Pseudotumors and Related Lesions of the Hepatobiliary Tract

119

Contents

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Abstract

Pseudotumors and inflammatory pseudotumors form a heterogeneous group of lesions characterized by the presence of an inflammatory infiltrate and proliferations of fibroblasts and myofibroblasts. Pseudotumors are mass-forming lesions, also in the hepatobiliary tract, that may mimic diverse malignancies. Specifically, pseudotumors can grow in the walls of extrahepatic bile ducts, with formation of stenosing and obstructive lesions clinically similar to cholangiocarcinoma. The term inflammatory myofibroblastic tumor (IMT) has been proposed as synonym for pseudotumor, with a generally benign behavior. However, a subset of these lesions show an aggressive growth pattern and even malignant behavior, suggesting a neoplastic character of some pseudotumors. Furthermore, about half of recently analyzed cases of IMT carry rearrangements of the anaplastic lymphoma kinase (ALK) locus on chromosome 2p23, causing aberrant ALK expression. Based on these findings, the classification of so-called pseudotumors, mainly the identification of reactive versus neoplastic forms, requires revision. A variant lesion that is sometimes classified together with IMT is Epstein-Barr virus-positive inflammatory pseudotumor (inflammatory pseudotumor-like follicular dendritic cell tumor).

Hepatobiliary Pseudotumors: A Heterogeneous Group of Intriguing Lesions

Introduction

Hepatic lesions resembling or being identical with inflammatory pseudotumors observed in other organs and in particular the lung of young male patients are now well recognized. The first reports on primary hepatic inflammatory pseudotumor (HIP; synonyms: pseudolymphoma, fibroinflammatory tumor, inflammatory myofibroblastic tumor, plasma-cell granuloma, inflammatory pseudotumor-like granuloma) date back to the 1950s–1970s (Pack and Baker 1953; Hertzer et al. 1971; Someren 1978), the case of Pack and Baker representing a 40-year-old man. Since that time, many cases have been described, albeit mostly as single reports or small series hepatic inflammatory pseudotumors (HIP; review: Craig 1997) now become increasingly recognized because of the more frequent use of advanced abdominal imaging methods, in particular ultrasonography and CT. But what is a "pseudotumor" ?

Pseudotumors: A General Overview

The term pseudotumor is, as such, rather a misnomer and may in fact be a very inaccurate term: a tumor, in its original meaning, is a mass lesion of whatever cause and composition, although now being more and more reserved for a bona fide neoplasm; so, what is a "pseudo-mass," and what is an adequate definition of it ? "The term is typically employed to connote the presence of a mass lesion (which, by definition, is a tumor)" that on clinical or pathologic grounds, or both, is thought to represent a neoplasm. Thus, the name "pseudoneoplasm" would be more à propos under such circumstances (Wick and Ritter 1997). The two authors just cited employ the term, "pseudoneoplastic lesions," but they will be right in their introductory statement that, even though the term is utterly unsatisfactory, it will remain in the active medical literature, because of the difficulty that is generally encountered in challenging an established clinicopathologic rubric. First employed to denote orbital masses producing exophthalmos (Birch-Hirschfeld 1930), pseudotumors have later been subdivided in accordance with predominant histologic features, including "inflammatory," "granulomatous," "xanthomatous," " plasmacellular," and "fibrous" variants. It has to be emphasized that, even though such adjectives may offer some (primary or first line) help in classification, they unfortunately surmise that the lesions so denoted may represent "entities." This approach operates such as general pathology or pathophysiology would not exist: it is obvious that a reactive process driven by an

exuberant inflammatory response may later develop fibrosis, or may accumulate numerous lipid-laden macrophages subsequent to hemorrhage or necrosis, classifying adjectives and therefore identifying a type of response within a timely sequence rather than an inherent feature. More interesting (and probably also more important) is the phenomenon as to why such apparently nonneoplastic lesions exhibit excessive growth, may show invasive characteristics, and may or may not spontaneously regress, in some way reflecting the behavior of an abnormally healing wound.

Inflammatory Pseudotumor Versus Inflammatory Myofibroblastic Tumor

The term inflammatory myofibroblastic tumor (IMT) has been proposed as a synonym of inflammatory pseudotumor and characterized as lesions containing myofibroblastic spindle cells and a mixed pattern of leukocytic infiltrates, including plasma cells, and exhibiting a generally benign behavior. The majority of lesions previously described as inflammatory pseudotumors meet this definition, but cases reported in past decades were described as having "spindle cells" often interpreted as fibroblasts, because the myofibroblast concept and the immunohistochemistry to identify myofibroblasts were not yet developed. However, the spectrum of IMT and related lesions has recently evolved along several lines, causing some misconceptions or even confusion regarding classification. First, a subset of IMT show an aggressive growth pattern and a malignant behavior rather than a benign phenotype, sometimes with formation of metastases ("aggressive form of IMT"). Based on this behavior, some IMTs were classified as neoplastic lesions, or reactive lesions, that have undergone malignant transformation. Secondly, about half of recently analyzed cases of IMT carry rearrangements of the anaplastic lymphoma kinase (ALK) locus on chromosome 2p23, causing aberrant ALK expression. As these lesions markedly deviate from the "purely" inflammatory and mixed-cellular forms of inflammatory tumors, mainly due to their neoplastic nature, these lesions are treated in a separate chapter.

