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Lymphomas: Basic points that radiologists should know

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Oncology; Lymphoma; Lymph nodes; Biopsy; CT scans

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.

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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 lymphomagenic 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:

- 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

![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.](image)

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

<table>
<thead>
<tr>
<th>Behaviour</th>
<th>Indolent</th>
<th>Aggressive</th>
<th>Highly aggressive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type</td>
<td>Small cell lymphoma</td>
<td>Diffuse large B-cell NHL</td>
<td>Burkitt</td>
</tr>
<tr>
<td>Survival without treatment</td>
<td>Follicular L (grade 1-2)</td>
<td>Follicular L (grade 3)</td>
<td>B-cell lymphoblastic L</td>
</tr>
<tr>
<td>Curability</td>
<td>Lymphoplasmacytic L</td>
<td>Mantle cell lymphoma</td>
<td>High grade B-cell L</td>
</tr>
<tr>
<td>Treatment</td>
<td>Splenic/marginal zone lymph node L</td>
<td>T-cell lymphoblastic L</td>
<td>T-cell lymphoblastic L</td>
</tr>
<tr>
<td>Presentation</td>
<td>Years</td>
<td>Months</td>
<td>Weeks</td>
</tr>
<tr>
<td>Generally not</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Delayed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lymph node</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High tumour mass</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Superior vena cava obstruction</td>
<td></td>
<td></td>
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</table>
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HIV, B-cell NHL and transplantation (Table 2) [5]. Definitive typing however will still depend on histopathological data.

The radiologist confronted with lymphoma

The radiologist will be confronted with two situations. The first and most frequent is staging and monitoring the disease. CT scanning is the crucial technique for imaging, as it assesses the extent of the disease according to the Ann Arbor 4-stage classification (Table 3), then allows it to be monitored according to Cheson’s 2-dimensional classic oncological criteria [6]. Depending on the location, ultrasound and MRI (central nervous system) will be indicated in addition. The results of the laboratory tests, osteo-medullary biopsy and possibly of a lumbar puncture will be added to this imaging-based classification, along with the clinical parameters for the prognostic indices for therapeutic management (the International Prognostic Index [IPI] score for aggressive NHLs, and the Follicular Lymphoma International Prognostic Index [FLIPI] for indolent lymphomas).

Whole body diffusion-weighted MRI has still to find its place in onco-haematology and at present is not recommended in current practice. The results appear promising. Most comparative studies are based on 18FDG PET as the reference technique. Whole body diffusion-weighted MRI has produced comparable results in staging Hodgkin’s and aggressive lymphomas, even better results for indolent lymphomas or in detecting visceral and bone locations [7,8]. Preliminary results concern small series and remain to be validated. The place of 18FDG PET has currently been validated for Hodgkin’s lymphomas and diffuse large B-cell NHL and will be addressed in another section.

The second situation is the discovery in imaging of tumour lesions where histopathological confirmation of the possible lymphomatous origin is required.

Forms of presentation

The lymph node form

The most well-known form of lymphoma is the lymph node form (Fig. 2). This is the classic form of Hodgkin’s lymphoma and low grade NHLs. Any lymph node area can be affected. A lymph node with a short axis of more than 1 cm is considered to be pathological.

Certain complications may also reveal the disease, such as superior vena cava obstruction, medullary compression or Horner’s syndrome. These are then aggressive forms of NHL [9].

The spleen, a lymphoid structure, is considered to be a lymphatic extension of the disease. It is involved in the initial stage in 30–40% of patients with Hodgkin’s disease and in 10–40% of those with NHL. Splenic locations mainly present as hypodense nodules of variable size (a few mm to several centimetres). A CT scan is effective for detecting splenic involvement, with values close to 90%, based on the detection of hypodense splenic nodules or a splenic index greater than 725 cm³ (L × W × T). When combined with 18FDG PET, sensitivity reaches 100% with 95% specificity [10].

