Review

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Pediatric-type follicular lymphoma: a short review

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Practice points

- Pediatric-type follicular lymphoma is characterized by localized nodal disease with a tendency to involve the head and neck region.
- Despite its indolent clinical course, it has a high proliferative index with blastoid features histopathologically.
- A prominent immunohistochemistry finding is a lack of *BCL2* expression and the absence of t(14;18) by cytogenetic analysis.
- A high index of suspicion is needed, given the significant overlap between the morphological and clinical features of pediatric-type follicular lymphoma and several disease entities.
- Complete surgical excision followed by observation can be the way to manage most cases, with the remaining needing an immunochemotherapy approach.
- The outcome is excellent and favorable, with a high cure rate and negligible risk of relapse or transformation.

Pediatric-type follicular lymphoma is an uncommon and newly recognized entity of lymphoid neoplasm commonly encountered in the young population. Despite its indolent clinical course and localized nodal involvement, it has been characterized by its high-grade histopathological features. The overlapping features between this disease and several entities have made approaching this unique entity significantly challenging, with all such features being reflected in the strict diagnostic criteria highlighted by the WHO 2016 lymphoid malignancy classification. Despite its characteristic high-grade histology, its cure rates have remained high, with relapse and transformation rarely occurring. Interestingly, several cases have achieved remission following nodal disease resection, possibly eliminating the need for chemotherapy and radiation and preventing long-term morbidities from later approaches in disease survivors.

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Despite follicular lymphoma being considered one of the common lymphoid malignancies in adults [1], it can occur, albeit at lower frequencies (1–2%), in young adults and children [2–4]. Pediatric-type follicular lymphoma (PTFL) is a recently recognized variant that has been labeled as a definite entity by the WHO classification in 2016 [5]. Although it is a rare entity commonly found in patients younger than 18, it is not limited to the pediatric age group and can develop in young adults [6,7]. PTFL has distinctive features that make it unique compared with that found in adults. In particular, it most commonly presents clinically as a localized disease and has favorable outcomes with a high cure rate; however, it has been characterized by its high-grade histological features [3,7,8]. Recently, the genomic landscape has been mapped with the presence and absence of specific genetic mutations that further describe this entity and differentiate it from the conventional follicular lymphoma, indicating a separate pathobiology [9–13].

Clinical characteristics

PTFL cases predominantly occur in those under 25 years old, with a median age of 15 years. Male predominance is observed in most reports, with a male-to-female ratio of around 10:1 [2,6–8,14]. Interestingly, a retrospective analysis

Table 1. Differences in morphological and clinical features with outcomes between pediatric type follicular lymphoma and usual (conventional) follicular lymphoma.

Feature	Pediatric-type follicular lymphoma	Usual follicular lymphoma
Age	Majority under 25 years old	Rarely in those aged <30 years
Sex	Male predominance (male:female of 10:1)	Equal male and female incidence
Stage at presentation	Localized disease (stage I/II)	Majority present with advanced stage
Extra-nodal involvement	Rare, and its presence clouds the diagnosis	Commonly present, especially in the bone marrow
Microscopic features	Aggregates of atypical blastoid cells in a follicular pattern with a starry sky pattern	Various degrees of centrocytes and centroblasts
Immunophenotypic and cytogenetic	Lack of *BCL2* expression and absence of *BCL2* and *BCL6* rearrangement	Strong expression of *BCL2* in addition to other B-cell antigens t(14;18) present in 85% of the cases *BCL6* rearrangement present in 15% of the cases
Genetic profile	Lack of mutations in epigenetic modifier genes *MAP2K1* and *IRF8* mutation	Presence of mutations in epigenetic modifier genes at considerable frequencies
Outcome and prognosis	Curable disease with favorable outcomes and negligible risk of relapse	Relapsing-remitting disease

Data taken from [1,5,23].