Pseudotumors as Neoplastic Versus Reactive Processes

Is HIP a reactive or a neoplastic process? Some features have been quoted that seemingly contradict the purely reactive/inflammatory nature of these lesions, including a progressive growth; a local recurrence; the development of noncontiguous, multifocal masses; an infiltrative border; the destruction of liver substance and bile ducts; and the vascular (portal-venous) invasion (Coffin et al. 1998). However, the histologic pattern of the majority of the lesions, lacking one typical target cell, their usually benign course, and the tendency for spontaneous regression do, on the other hand, not favor a bona fide neoplastic process. We therefore propose that the majority of HIPs are in fact reactive lesions, i.e., pseudotumors, but that a subset of hepatic lesions with a similar morphology have a neoplastic phenotype, in particular those with a proliferation of follicular dendritic reticulum cells. These lesions are discussed in the following chapter. In addition, there might be instances where part of a mass exhibits the histologic features of HIP, but this component may represent a reaction to a hidden and later overt malignant neoplasm, e.g., a sarcoma, thus initially causing the false impression of malignant HIP.

Inflammatory Pseudotumor of the Liver (Hepatic Inflammatory Pseudotumor, HIP; Hepatic Inflammatory Myofibroblastic Tumor, HIMT), Probably Reactive Forms (Excluding IgG4-Related Disease)

Introduction

The lesions described in this section comprise reactive phenotypes that, on the one hand, correspond to what has been described as inflammatory pseudotumors in the "pre-myofibroblast era" and

to lesions now termed inflammatory myofibroblastic tumors, however with the exception of those which have documented ALK gene rearrangements, expression of EBV, and/or signs of a bona fide neoplastic cell lineage. Also inflammatory pseudotumors developing in the setting of systemic IgG4 sclerosing disease are excluded from this section and are discussed in a separate chapter. As specified above, "type A" as a working formulation denotes a heterogeneous group of reactive and probably nonneoplastic lesions that awaits further classification. For this purpose, the older term, inflammatory pseudotumor, is employed in this section, also for comparative lesions in regard to the older literature.

Epidemiology

HIP has been first described in 1953 (Pack and Baker 1953) and has since been documented numerous times in the literature, mostly under the term inflammatory pseudotumor, notwithstanding the fact that the term inflammatory myofibroblastic tumor was already coined in 1995. HIP occurs at all ages. The age of the patients at presentation of the lesion ranges from infancy (3 months; Lee and DuBois 2001; 9 months; Lacaille et al. 1999) or early childhood up to more than 80 years, with a male to female ratio varying considerably from one series to other, from about 1:1 up to 8:1 (Horiuchi et al. 1990; Shek et al. 1993). HIP occurring in the pediatric age group has been reported several times (Someren 1978; Newbould et al. 1992; Passalides et al. 1996; Bankole-Sinni et al. 1997; Ueda et al. 2003; Swinney et al. 2006). The overall prevalence of HIP is not known, but in a study on 403 consecutive patients carrying a total of 717 focal liver lesions and undergoing liver resection, three patients proved to have HIP accounting for 0.7 % of all patients and 0.4 % of all focal liver lesions (Torzilli et al. 2001). The features of the lesions have been described in numerous reports.