Presentations by organ

Extranodal lymphomas, particularly in cases of NHL, can involve any organ. While secondary extension from a

Table 2

<table>
<thead>
<tr>
<th>Context</th>
<th>Frequent types</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coeliac disease and jejunal site</td>
<td>T-cell lymphoma</td>
</tr>
<tr>
<td>Skin involvement</td>
<td>Epidermotropic T-cell lymphoma</td>
</tr>
<tr>
<td>Child Isolated splenomegaly</td>
<td>Burkitt’s L, lymphoblastic L</td>
</tr>
<tr>
<td>HIV</td>
<td>Mantle cell L, splenic marginal</td>
</tr>
<tr>
<td>ENT</td>
<td>zone L</td>
</tr>
<tr>
<td>Post-transplantation</td>
<td>Burkitt’s L and buccal cavity</td>
</tr>
<tr>
<td>Gastric lesion and H. pylori</td>
<td>Diffuse large B-cell NHL</td>
</tr>
<tr>
<td>Auto-immune (Sjögren, Hashimoto)</td>
<td>B-cell NHL linked to EBV</td>
</tr>
<tr>
<td></td>
<td>MALT NHL</td>
</tr>
</tbody>
</table>

Table 3

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Only one supra- or subdiaphragmatic lymph node area affected</td>
</tr>
<tr>
<td>2</td>
<td>Two or more lymph node areas affected on the same side of the diaphragm</td>
</tr>
<tr>
<td>3</td>
<td>Lymph nodes affected on both sides of the diaphragm</td>
</tr>
<tr>
<td>4</td>
<td>Visceral involvement distant from a group of lymph nodes</td>
</tr>
<tr>
<td>Letter A</td>
<td>Absence of general signs (fever, nocturnal sweating, weight loss &gt; 10%)</td>
</tr>
<tr>
<td>B</td>
<td>At least one general sign</td>
</tr>
<tr>
<td>E</td>
<td>Extranodal involvement contiguous with a lymph node lesion</td>
</tr>
</tbody>
</table>

Figure 2. Mediastinal lymph node in stage 2 Hodgkin’s disease. CT scan with injection - coronal slice. Mediastinal and axillary lymph node masses.
disseminated form is the most frequent, isolated primary lesions, although rare, are possible. The criteria for such a primary lesion are based on the absence of distant sites (lymph nodes outside of the adjacent drainage area, spleen, bone marrow or any other distant lymphoid structure) and on the absence of circulating lymphomatous cells, and thus conflict with any possible involvement by contiguity or with a stage IV disseminated lymphoma.

Whatever organ is concerned, these lymphoma lesions will have a number of common imaging characteristics which are bound to suggest this diagnosis, or challenge the more frequent diagnoses of primary or secondary tumours, and indicate that biopsy samples should be taken, thus avoiding unnecessary surgery. The efficacy of radio-guided biopsy is high at around 90% [11].

The digestive tract
Digestive involvement occurs in 10–30% of NHL patients, in decreasing order affecting the stomach, the small intestine, the pharynx, the colon and the oesophagus [12]. Identified risk factors include infection by H. pylori, coeliac disease, chronic inflammatory diseases and post-transplant immunosuppression. The prevalence of the various types of lymphoma varies depending on the site. B-cell and MALT NHLs are the most common. Irrespective of the site, certain common characteristics can be seen: hypovascularity of the lesions (masses, infiltration), the classic absence of occlusive repercussions despite the large size of the lesions due to the absence of an associated desmoplastic reaction, preservation of the fat planes, multifocal lesions, associated lymph node masses [13].