of a cohort of patients reported cases beyond the commonly reported age group, ranging from 18 to 61 years, which share similar histological features [7]. PTFL can be characterized by its indolent clinical behavior. Moreover, studies have shown that more than two-thirds of affected patients presented with localized disease (stage I/II) with a propensity to involve the head and neck regions [2–4,7,8,15–17]. Although extra-nodal involvement has been reported in a wide range of cases involving the testis, abdomen and bone marrow, these cases occur much less frequently than does nodal involvement [7,8,18,19]. This differs from the conventional follicular lymphoma, in which extra-nodal involvement, including the bone marrow, occurs more frequently (see Table 1) [1,20]. However, extra-nodal involvement (e.g., of the testis, GI tract,, bone marrow), previously labeled as extra-nodal involvement of PTFL, has recently been excluded from the revised WHO classification of lymphoid neoplasm based on morphological and clinical differences [5,21].

Morphology

Microscopic & immunophenotyping features

PTFL can be characterized by extensive effacement of the nodular architecture by the monomorphic distribution of atypical cells in a follicular pattern. These follicles are large and expansile with a loss of polarization. The cells mostly have blastoid features, medium to large cells showing round/oval nuclei, small nucleoli, finely clumped chromatin and scant cytoplasm. Alongside these blastoid cells, the mixture of tangible body macrophages gives a starry sky appearance [2,4,22,23]. The mantle zone is thin and appears to be pushed by the neoplastic follicles. Although previously labeled to have grade 3 histology (FL grading system), a proportion of cases lack centrocytes and centroblasts. Their presence appears to be rare and unobtrusive, for which grading is not recommended in the microscopic description of this disease entity [4,5,16,23]. The presence of a component of diffuse large B-cell lymphoma is not included in the diagnosis. Notably, the presence of reactive and neoplastic follicles within the same lymph node, a finding called "node within a node," makes diagnosis challenging and needs careful examination and inspection in order not to overlook the diagnosis of the neoplastic process (see Figure 1) [4,10,16].

Immunohistochemistry is derived from germinal center B cells and characterized by CD10, BCL6, CD20 and CD79a positivity. Unlike its adult counterpart, PTFL lacks the expression of BCL2 (see Figure 1). Reports have shown that around 20–30% of the cases could be positive for BCL2, albeit weakly [4,7,23]. Previous observations from retrospective studies found that cases with BCL2 expression were associated with advanced stage at presentation and occurred in a relatively older population of children (>12 years old) and young adults, although such findings were not consistently observed in other observational studies [2,7,15]. Ki-67 staining reveals the presence of a high proliferation index of more than 30%, with the majority ranging from 40% to 90% [3,7,23]. MUM1/IRF4 expression is usually negative [5]. Liu *et al.* reported that all cases of PTFL involving the Waldeyer's ring in their cohort (eight cases) showed uniform positivity for MUM1, whereas only 5.5% of nodal cases exhibited the same [16]. Cases with MUM1 positive likely represent another entity, namely large B-cell lymphoma with *IRF4* rearrangement [5,24].