Selected References Pack and Baker 1953; Hertzer et al. 1971; Someren 1978; Heneghan et al. 1984; Anthony and Telesinghe 1986; Grouls 1987; Lee et al. 1987; Morita et al. 1987; Collina et al. 1987; Kessler et al. 1988; Joh et al. 1988; Levitt et al. 1988; Hirasawa et al. 1988; Standiford et al. 1989; Jimenez-Mejias et al. 1989; Irie et al. 1989; Li et al. 1989; Lupovitch et al. 1989; Andreola et al. 1990; Horiuchi et al. 1990; Imafuku et al. 1990; Jakab et al. 1990; Imazato et al. 1990; Tsao et al. 1990; Lopez et al. 1990; Feng 1991; Alonso et al. 1991; Isobe et al. 1991; Jackson and Gatling 1991; Pokorny et al. 1991; Gollapudi et al. 1992; Hata et al. 1992; Lopez 1992; Ozeki et al. 1993; Shek et al. 1993; Fukuya et al. 1994; Maeng et al. 1994; Noi et al. 1994; Jais et al. 1995; Schmid et al. 1996; Uetsuji et al. 1996; Gosavi et al. 1997; Kafeel and Telesinghe 1997; Nonomura et al. 1997; Ogawa et al. 1998; Tajima et al. 1998; Gluszek et al. 1999; Kim et al. 1999; Lacaille et al. 1999; Voss et al. 1999; Yoon et al. 1999; Ishida et al. 2000; Toda et al. 2000; Kaneko et al. 2001; Lee and DuBois 2001; Levy et al. 2001; Sakai et al. 2001; Torzilli et al. 2001; Yan et al. 2001; Casassus-Builhe et al. 2002; Sakai et al. 2002; Saito et al. 2002; Wang et al. 2002; Biecker et al. 2003; Inaba et al. 2003; Koea et al. 2003; Maiga et al. 2003; Rai et al. 2003; Schneider et al. 2003; Aguirre-Garcia 2004; Del Fabbro et al. 2004; Fritzsch et al. 2004; Lo et al. 2004; Papachristou et al. 2004; Seki et al. 2004; Tamsel et al. 2004; Alimoglu et al. 2005; Bernard et al. 2005; Cohen et al. 2005; Colakoglu et al. 2005; Druez et al. 2005; Locke et al. 2005; Nishimura et al. 2005; Sandilla et al. 2005; Sasahira et al. 2005; Teranishi et al. 2005; Akatsu et al. 2006; Kawamura et al. 2006; Kim et al. 2006; Koide et al. 2006; Park et al. 2006; Schuessler et al. 2006; Swinney et al. 2006; Diaz-Torné et al. 2007; Gohy et al. 2007; Hoosein et al. 2007; Kai et al. 2007, Perera et al. 2007; Schnelldorfer et al. 2007; Sturm et al. 2007; Tsou et al. 2007; Vassiliadis et al. 2007; Weiss et al. 2007; Yamaguchi et al. 2007; Alfieri et al. 2008; Chen et al. 2008; Peddu et al. 2008; Singh et al. 2008; Ashcroft et al. 2009; Chong et al. 2009; Ganesan et al. 2009; Geramizadeh et al. 2009; Goldsmith et al. 2009; Jover Diaz et al. 2009; Lee et al. 2009; Manolaki et al. 2009, Milias et al. 2009; Mouelhi

et al. 2009; Ueda et al. 2009; Al-Jabri et al. 2010; Brage-Varela et al. 2010; Bruyeer and Ramboer 2010; Deng et al. 2010; Sari et al. 2010; Tang et al. 2010; Terada 2010; Faraj et al. 2011; Hasan et al. 2011; Herek and Karabulut 2011; Jerraya et al. 2011; Ntinas et al. 2011; Renzing et al. 2011; Kawaguchi et al. 2012; Patnana et al. 2012.

Clinical Features

The lesions are mostly solitary (multiple in about 20 %), more frequently right sided (about 60 %), and located within the liver substance or extended into the hilar/perihilar area. However, multiple nodules of HIP occur and may mimic liver abscesses, primary hepatic malignancy, or hepatic metastases. The size of the lesions is variable but may reach a diameter of up to 25 cm. HIP may clinically present as fever of unknown origin (FUO; mainly low-grade intermittent fever), malaise, vague abdominal symptoms and pain, an epigastric mass, a history of weight loss, jaundice, and/or laboratory data suggesting an inflammatory process (leukocytosis, raised erythrocyte sedimentation rate, polyclonal hyperglobulinemia). In part of the patients, HIP was asymptomatic and was detected as an "incidentaloma" or liver metastasis. In case the masses develop close to large bile ducts or within the bile duct wall, HIP may cause stenosis of intrahepatic bile ducts and sometimes obstructive jaundice, either due to involvement of the hilar compartment of the liver and/or large extrahepatic bile ducts, sometimes mimicking perihilar bile duct carcinoma, or as a disease centered around the intrahepatic biliary tree, or involving both the hepatobiliary and pancreatic system. HIP evolving with a ductocentric pattern can develop a hamartoma-like growth associated with fibrosclerosis, a lesion proposed to be termed "fibroductal variant" of HIP (Terada et al. 1992). HIP can develop in extrahepatic bile ducts, including the mid-portion, and closely mimic cholangiocarcinoma (Vasiliadis et al. 2014). HIP also occurs in the distal part of the common bile duct, i.e., close to the pancreatic head (Haith et al. 1964). A further distinct feature of HIP is endophlebitis, sometimes obliterative, involving medium-sized to large branches of the portal vein and eventually resulting in portal hypertension (Kaneko et al. 1984; Chen 1984; Heneghan et al. 1984; Horiuchi et al. 1990; Tsao et al. 1990), and also developing in young children (Someren 1978), sometimes with the angiographic demonstration of the portal vein draining into the lesion to be occluded (Hata et al. 1992).

In a minority of cases, HIP may undergo spontaneous regression. This regression process may take several months and can occur in the absence of any treatment, owing to unknown reasons, but it may be assumed that HIP regress when the causative factors, in particular infection, have disappeared (Gollapudi et al. 1992; Levy et al. 2001; Biecker et al. 2003; Druez et al. 2005; Koide et al. 2006; Tsou et al. 2007; Yamaguchi et al. 2007; Peddu et al. 2008; Chong et al. 2009; Brage-Varela et al. 2010; Jerraya et al. 2011). Regression of HIP has been achieved by treatment with nonsteroidal anti-inflammatory drugs (Colakoglu et al. 2005; Vassiliadis et al. 2007).