The stomach is the most frequent site of extranodal lymphomas. In this location, they are mainly MALT lymphomas associated with chronic infection by H. pylori with a low to high grade sequence, followed by diffuse large B-cell NHL. While there is normally no lymphoid structure in the stomach, chronic H. pylori infection is associated with the development of lymphoid structures in the lamina propria which are the origin of the lymphomatous process. Diagnosis is essentially by endoscopy. Radiology techniques may show gastric involvement as ulcers (50% of cases), masses (36%) or mucous nodules with central ulceration [14]. The differential diagnosis is gastric adenocarcinoma. In the case of parietal infiltration with thickening of the folds, retained gastric distensibility is an argument against gastric linitis (Figs. 3 and 4). Preservation of perilesional fat planes, particularly where there is a large mass, and the presence of voluminous lymphadenopathies beyond the adjacent drainage area (under the renal hilum) point to a lymphoma.

In the small intestine, B-cell NHLs predominate in the ileum, T-cell NHLs being more frequently found in the jejunum associated with coeliac disease [15]. Different basic forms are possible: single or multiple mucous nodules, diffuse or focal parietal infiltration and thickening. The most suggestive is aneurysmal parietal thickening (Fig. 5). The differential diagnoses are mainly adenocarcinoma and stromal tumours (Fig. 6). A widespread or multifocal appearance, the absence of occlusive repercussions despite a large tumour volume, and the absence of hypervascularisation are signs pointing towards a lymphoma. In the large intestine, predominantly in the caecal and rectal regions,

The liver
Unlike the visceral hepatic extension of stage IV lymphomas, present in 15% of cases, primary lymphomas of the liver are rare (<1% of extranodal lymphomas) [17]. They are mainly large B-cell NHLs. The other types described (immunoblastic, lymphoblastic, Burkitt’s, MALT lymphomas) represent less than 5% of cases. Aetiological factors reported include HCV (21% of cases in France), EBV and HIV infection, as well as auto-immune diseases for MALT lymphomas [18,19]. They are typically discovered at a mean of 55 years (5–87 years), with a male/female ratio of 2.3/1, in a context of
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Figure 5. Forms of B-cell non-Hodgkin’s lymphomas found in the small intestine. CT scan with injection. a: infiltration of the final loop and contact mesenteric extension. Absence of occlusion despite the size of the tumour; b: circumferential involvement of the final loop. The ileal lumen is preserved; c: aneurysmal form. Slightly enhanced circumferential thickening contrasting with the luminal distension at this point.

Figure 6. Differential diagnoses of lymphomas of the small intestine. CT scan with injection. a: aneurysmal shaped stromal tumour. Unlike in lymphomas, the tumoral wall has a vascularised appearance with an increase in the calibre of the vessels supplying it; b: adenocarcinoma of the small intestine. Stenosing jejunal thickening associated with infiltration of the mesentery at this point.
abdominal pain or discomfort [20]. Jaundice is uncommon (10–20% of cases). The often large, solitary nodular form, which can measure more than 10 cm, is the most common (50–60% of cases), followed by the multinodular form in 40% of cases, the diffuse infiltrating form being exceptional [21,22]. With ultrasound, the nodules are usually hypoechoic, sometimes anechoic. In a CT scan, these spontaneously hypodense lesions exhibit variable behaviour after injection (absence of enhancement in half of the cases, in patches for one third, possibly with a necrotic centre, ring enhancement in 16–29% of cases, with a trend towards isodensity in the late stage) (Fig. 9). After chemotherapy, calcification is possible. In MRI, there is spontaneous T1 hypointensity, often marked T2 hyperintensity, as with diffusion weighting, enhancement being again variable. Forms of diffuse infiltration are possible [21]. Given its rarity, the most frequent diagnoses suggested are hepatocellular carcinoma, particularly when there is hepatitis C, and hepatic metastases. Median survival time in the literature is 15.3 months [20,23]. After liver transplantation, two forms are described: a rapidly progressing precocious form, mainly involving the pedicul, region and which may locally invade the liver in connection with EBV and respond favourably to a reduction in or discontinuation of immunosuppressants, and a later, EBV negative form with a poorer prognosis.