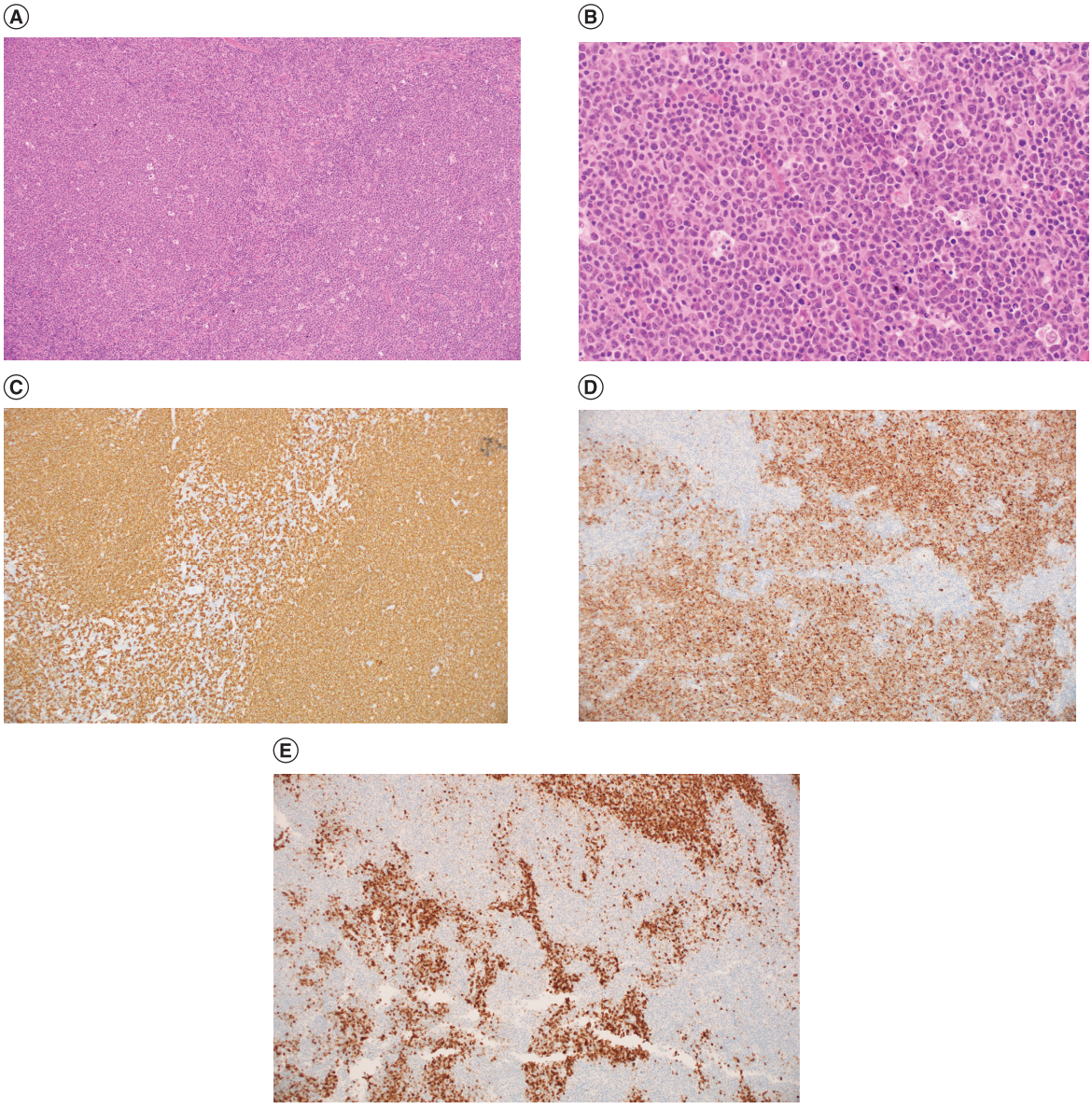

Figure 1. Microscopic features of pediatric-type follicular lymphoma. (A) Fully effaced lymph node architecture by large expansile follicles. **(B)** Follicles composed of monotonous intermediate-sized atypical lymphoid cells with blastoid appearance and starry sky pattern. **(C)** CD20 stain. Closely packed follicles. **(D)** Strongly expressed CD10 highlighting serpiginous follicles. **(E)** Negative BCL2 expression in the neoplastic follicles.

Atypical cells show negative staining for IgD, with positive findings being limited to thin and constricted mantle zones. PD-1 staining represents follicular helper T cells being pushed by the neoplastic cells at the periphery of the follicles [16].

One of the recently described novel markers (*FOXP-1*) belongs to the FOX transcription factor family responsible for B-cell development and function, which was uniformly positive in around 95% of the cases in a recently published study [10]. It can be used as a marker to differentiate PTFL from reactive follicular hyperplasia. Although the significance of its impact on outcomes has yet to be clarified in PTFL, it has been associated with inferior outcomes in cases of conventional FL and diffuse large B-cell lymphoma [25,26].