Imaging Features

Ultrasonography usually reveals hypoechoic and well-circumscribed masses (Abehsera et al. 1995; Nam et al. 1996; Sakai et al. 2002). As sonography sometimes reveals enlarged locoregional lymph nodes, a primary hepatic malignancy may be suspected (Fukuya et al. 1994; Schuessler et al. 2006). At non-enhanced CT, HIPs appear as an irregularly shaped, inhomogeneous mass with an internal low-density area in the delayed phase (Noi et al. 1994; Lim and Lee 1995; Lee and DuBois 2001; Nishimura et al. 2005; Goldsmith et al. 2009; Herek and Karabulut 2011) or ill-defined, hypoattenuating lesions, whereas at contrast-enhanced CT, the masses exhibit central hypoattenuating areas with an iso- or hyperattenuating and thickened periphery or a multiseptate appearance (Yoon et al. 1999). HIP may show calcifications (Lacaille et al. 1999). Changes in tumor size during relatively short

time periods are often observed in HIP, in contrast to most other hepatic mass lesions. An enhancement of the peripheral regions of HIP was often observed in the early phase of contrast medium dynamic CT, thought to be due to abnormal vessels located in peripheral parts of the lesions and to result from obliteration of preexisting vessels in portal tracts within the inflamed tissue (Seki et al. 2004). HIP may present capsular retraction as an uncommon imaging fining (Ganesan et al. 2009).

Findings at MRI have been reported several times (Flisak et al. 1994; Kelekis et al. 1995; Borgonovo et al. 1998; Yan et al. 2001; Sakai et al. 2002; Hasan et al. 2011), showing no early arterial enhancement (a key difference to hypervascular HCC), peripheral enhancement, septum and small nodular enhancement occurring in portal venous, and delayed phases. MRI with mangafodipir trisodium might help distinguish HIP from HCC (Materne et al. 1998; Mortele et al. 2002). On post-gadolinium gradient-echo (GE) images, an early, intense, and peripheral enhancement was followed by a homogeneous, complete, and persistent enhancement, and during follow-up, a peripheral intense rim appeared on precontrast T1-weighted images (Mortele et al. 2002). Due to the high content in metabolically active cells of diverse leukocyte lineages, HIP can be diagnosed by PET based on its markedly increased 18 F-FDG uptake (Kawamura et al. 2006; Chong et al. 2009). The high macrophage content of most HIP allows the detection of these lesions by use of ferumoxide-enhanced MR imaging (Kato et al. 2004).

Pathology

Macroscopy

At gross examination, HIPs are rather firm or even hard masses (depending on the collagenization) with usually sharp margins. The cut surface is variegated, gray to whitish or yellow, and sometimes with signs of hemorrhage and/or necrosis (Figs. 1 and 2).

Histopathology

Histologically, the "background" tissue consists of fibroblasts/fibroblastoid cells and myofibroblasts. The cellularity of the spindle cell component may be so abundant that these areas may be misdiagnosed as sarcoma. The spindle cell areas are frequently heavily collagenized, producing a hyaline or sclerosed aspect of the tissue. The vascularity may be high, reflected by the imaging presentation as a vascular tumor (Shek et al. 1993). The tissue is densely infiltrated by a mixed population of leukocytes, usually with a predominance of small lymphocytes. In some instances, the lymphocytic infiltration exhibits a density and a monomorphic presentation

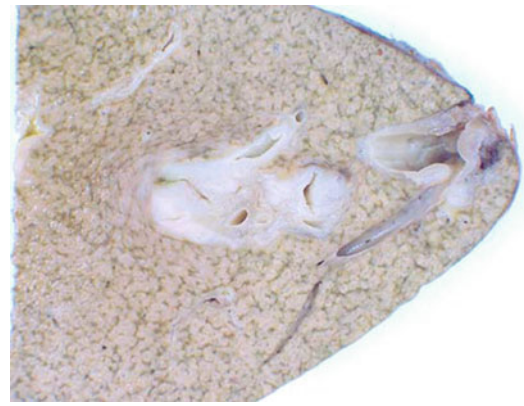

Fig. 1 Inflammatory pseudotumor of the hepatobiliary tract. A whitish tissue has developed around intrahepatic bile ducts, causing duct stenosis

Fig. 2 Stenosing inflammatory pseudotumor of the extrahepatic bifurcation. The lesion macroscopically mimics stenosing cholangiocarcinoma

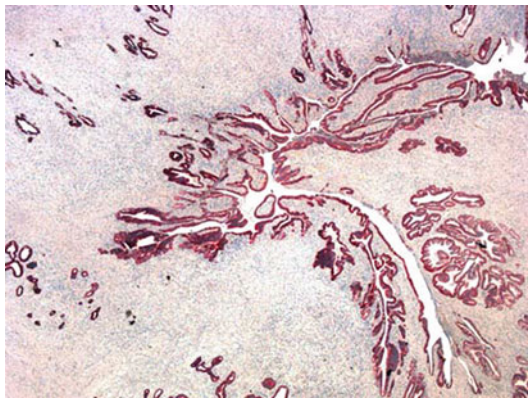

Fig. 3 The extrahepatic bile ducts are markedly stenosed by an inflammatory tissue (cytokeratin 7 immunostain)