The pancreas

While peripancreatic and pancreatic extensions of a retroperitoneal or mesenteric lymphomatous lymph node infiltration do not in general pose a problem, isolated involvement of the pancreas can still be a diagnostic trap (Fig. 10). Analysis of the literature shows that the diagnosis was made from biopsies in 28% of cases and by laparotomy in 65% of cases. Ablation surgery was performed in 21% of cases [24].

Pancreatic lymphomas are rare, representing less than 1% of NHL. They make up only 1.3 to 1.5% of malignant
pancreatic tumours [25,26]. They are more common in men (male/female ratio 13/3). Clinical signs are not very specific, being mainly weight loss and abdominal pain. An epigastric mass was however observed in more than half of the cases and measured more than 6 cm in 70% of patients [27]. Imaging characteristics are those common to lymphoma lesions: hypochic masses that are hypointense with poor enhancement, often well delimited, and usually homogeneous. Certain diffuse infiltrating forms (12% of cases) may look like acute pancreatitis, with enlargement of the gland, peripancreatic infiltration and elevation of blood amylase, but without the clinical symptoms of pancreatitis. Enlargement of the gland can sometimes lead one astray towards a diagnosis of auto-immune pancreatitis. In MRI, the homogeneous nature, more marked hyperintensity with T2-weighting, the absence of late enhancement and of a capsule point towards a lymphoma [28]. No case of calcification has been described in the absence of treatment. Laboratory results are not very specific, with CA19-9 levels being normal or slightly raised. However, when faced with a pancreatic mass, various signs should attract attention, including a discordant large mass with no jaundice despite a predominantly cephalic location, moderate dilatation of the pancreatic duct, few vascular repercussions, a raised serum LDH concentration, or lymphadenopathies present below the level of the renal veins, and suggest the possibility of a lymphoma rather than an adenocarcinoma [29,30].

Urogenital involvement
In cases of known lymphoma, while renal involvement is frequent, being present in nearly a third of cases, and reaching 30 to 60% on autopsy [31], it is rarely symptomatic (pain in the side, haematuria, nocturnal sweating, fever). It usually occurs during extension of diffuse NHL (intermediate to high grade B-cell NHL, Burkitt’s lymphoma). Diffuse infiltration can also be the cause of renal impairment by compressing the tubules. Despite this high frequency, less than 8% of patients have lesions detected by CT scans. Most renal involvement results from haematogenous dissemination or extension from the neighbouring retroperitoneum, and isolated primary lesions are rare (< 1% of extranodal lymphomas) [32]. The most common forms are intermediate or high grade B-cell or Burkitt’s lymphomas. Various forms are described in imaging: intraparenchymal hypoechoic multiple nodules or solitary masses, only slightly enhanced following administration of contrast agent, perirenal infiltration, infiltrative nephromegaly. In a CT scan, acquisition in the

![Figure 8. Differential diagnosis of colic forms. a: undifferentiated carcinoma of the right colon with a pseudoaneurysmal form. The colloid forms of colic adenocarcinomas may appear hypodense similarly to lymphomatous infiltrations; b and c: rectal linitis. In the CT scan, the hypodense parietal infiltration is stenosing. In the MRI, there is marked contrast uptake pointing towards a carcinomatous infiltration.](image-url)
Figure 9. Classic forms of lymphoma in the liver. Injected CT scans. a: multiple hypodense nodules of a stage 4 large B-cell NHL; b: large hypodense mass with no distal biliary repercussions, no capsular retraction nor vascular invasion; c: perihilar hypodense infiltration with no vascular repercussions on the portal structures; d: hypodense nodules in a patient with a kidney transplant for polycystosis. The main nodule of the right lobe of the liver has a rosette-like appearance.