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Cytogenetic & molecular profile

The most prominent cytogenetic feature of PTFL is the lack of t(14;18). In addition, fluorescence *in situ* hybridization shows negative findings for *BCL2*, *BCL6*, *MYC*, *IRF4* rearrangement or *BCL2* amplification [4,5,23], whereas PCR analysis shows immunoglobulin gene rearrangement defining the clonality of neoplastic cells.

PTFL is of low genomic complexity, with previous suppositions suggesting it to be a "*benign clonal proliferation with low-malignant potential*" [12]. Specifically, the low genomic burden of this entity was reported in a previous genomic-based study, which identified low copy number alteration per case compared with conventional FL (mean, 0.77 vs 9 copy number alteration per case). Earlier studies evaluating the genetic basis of the disease found the presence of an alteration in the 1p36 locus related to copy number neutral loss of heterozygosity of this locus targeting TNFRSF14, on contrary cases of conventional FL frequently showed loss of this focus [12,36].

In contrast to conventional FL, this neoplasm lacks mutations in the epigenetic modifiers *CREBBP*, *EZH2* and *KMT2D* [12]. The genomic landscape of PTFL has evolved recently, with recent findings identifying a recurrent mutation in specific genes. Among these genes, the most reported mutations are *TNFRSF14*, *MAP2K1* (which encodes *MEK1* and is a regulator of *ERK1/2*) and *IRF8* (see Table 1) [9,11–13].

Differential diagnosis

One of the challenges and difficulties in establishing a diagnosis is the presence of significant overlap between the morphological and clinical features of PTFL and those of several other entities. These include reactive follicular hyperplasia, pediatric nodal marginal zone lymphoma (PNMZL), large B-cell lymphoma with IRF4 rearrangement, BCL2 translocation-negative conventional (usual) FL and grade 3B FL.

Some morphological overlap can exist between PTFL and reactive follicular hyperplasia, specifically the starry sky appearance of the hyperplastic follicles. However, the malignant features of the generalized architectural effacement, the presence of atypical cells with blastoid features and the loss of polarization in the follicles favor PTFL. Importantly, clonality alone cannot be used as a diagnostic criterion, given that a clonal population can also be seen in follicular hyperplasia [16,23,27,28].

The similarity in clinical presentation between PTFL and PNMZL creates another challenge in diagnosis [23]. PNMZL is characterized by the presence of progressively transformed germinal centerlike changes [29,30]. In addition, interfollicular areas are expanded and contain CD20-positive B cells, which appear to be limited in PTFL. Moreover, PNMZL is characterized by a polymorphous population of monocytoid, plasmacytoid and plasma cells and few transformed blasts in contrast to the monomorphic population of blastoid cells in PTFL.

Immunophenotypically, PNMZL lacks the germinal center markers (e.g., CD10- and BCL6-negative) and may co-express CD43. In contrast, PTFL rarely expresses CD43 [16,23,30]. A recent study identified overlapping features between the two entities, including a similarity in molecular profile with recurrent alterations in *MAP2K1*, *TNFRSF14*, and *IRF8* genes [37]. The similarity in the molecular signature based on this recent study suggests that PTFL and PNMZL are variants of one disease.

Large B-cell lymphoma with *IRF4* rearrangement has recently been added as a new provisional entity with a propensity to involve the head and neck region with limited staging [5]. In contrast to PTFL, the aforementioned condition has no sex predominance and appears to occur equally among males and females. It is characterized by various architectural patterns such as follicular, diffuse or both. Large B-cell lymphoma with *IRF4* rearrangement lacks the starry sky appearance seen in PTFL. Moreover, an immunohistochemistry study showed BCL2 positivity in more than half of the cases with strong *IRF4/MUM1* expression [14,23,24,31].

BCL2 translocation-negative conventional (usual) FL accounts for around 15% of conventional FLs [14]. It commonly lacks CD10 expression but expresses CD23. Despite its low grade (1–2 out of 3), it has a high proliferation index [32]. BCL6 translocation can be present in 10–15% of the cases [33,34].