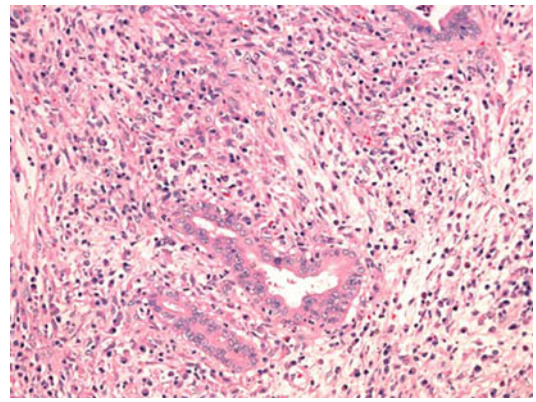

Fig. 4 Hepatobiliary inflammatory pseudotumor. The tissue surrounding bile ducts is infiltrated with lymphocytes, plasma cells, and macrophages, and there is an increase in fibroblastoid cells (hematoxylin and eosin stain)

reminiscent of malignant non-Hodgkin's lymphoma, one reason why such lesions have also been termed pseudolymphoma of the liver (Grouls 1987). Lymphocytes may be arranged as aggregates or lymph follicles, even with formation of germinal centers. In addition to lymphocytes, various amounts of mature plasma cells and plasmacytoid elements are regularly noted and may sometimes form dense clusters or aggregates mainly around blood vessels, previously having led to the term plasma-cell granuloma (Anthony 1993). Neutrophil and eosinophil granulocytes are mostly sparse, with the exception of cases with development of microabscesses or with cholangitic features. As a reaction to tissue breakdown, numerous foamy macrophages may be in evidence, sometimes resulting in a so-called pseudoxanthomatous reaction (Figs. 3, 4, 5, 6, and 7). Some of the tumors contain increased numbers of dendritic cells, including giant atypical reactive dendritic cell forms (Sturm et al. 2007).

Histopathologic diagnosis by needle biopsy is possible and has been reported (Irie et al. 1989; Nakama et al. 2000; Hosler et al. 2004; Sari et al. 2010; Kawaguchi et al. 2012). Fine-needle aspiration shows hypocellular smears with an admixture of various cell types including plasma cells, macrophages, lymphocytes, fibroblasts, and granulation tissue fragments. Foamy macrophages are a striking feature in some cases (Lupovitch et al. 1989; Yoshida et al. 2003; Hosler et al. 2004; Kawaguchi et al. 2012).

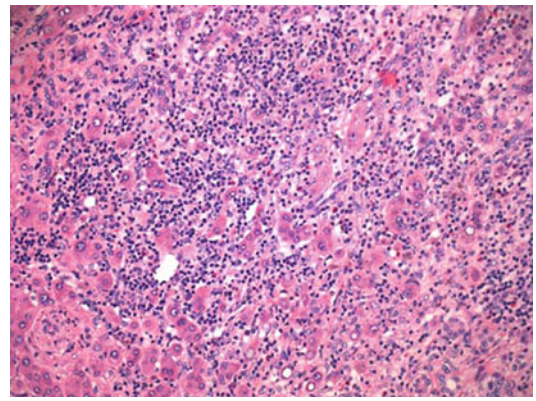

Fig. 5 Hepatobiliary inflammatory pseudotumor. An infiltrate rich in lymphohistiocytic cells has caused dissociation of hepatic parenchyma (hematoxylin and eosin stain)

Immunohistochemistry

The spindle cells found in HIP are vimentin positive, and at least part of them express alpha-smooth muscle actin (alpha-SMA), classifying these cells as myofibroblasts. In contrast to smooth muscle cells found in the muscle layer cells (Montani et al. 2010) and in leiomyomas and leiomyosarcomas of the gastrointestinal tract, the spindle cells of inflammatory pseudotumors are negative for the smooth muscle-specific cytoskeletal protein, smoothelin (Coco et al. 2009). Myofibroblasts do not seem

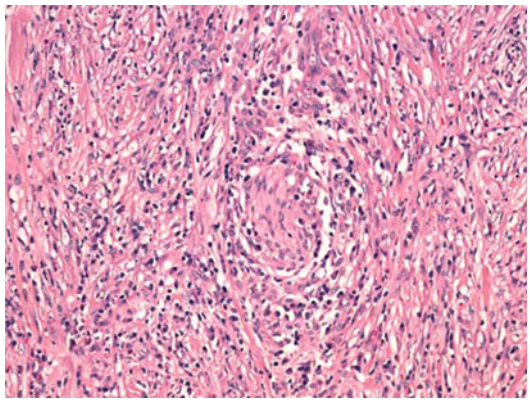

Fig. 6 Inflammatory pseudotumor of the liver. Lymphocytes, plasma cells, and macrophages infiltrate a tissue with abundant fibroblastoid cells and are present around a small nerve (center; hematoxylin and eosin stain)