Figure 10. Peripancreatic lymphomatous infiltration by a follicular lymphoma.

nephrographic (venous) phase is essential for detection of these hypovascular or poorly enhanced lesions, particularly those in the medullary portion of the kidneys (Fig. 11). The secretory phase is necessary when there is sinus infiltration [33]. In MRI, lymphomatous lesions appear hypointense with T1-weighting and hyperintense with T2-weighting and appear poorly vascularised [34]. The differential diagnoses to consider in the case of isolated lesions are pyelonephritic foci, usually hyperdense in the delayed phase, renal emboli and infarction, and renal metastases (breast, stomach, melanoma). Where there is a solitary tumour, the hypovascular character and the absence of venous invasion can suggest a lymphoma and indicate that the tumour should be biopsied. Isolated perirenal lesions in the form of tissue sheathing and/or compressing the kidney are rare. Diffuse infiltration of the kidneys is still a form which is difficult to diagnose. Combined with nephromegaly, a loss of corticomедullary differentiation, infiltration of the sinus fat, or sheathing of the cavities may be seen.

Primary testicular lesions occur between 60–70 years of age as gradual painless hypertrophy. It is the commonest aetiology for testicular masses after the age of 60 [35]. With ultrasound the lesion appears as infiltration or a
hypoechoic mass. Ovarian lesions are B-cell NHLs, and are found particularly in the context of Burkitt’s lymphoma [36].

The thorax

Thoracic lymphomas are the main expression of mediastinal lymph node involvement which is present in 80% of HLs [37]. Pulmonary involvement is only found in 5% of HL patients. Primary thoracic extranodal lesions are mainly MALT NHLs. This type is typically found in the stomach, the small intestine, the eye and salivary glands and is associated with auto-immune diseases (Hashimoto’s thyroiditis). While adenomegalies are frequent in the initial stage if there are pulmonary lesions of HL, an isolated pulmonary lesion is possible in NHL [38]. The main pulmonary forms mimic many tumoral or inflammatory conditions and include nodules and masses with or without cavitation, condensations, ground glass opacities, endobronchial masses and reticular interstitial syndrome (Fig. 12) [39]. In the particular context of immunosuppression, the most frequently encountered lesions are multiple nodules. Pleural involvement in the form of thickening, isolated or multiple nodules occurs in 16% of NHLs, essentially as disseminated or recurring forms [40]. Effusions are more common, either through direct lesions or by lymphatic obstruction originating from lymph nodes.

Cardiac involvement is rare, whether by direct extension (corresponding to Ann Arbor stage E) or haematogenous or lymphatic diffusion (Fig. 13). Right atrial cavity lesions are the most frequent, often associated with extension involving more than one cavity and the pericardium. In MRI these masses appear isointense with T1-weighting, heterogeneously hyperintense with T2-weighting and are heterogeneously enhanced after gadolinium injection [41]. The prognosis for these forms is poor.

The central nervous system

The CNS is involved in 10–15% of systemic lymphomas, mainly in the form of a leptomeningeal lesion in large B-cell NHLs. Primary lesions represent less than 1% of cases of NHL. The most common locations for the single or multiple masses are the deep grey matter, the corpus callosum, the
Figure 12. CT scans of pulmonary lymphoma lesions. a: irregular MALT lymphoma nodule; b: diffuse B-cell NHL nodule; c: atelectatic condensation of a MALT lymphoma; d: right lower lobe excavated opacity and posterior segmental condensation of the right middle lobe in Hodgkin’s disease.

Periventricular and subependymal zones, and the cerebral hemispheres. MRI is the preferred technique for exploring and staging central nervous system lymphomas. In MRI, these lesions appear iso- or hypointense with T1 weighting, enhancing after injection (Fig. 14) [42]. The principal differential diagnoses are secondary lesions and abscesses where there are multifocal lesions, and glioblastomas in pericentral sites.

Spinal forms include epidural extensions from a perispinal or spinal column lesion and focal or disseminated extra and intramedullary intradural lesions. The main differential diagnoses are metastases of intra or extranervous origin and granulomatoses (neurosarcoidosis).

