. Early TdT-positive B-cell precursors have been characterized extensively in bone marrow, where they have long been recognized morphologically as benign immature lymphoid cells, or so-called hematogones. Numerous studies have described comprehensively the morphologic and immunophenotypic characteristics of these cells and their differential diagnosis with acute lymphoblastic leukemia. These studies established that TdT-positive lymphoid cells in bone marrow are B-cell precursors showing a range of maturation, increase numerically in a variety of reactive conditions, and are most numerous in infants and children.

The presence of benign TdT-positive B-cell precursors has been documented in peripheral blood and, more recently, in reactive human tonsils in both children and adults. The presence of TdT-positive lymphoid cells has been evaluated in a limited number of studies assessing benign lymph nodes, spleen, and terminal ileal Peyer patches from children and adults. However, the latter cases usually were included as normal control cases in studies addressing other issues. Thus, a systematic study of the frequency, distribution pattern, morphologic features, and immunophenotype of these cells is lacking. Importantly, a better characterization of the frequency of the TdT-positive lymphoid cells in benign lymphoid tissue is needed, since TdT immunohistochemical analysis is used in the evaluation of lymph nodes for partial involvement by lymphoblastic leukemia/lymphoma, a neoplasm characteristically positive for TdT expression. Since lymphoblastic malignant neoplasms are encountered most commonly in pediatric patients, such an assessment of TdT-positive lymphoid cells is particularly relevant to the histopathologic evaluation of lymphoid tissue obtained from children.

By using immunohistochemical analysis, we demonstrated the consistent presence of TdT-positive lymphoid precursors in benign lymph nodes from children. These cells coexpress variable levels of CD79a and CD10, and they are positive for CD34 in a subset of cells. These cells share morphologic and immunophenotypic features with their counterparts in bone marrow and peripheral blood, including CD10 and CD79a expression that is characteristically of variable intensity. In addition, the distribution, frequency, and immunophenotype of TdT-positive cells in lymph nodes seem to correlate with the overall size of the lymph node.
closely resemble those previously described in lymph nodes of the midterm fetus.19

Previous studies using immunohistochemical analysis7,9 or enzymatic methods11 documented rare nodal TdT-positive cells. Meru et al.8 observed variable numbers of TdT-positive cells, ranging from 0 to 25 cells per high-power field in areas of maximal density. We obtained similar results, although the frequency of TdT-positive cells in the present study frequently was higher than that reported by other investigators. This apparent discrepancy may be due to the fact that our study included lymph nodes obtained exclusively from pediatric patients, while the previous studies included a mixture of pediatric and adult cases. In addition, the majority of our patients had a history of concurrent or past malignant neoplasms. McKenna et al.18 in their recent study of hematogones in 662 bone marrow specimens, showed that the number of benign lymphoid precursors is higher in younger patients and in the setting of malignancy, bone marrow transplantation, and postchemotherapy. The number of such precursors in lymph nodes may follow the same pattern, although in our study, we found no difference between patients with and patients without a history of malignancy. Likewise, it is uncertain whether reactive lymph nodes from adults contain similar numbers and distribution of TdT-expressing lymphoid cells. Further studies might be needed to address this issue. Meru et al.8 found no statistical correlation between patient age and the frequency of TdT-positive cells in tonsils. In our study, we found no significant correlation with age in patients younger than 1 year to 17 years of age, although patients older than 10 years of age generally had fewer cells than younger patients. These cells were most numerous in larger lymph nodes (generally larger than 1 cm), perhaps reflecting the intensity or duration of immune stimulation.

We found that benign TdT-positive cells had a consistent distribution pattern in reactive lymph nodes. They always were located in the immediate vicinity of lymph node sinuses and capillaries, most often in the medulla and occasionally in the cortex. Few cells were present within sinuses, including the subcapsular sinus. The distribution of TdT-positive cells always was patchy and did not distort or efface normal architecture. Even in cases with a high density of such cells, they did not form clusters larger than 4 to 5 cells. We never observed large cohesive sheets of TdT-positive cells within or around sinuses. These features contrast with those of partial lymph node involvement by lymphoblastic lymphoma, which occurs in about one third of cases.12 In such cases, large clusters and sheets of TdT-positive cells selectively expand the paracortex.12 Differences in cytologic features also are useful for distinguishing between malignant lymphoblasts and benign TdT-positive cells. Whereas the former typically have a relatively monotonous appearance, nodal TdT-expressing lymphoid cells usually show a range of morphologic features. The immunophenotypic findings often match these features, in that for any given antigen, malignant lymphoblasts tend to express more uniform levels of that antigen than do their benign TdT-positive counterparts. Last, most cases of lymphoblastic lymphoma are of T-cell lineage,12 which is in contrast with benign TdT-positive cells, which have a B-cell phenotype. Nevertheless, as with any type of tumor, we recommend integrating all these features when making a diagnostic decision. Of note, owing to their patchy distribution, these cells represent less than 1% of the overall lymph node cell population, making them more difficult to detect by flow cytometric analysis of a cell suspension at the limits of sensitivity used routinely in the diagnostic setting. This makes their presence a less likely problem in the differential diagnosis with a lymphoblastic neoplasm when the lymph node is evaluated by flow cytometry.

We demonstrated that most benign lymph nodes from pediatric patients contain a variably sized population of TdT-positive precursor B cells. These cells show a characteristic morphologic and immunophenotypic spectrum of maturation and have a consistently patchy distribution in the areas immediately adjacent to medullary and cortical sinuses. These features should aid in the differential diagnosis with partial lymph node involvement by lymphoblastic lymphoma.

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References