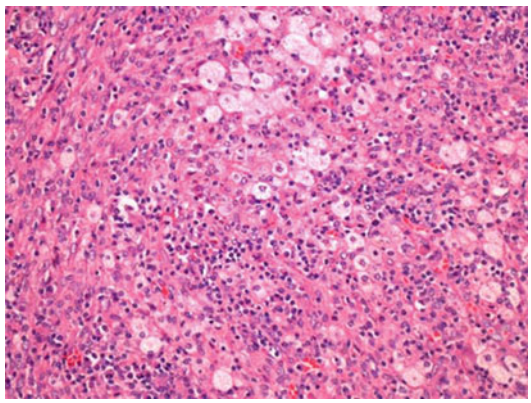

Fig. 7 Inflammatory pseudotumor of the liver. This lesion contains numerous lipid-rich, foamy macrophages (pseudoxanthoma cells; hematoxylin and eosin stain)

to express smoothelin (Nam et al. 2012). Immunohistochemically, the lymphocyte population is typically a mixture of T and B lymphocytes (Kim et al. 1999), even though T (in particular CD8-reactive) cells may predominate, also in portal tracts of the liver adjacent to the tumor, and the plasma cells are polytypic (Uccheddu et al. 1995; Sakai et al. 2001), showing a mixture of IgA-, IgM-, and IgG-producing cells and the expression of both lambda and kappa light chains (Tsao et al. 1990). However, in one reported case, most of the lymphocytes and plasma cells produced IgA and predominantly lambda light chains (Grouls 1987). Macrophages in the infiltrate are frequently activated and CD68-positive.

Attempts for a Histologic Classification of HIP

Referring to the predominant histologic patterns, attempts have been undertaken to classify the lesions in more detail. Someren categorized HIP into three morphotypes, i.e., hyalinized sclerosing, xanthogranuloma, and plasma-cell granuloma types (Someren 1978). As in other lesions with heterogeneity owing to an internal gradient of cellular responses, such a classification is prone to considerable sampling variation and therefore of doubtful significance. Based on 74 previously reported cases and an own pediatric patient, Lee and DuBois have recently proposed a novel classification (Lee and DuBois 2001). In contrast to histopathologic categories, they put emphasis on the clinical/radiologic presentation, i.e., solitary or multiple lesions. Their type 1 or solitary HIPs were typically large and centrally located lesions, exhibited a mean diameter of 7 cm, showed a low-density central core on CT representing necrosis, have a hypovascular or even avascular angiographic appearance, and were a predominant variant of HIP in children (16/18). The more central location of type 1 lesions may result in bile duct stenosis and portal venous and obliterative endophlebitis, as discussed below. Owing to these complications, type 1 according to Lee and DuBois may require surgery more frequently than other manifestations of HIP. Type 2 or multiple HIPs are typically smaller lesions (2–6 cm diameter) in a bilobar distribution, occur in adults and children, and are less likely to involve bile ducts and/or portal vein branches (Lee and DuBois 2001). In CT, the nodules are solid and exhibit a low but uniform density, similar to metastases, so that exclusion of malignancy is a major concern in case of type 2 lesions.

Differential Diagnosis

The differential diagnosis of HIP mainly includes chronic liver abscess, malignancy (sarcoma; malignant fibrous histiocytoma; lymphoma; desmoplastic carcinoma), benign mesenchymal tumors, and (sclerosed) vascular tumors.

Biology of Disease

In situations without biliary or vascular involvement and complications, or large lesions, the clinical course is usually benign and self-limiting; it sometimes even undergoes spontaneous regression (Gollapudi et al. 1992; Zamir et al. 1998; Young et al. 1998; Soudack et al. 2000; Nakama et al. 2000; Levy et al. 2001), but in patients with large and non-resolving lesions, hepatectomy is now advocated to be the treatment of choice in comparison with conservative therapy (Mangiante et al. 1997), even though selected patients may profit from conservative treatment, including antibiotics (Jais et al. 1995; Casassus-Builhe et al. 2002). The question as to whether HIP may undergo malignant evolution is not settled so far. A patient with HIP has been described where a subsequent evolution into malignant non-Hodgkin's lymphoma occurred (Pecorella et al. 1999).

