PEDIATRIC LYMPHOMATOID PAPULOSIS WITH CYTOTOXIC IMMUNOPHENOTYPE: CASE REPORT

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According to the WHO/EORTC classification, lymphomatoid papulosis (LyP) is a recurrent, selfhealing papular eruption belonging to the spectrum of cutaneous CD30+ lymphoproliferative disorders. The neoplastic cell is typically a CD4+ T-lymphocyte, also manifesting CD30 expression. Three main histologic subtypes are recognized: type A (histiocytic), type B (mycosis fungoides-like), and type C (anaplastic large cell lymphoma-like). Although it is well documented in adults, LyP is very uncommon in children. Clinical presentation and histopathologic features of LyP in children are comparable to those in adults. A 6-year-old boy presented with multiple erythematous papules, measuring up to 1 cm in diameter, located predominantly on his legs. Over time, some papules would necrotize, as new ones appeared. His past medical history was unremarkable. According to this clinical presentation, referring differential diagnosis included Langerhans cell histiocytosis or lymphoproliferative disease. Punch biopsy of skin lesions was performed. Microscopically, skin sections showed a dense, nodular, perivascular infiltrate that extended throughout the full thickness of the dermis. The infiltrate consisted predominantly of large, blastlike lymphoid cells with moderate amounts of cytoplasm and irregular vesicular nuclei with prominent nucleoli. Relatively frequent mitotic figures and apoptotic bodies were seen. The blasts were admixed with occasional small lymphocytes, neutrophils and eosinophils. The overlying epidermis was ulcerated, focally infiltrated with lymphoid cells. Immunostaining revealed blast cells of T-cell lineage, with expression of pan-T-cell antigens CD2, CD3, CD5, and lack of CD7. They were positive for CD8 and the cytotoxic molecules perforin, TIA-1 and granzyme B, but negative for CD4. They also showed strong expression of CD30 and weak, focal expression of CD56, but absence of CD57. Staining for beta-F1 was positive, and

there was no evidence of Epstein-Barr virus, either by immunohistochemistry (LMP1) or in situ hybridisation (EBERs). These features were consistent with a CD30-positive lymphoproliferative disorder with a cytotoxic phenotype. Definitive diagnosis required clinical correlation. The patient underwent thorough hematologic work-up, and no evidence of underlying systemic lymphoma was detected. Also, transformation of pre-existing mycosis fungoides was excluded. Final diagnosis was consistent with CD8+ lymphomatoid papulosis type C. The patient received only topical corticosteroid therapy, followed by complete regression of skin lesions. After 7-month follow-up, the patient was healthy, with no evidence of disease. The rarity of childhood LyP, the multifocal skin lesions and the atypical histologic features can produce erroneous diagnosis of malignancy, leading to unnecessary aggressive treatment. Nevertheless, compared with the general population, patients with childhoodonset LyP have a significantly increased risk of developing non-Hodgkin lymphoma. That is why they should be carefully monitored throughout their lives.

OTENTIAL MARKERS OF MELANOMA PROGRESSION: PRELIMINARY STUDY ON NODULAR MELANOMA

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Melanoma is one of the most aggressive cancers with a constantly increasing incidence, the migration, invasion and growth of which depend on adhesive and proteolytic mechanisms of neoplastic cells. Matrix metalloproteinases (MMPs) involved in degradation and remodeling of surrounding tissue play a critical role in tumor progression. Laminin and galectin-3, involved in cell adhesion, migration and growth are also substrates for MMP-2 and MMP-9. Therefore, we presumed that they might be used as potential markers of melanoma progression. Protein expression patterns of MMP2, MMP9, laminin, and galectin-3 were determined by immunohistochemical analysis of tumor tissue obtained from 27 nodular melanoma cases (15 female and 12 male). The values obtained were correlated using Spearman correlation rank.