ENT involvement concerns Waldeyer’s ring structures (the tongue, palate and tonsils), the sinus cavities and the orbit. The absence of lymph node necrosis and contact bone erosion points to a lymphomatous disease rather than a squamous cell carcinoma. When the salivary glands or thyroid are affected, there is frequently an associated auto-immune condition (Sjögren, Hashimoto).
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Bone and soft tissue involvement
There is bone involvement in 5% of diffuse lymphomas. These cases only represent less than 5% of primary bone tumours and are essentially NHLs [43, 44]. The lesions are typically lytic (70%) but mixed forms have been reported. A solitary metaphyseal/diaphyseal lesion with a periosteal reaction (60% of cases) associated with a soft tissue mass is suspect. In MRI, definite hyperintensity in T2-weighted and STIR sequences and T1-weighted hypointensity suggests these lesions [45].

Conclusion
Primary forms of lymphoma can be diagnostic traps because of their polymorphism and rarity. Lymph node forms occur most frequently. CT scans are the crucial radiological exploration technique for staging and monitoring.

**TAKE-HOME MESSAGES**
- The lymph node form of lymphomas is the most frequent.
- Primary extranodal lymphomas are rare and pose problems of differential diagnosis with primary or secondary lesions. Certain common signs will suggest this condition: a frequent hypovascular appearance, discordance between the volume of the tumour and the moderate repercussions on the actual organ (digestive tube, liver, kidneys).
- A biopsy, usually guided by imaging, will confirm the diagnosis and type the lymphoma.
- Certain situations (post-transplantation, immunosuppression, coeliac disease, *Helicobacter pylori* infection) are conducive to the development of lymphomatous conditions and should evoke the diagnosis.
- Staging using imaging is based on a thoracic-abdominal-pelvic CT scan.
- Extension of the disease is assessed using the Ann Arbor classification.
- Therapeutic management is based on typing, staging and the clinical-laboratory scores.

Clinical case
You are confronted with four hypodense hepatic lesions in the portal phase found in a CT scan (Figs. 15–18).

Questions
1. What is your analysis of the images?
2. What are your hypotheses?
3. What are the signs for or against a lymphomatous origin?
Answers

These four lesions appear hypodense in the portal phase. Certain criteria will act as pointers.

Patient 1 (Fig. 15) has a large lesion with discretely lobulated contours. No vascular structure is visible within it. The liver does not appear to be dysmorphic. The absence of contrast uptake by fibrous stroma does not point towards an intrahepatic cholangiocarcinoma. In the first instance, a secondary hepatic lesion is suggested.

Patient 2 (Fig. 16) has a large lesion in the right lobe of the liver. There is central calcification and clumps of peripheral enhancement can be seen. Calcifications within lymphomatous lesions are rare and found following treatment. An angioma is strongly suspected here. Additional MRI will provide the diagnostic confirmation.

Patient 3 (Fig. 17) has signs of chronic liver disease with dysmophia and portal hypertension. In the first instance therefore, this suggests a primary hepatocellular carcinoma with washout in the portal phase. To comply with the non-invasive diagnostic criteria of HCC with cirrhosis, arterialisation is necessary prior to washout to confirm the latter (CT scan or MRI).

Patient 4 (Fig. 18) has a discretely hypodense homogeneous lesion. No capsular or biliary repercussions are visible. Above all, there are no distortions within it in size or direction of the vascular structures of the portal and hepatic veins. This is highly suggestive of an infiltrating lymphoma. MRI may show hyperintensity in a T2-weighted sequence and diffusion of the lesion. A biopsy will provide the diagnosis (here a lymphoplasmacytic lymphoma).
Figure 18. Man of 60 years of age. CT scan with injection - axial slice: MPR (a) and MIP (b). Hypodense nodule in the right lobe of the liver discovered by chance.

Disclosure of interest

The author declares that he has no conflicts of interest concerning this article.

References