Etiology and Pathogenesis

HIP is often associated with diverse conditions that may deliver clues as to pathogenic pathways involved (Lyons et al. 1993). In regard to pathogenic considerations, the apparently reproducible association of HIP and chronic cholangitis (Gough and Chakrabarti 1993; Nakanuma et al. 1994) or with recurrent pyogenic cholangitis (Yoon et al. 1999) merits particular attention. Yoon and coworkers investigated on 13 inflammatory hepatic pseudotumors evolving in 10 patients. All these patients had CT features of recurrent pyogenic cholangitis, such as hepatolithiasis, intrahepatic bile duct stricture and dilatation, common bile duct calculi, pneumobilia, or signs of atrophy/hypertrophy complex of the liver. The presence of chronically infected extrahepatic and/or intrahepatic bile ducts may, therefore, represent a risk factor for the development of IPTL, and a marked local host response to bacterial infection may in fact be the driving force for the establishment of a tumor-like, exuberant repair reaction, also involving strong immune reactions of both the cellular and humoral type. A causal relationship to local bacterial infection is further indicated by the observation of a spatial connection between HIP and adjacent abscesses (Jimenez-Mejias et al. 1989). Furthermore, situations with dilatation of bile ducts and frequently complicated by bacterial infection may later be accompanied by HIP (Terada et al. 1992; Kuhara et al. 1994). A possible significance of more remote visceral infections is indicated by the finding of HIP occurring together with diverticulitis (Coleman and Rees 1999) and with chronic abdominal abscesses followed by sepsis (White et al. 1997). In one case, fine-needle aspirate yielded a growth of *Klebsiella* organisms (Kafeel and Telesinghe 1997) and, in another, *Escherichia* and *Actinomyces* species (Schmid et al. 1996). In a patient with multiple HIP, *Enterococcus durans* bacteremia was detected (Jover Diaz et al. 2009). A further argument for the pathogenetic significance of infections is offered by the observation that HIP can regress under antibiotic therapy (Casassus-Builhe et al. 2002). HIP was also found in association with primary hepatic actinomycosis (Tamsel et al. 2004) and amebic infections (Alfieri et al. 2008) and with *Mycobacterium* tuberculosis infection in an immunocompetent child (Manolaki et al. 2009). The observation of HIP in association with HIV infection (Tai et al. 1998) might, if not a fortuitous coincidence, suggest a role of deranged immune responses to the more frequent bacterial infections in this disorder.

That immune or autoimmune mechanisms may play a role in the pathogenesis of HIP is suggested by the observation of such lesions in patients with primary biliary cirrhosis (Rai et al. 2003; Koide et al. 2006), primary sclerosing cholangitis (Toda et al. 2000), and after liver transplantation (Lykavieris et al. 2000); in patients with Crohn's disease (Papachristou et al. 2004; Mouelhi et al. 2009; Renzing et al. 2011) and rheumatoid arthritis (Diaz-Torné et al. 2007); and in the setting of Sjögren's syndrome (Hosokawa et al. 1998). HIP has been observed as the first manifestation of Crohn's disease (Amankonah et al. 2001), and it was speculated that the lesion may be related to underlying inflammatory bowel disease. Furthermore, inflammatory reactions

located elsewhere may be modified by the presence of HIP; e.g., both fever and recurrent arthritis related to gout completely resolved after surgery of HIP (Takahashi et al. 2001). HIP has been observed to complicate severe congenital neutropenia (Kostmann's disease; Hsiao et al. 1999; Schneider et al. 2003) and to occur in a hereditary disorder associated with the development of hepatic abscesses, Papillon-Lefevre syndrome (Czauderna et al. 1999).

Some pseudotumors seem to develop in association with collapsed liver cysts or form a residuum of biliary cystadenoma (Hoosein et al. 2007). HIP has been observed in association with gallstones (Al-Jabri et al. 2010), including hepatolithiasis (Ueda et al. 2009; Terada 2010); biliary atresia (Wang et al. 2002); intra-abdominal foreign bodies, e.g., intrahepatic wooden toothpicks or migrated fishbone (Del Fabbro et al. 2004; Perera et al. 2007); abdominal malignancy (Nishimura et al. 2005), GISTs (Lo et al. 2004); and leukemias (Isobe et al. 1991; Tajima et al. 1998). It has developed as a possible complication of percutaneous radiofrequency ablation of hepatocellular carcinoma (Lee et al. 2009). HIP was found during pregnancy (Maze et al. 1999).

Inflammatory Myofibroblastic Tumors of the Liver and Related Lesions

Introduction

Inflammatory myofibroblastic tumor (IMT) is a distinctive mesenchymal tumor that has emerged from within the heterogeneous group of inflammatory pseudotumors as an entity characterized by neoplastic features, a proliferation of myxoid spindle cells, an inflammatory infiltrate, and a rearrangement of anaplastic lymphoma kinase (ALK) in at least 50 % of the cases (reviews: Dehner 2004; Gleason and Hornick 2008). ALK rearrangement of the respective locus on chromosome 2p23 causes aberrant ALK expression in the neoplastic cells (Griffin et al. 1999). The term, IMT, implies that the neoplastic cell lineage characterizing IMT is a myofibroblast or a myofibroblast-like cell. However, alternative views regarding the cell of origin have been formulated, e.g., a proliferation of fibroblastic reticulum cells, a subtype of cell of the accessory immune system (Nonaka et al. 2005).

IMTs occur primarily during the first two decades of life (Coffin et al. 2007) and chiefly arise in the lung, retroperitoneum, and abdominopelvic region (Coffin et al. 1995). IMTs also occur in the pediatric age group (Mergan et al. 2005; Ernst et al. 2011). Less common localizations comprise the central nervous system, salivary glands, thyroid, larynx, breast, spleen, urinary bladder, and skin.

