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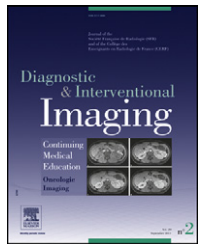

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Lymphomas: Basic points that radiologists should know

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Abstract Lymphomas affect the lymphoid system and may be expressed in a variety of ways and behave in different fashions. The polymorphism of their expression, depending on the organ involved, their variable aggressiveness and their relative rarity compared with primary or secondary diseases sometimes makes it difficult to diagnose them from imaging. Knowledge of predisposing factors and radiological signs should help suggest this diagnosis and thus lead to biopsy samples being taken to confirm it.

Definition and nosological context of lymphomas

Lymphomatous proliferations are defined as the clonal malignant proliferation of a mature lymphocyte from a secondary lymphoid structure, a lymph node or an extranodal structure (the spleen, structures attached to the mucosae such as Peyer's patches). They thus contrast with acute leukaemias or myeloproliferative syndromes arising from an immature cell of medullary origin. They are malignant variants of lymphocytes stuck at a specific stage in their differentiation, with their own morphological and immunophenotypic characteristics. Since there are many of these stages, malignant lymphomas cover a range of very heterogeneous conditions in terms of their forms of presentation, their development profile and their prognosis. We shall describe a certain number of aspects of these lesions according to the organs affected and their differential diagnoses.

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In 2008, more than 355,000 new cases of non-Hodgkin's lymphomas (NHL) were recorded worldwide, which were responsible for 191,400 deaths [1]. Following a period of increase until the 1990s, mainly due to NHL associated with AIDS, the incidence in Western Europe has stabilised at around 10.5 and 7.4/100,000 in men and women respectively. NHLs are more common in developed countries. In France, 10,800 new cases were diagnosed in 2010, NHLs making up 3% of all cancers, and in sixth position in terms of frequency for women and seventh for men.

The causes of the condition are still not known. Certain lymphomatogenic factors — viral (HCV, HIV, HTLV-1, EBV, Herpes virus) and bacterial (*Helicobacter pylori*) — have been recognised, most often restricted to certain types of lymphoma. Other risk factors are chronic immunosuppression, particularly of drug origin (post-transplantation), exposure to certain substances (dioxin, agricultural pesticides) or a history of chemotherapy (alkylating agents).

Treatment is based on a variable combination of chemotherapy, radiotherapy and immunotherapy, adapted to each type. In addition to classic staging of the disease, knowledge of the various extranodal presentations is essential in order to avoid any inopportune corticosteroid therapy or unnecessary surgery before diagnostic confirmation.

How are they classified?

The diagnosis of lymphoma is based on pathological histology. Conventional examination, in the first instance, distinguishes on a morphological basis between Hodgkin's lymphoma (HL), characterised by the presence of Reed-Sternberg cells (40% of lymphomas), and non-Hodgkin's lymphomas (NHL) (60%), classified according to criteria concerning their architecture (follicular or diffuse) and morphology (small or large cells). An immunophenotypic examination should also be used to determine the B- (85% of cases) or T-cell (15%) origin, along with cytogenetic and biomolecular investigations to look for acquired characteristic translocation anomalies of the different types (e.g. *t* (14;18)(q32;q21) of follicular NHLs). According to the WHO classification update in 2008 [2], this has resulted in 43 different conditions divided into four main groups (mature B-cell, mature T-cell and NK-cell neoplasias, Hodgkin's lymphoma and post-transplant lymphoproliferation disorders).

There are several ways for the radiologist to try to learn the broad outlines of this classification:

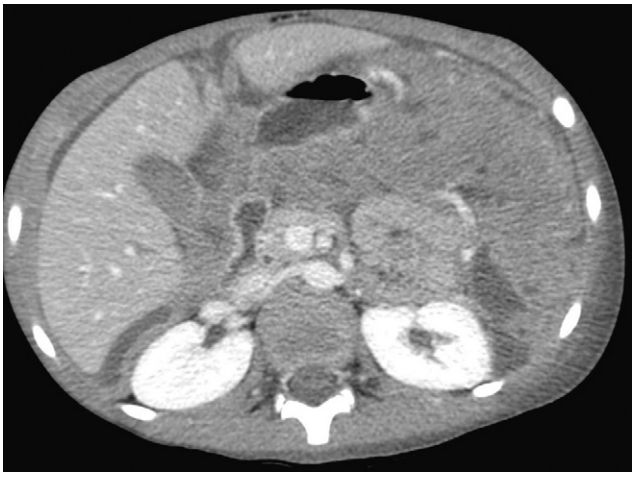

Figure 1. Burkitt's lymphoma with a large tumour mass in a child. CT scan with injection. Diffuse infiltration of the mesentery, the loops of the small intestine and the peritoneum, particularly in the paracolic gutters and the lesser omentum.

- through typical presentations or development profiles, the approach to treatment following directly from them (Table 1). It is simpler then to distinguish two groups:
  - aggressive lymphomas (physiologically or clinically) in particular with high Ki67 proliferation marker expression (Burkitt's lymphoma, diffuse large B-cell lymphoma), a large tumour mass (Fig. 1), an aggressive clinical presentation with general signs (peripheral T and mantle cell lymphomas), rapid development [3] and requiring major treatment,
  - indolent lymphomas of which 80% are slow growing follicular B-cell NHLs, which may transform in 9—10 years' time into high grade lymphomas (15 to 40%), where the decision concerning treatment will depend on the type and mass of the tumour [4];
- through being aware of the preferential frequency of the types of lymphomas depending on age, the patient's condition or the extranodal location: Burkitt's lymphoma and lymphoblastic lymphoma in children and young adults, MALT lymphoma of the stomach, T-cell lymphoma of the small intestine with coeliac disease, mantle cell lymphoma or splenic marginal zone lymphoma in the case of isolated splenomegaly, high grade B-cell NHL (immunoblastic, large cell, Burkitt) and

Table 1 Classification and main development profiles of non-Hodgkin's lymphomas.

Behaviour	Indolent	Aggressive	Highly aggressive
Type	Small cell lymphoma Follicular L (grade 1-2) Lymphoplasmacytic L Splenic/marginal zone lymph node L	Diffuse large B-cell NHL Follicular L (grade 3) Mantle cell lymphoma	Burkitt B-cell lymphoblastic L High grade B-cell L T-cell lymphoblastic L
Survival without treatment	Years	Months	Weeks
Curability	Generally not	Some	Some
Treatment	Delayed	Yes	Yes
Presentation	Lymph node	Medullary compression Horner's syndrome	High tumour mass Superior vena cava obstruction