Finally, grade 3B FL is an important differential diagnosis, given the high-grade histopathological features. However, grade 3B FL is characterized by widespread lymphadenopathy rather than the localized disease involvement seen in PTFL. Furthermore, grade 3B FL is characterized by diffuse sheets of centroblasts with follicular growth patterns, although it can be limited. An important consideration is that BCL2 expression can be absent in around 29–55% of the cases, as well as t(14;18) translocation detectable at lower frequencies (5–13%), both of which cannot be used as a sole feature to distinguish between the two entities. Clinical correlation and careful morphological assessment are needed for proper evaluation [38].

Table 2. WHO 2016 diagnostic criteria for pediatric-type follicular lymphoma.

Features required for diagnosis	Supportive features, but not required for diagnosis
• Morphological architectural effacement of the lymph node, minimally partial effacement • Predominant follicular proliferation • Positive BCL6 by immunohistochemistry • Weakly positive or negative BCL2 by immunohistochemistry • High Ki-67 (>30%) • No molecular rearrangement of BCL2, BCL6 or IRF4 or aberrant IG • No amplification of BCL2 • Lymph node involvement only • Limited-stage disease (I–II)	• Morphological follicular expansion • Presence of blastoid cells, intermediate in size without centrocytes • Younger age (<40 years) • Male gender

Data taken from [5].

Diagnostic criteria

The revised version of the fourth WHO classification of lymphoid malignancy has set strict criteria for the diagnosis of PTFL to overcome the various features described in the literature that overlap with the characteristics of other diseases (see Table 2). These criteria comprise morphology, immunohistochemistry and genomic and clinical features [5]. Recently, a summary of the fifth WHO classification of lymphoid malignancy and an alternate classification scheme, the International Consensus Classification, have been published. The updates show no significant difference from the prior fourth WHO classification established for PTFL [39,40].

Management & prognosis

The rarity of PTFL makes it difficult to conduct a prospective randomized trial to determine the most optimal therapeutic strategy. Morbidity can be driven by treatment and short- and long-term toxicity rather than the disease itself. Management strategies comprise a conservative approach of watch–wait following complete excision, immunochemotherapy and a minimal number of cases undergoing field radiation [2,3,8,35]. Chemotherapy regimens described in the reported literature were primarily based on non-Hodgkin lymphoma-Berlin-Frankfurt-Munster protocols, which are likely to be anthracycline-based (R-CHOP-like) for cases where complete resection is not achieved [8]. Nonetheless, the overall outcomes have been excellent, and the risk of relapse remains low among the reported treatment modalities [7,8,15,23]. Such a fact lends credence to a conservative approach in cases with an entirely resectable lesion to avoid the long-term effects of chemotherapy in younger populations.

More patients younger than 18 years underwent complete excisions alone compared with young adults. Indeed, Louissaint *et al.* reported that 88% of patients under the age of 18 underwent excision alone, whereas only 30% of those above the age of 18 underwent the same [13]. This difference could be explained by previous presumptions that PTFL cases occurring in adults might not be biologically equivalent to those in children and concerns regarding overlap with high-grade FL, for which the majority of the adult population underwent chemoimmunotherapy. However, recent studies in the genomic landscape of PTFL have described similar recurrent mutations occurring in children and adults, indicating a similarity in pathobiology that is unlikely affected by age [12,13].

PTFL is characterized by indolent clinical behavior with a high cure rate, unlike conventional (usual) FL, which is typically characterized by a relapsing–remitting course. Multiple observations have consistently reported an overall survival of more than 95% [8,9,23]. The transformation to high-grade lymphoma is unusual [4]. Attarbaschi *et al.* reported a 2-year event-free survival of 94% ± 5% and 2-year overall survival of 100% among their cohort of 63 patients [8].

Conclusion & future perspective

PTFL is a recently recognized, rare entity with unique characteristic features distinguishing it from conventional FL. It has a high cure rate with negligible risk of relapse and transformation. Most cases are successfully treated through complete surgical excision without the need for chemoradiotherapy. Expansion in research exploring the genomic basis of the disease will likely evolve with time, which will help reveal its unique features. Future descriptive studies defining this entity's most effective therapeutic intervention are needed.

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