Inflammatory Myofibroblastic Tumors of the Hepatobiliary Tract

IMT of the liver is a rather common intra-abdominal manifestation of IMTs (Beauchamp et al. 2011). In a fine-needle aspiration cytologic study of 20 cases, nine were localized to the liver (Stoll and Li 2011). IMT of the liver was also found in children (Mergan et al. 2005). IMT very rarely develops in the bile duct system. The lesions developing in the distal part of the common bile duct usually make part of the syndrome IgG4-associated sclerosing disease (Yamamoto et al. 2009). IMTs not associated with IgG4 disease have very rarely been reported (Stamatakis et al. 1979; Bolla et al. 1988; Ikeda et al. 1990; Imafuku et al. 1990; Fukushima et al. 1997; Saint-Paul et al. 1999; Worley et al. 2001; Ashcroft et al. 2009) and also develop in the ampullary/periampullary region (Leese et al. 1986; Price et al. 1993). IMTs can develop in the hilar region, producing extending structures and mimicking a Klatskin tumor (Worley et al. 2001). Bile duct IMTs are usually lesions with a benign behavior, but aggressive forms have also been reported. In a 63-year-old female patient with aggressive hilar IMT, pulmonary metastasis developed (Kim et al. 2011).

Pathology

The typical feature is a growth of slightly eosinophilic spindle cells resembling fibroblasts, with bland-looking nuclei and absent mitoses or a low mitotic count. The spindle cells, which are immunohistochemically myofibroblasts, may be embedded in a myxoid or hypocellular fibrosclerotic matrix (Fig. 8). In a minority of cases, a round cell transformation of IMT was observed (Chen and Lee 2008). Cellular atypia is more frequent in aggressive variants of IMT. Cellular atypia was found in 69 % of tumors that recurred and in all cases showing malignant transformation (Hussong et al. 1999).

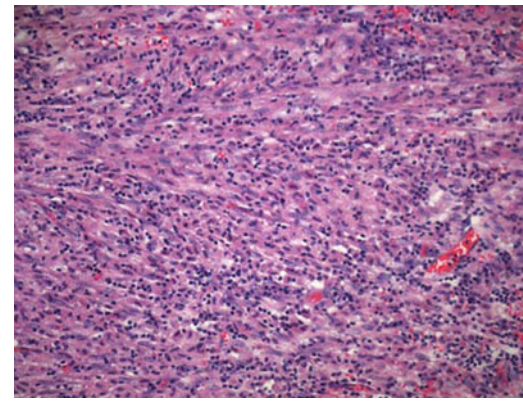

Fig. 8 Myofibroblastic pseudotumor of the liver. A dense collection of myofibroblasts and other spindle cells is infiltrated with lymphocytes (hematoxylin and eosin stain)

Immunohistochemistry

Apart from aberrant ALK expression (see below), hepatic IMT cells have been shown to be positive for alpha-SMA (Qiu et al. 2008) and actin (100 %; Stoll and Li 2011), supporting the myofibroblastic character of these cells (Fig. 9). Aggressive IMT may show nuclear p53 expression (Hussong et al. 1999).

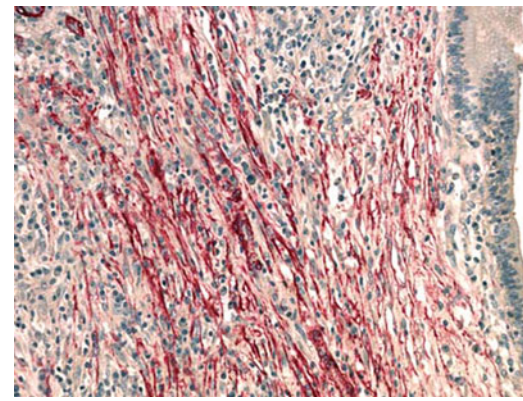

Fig. 9 Myofibroblastic pseudotumor of a bile duct. The inflamed tissue contains numerous spindle cells with reactivity for smooth muscle actin. The inflammatory infiltrate contains numerous plasma cells (alpha-SMA immunostain)

Biology of Disease

IMTs usually show a rather indolent course, but may exhibit invasive growth and spread. Currently, IMT is considered to be an intermediate-grade neoplasm that can recur in up to 25 % of cases (Hussong et al. 1999). However, in the subset of abdominal and pelvic IMTs, the recurrence rate was 85 % in one investigation (Coffin et al. 2007), suggesting an important role for the site of origin. Rarely, IMT can undergo malignant transformation, with locoregional and/or distant metastases (Hussong et al. 1999; Ernst et al. 2011). In a study of 24 cases, 8 % underwent malignant transformation (Hussong et al. 1999). Low-grade myofibroblastic sarcoma, which may be confounded with IMT, is currently not regarded as a member of the family of ALK-positive tumors (Qiu et al. 2008).

ALK Rearrangements in Hepatic IMT

IMTs primary to the liver may show ALK gene rearrangements with variable fusion partners. An unusual rearrangement is that between the Ran-binding protein 2 and ALK, associated with nuclear membrane expression of ALK (Chen and Lee 2008). Aberrant anaplastic lymphoma kinase (ALK) expression caused by ALK gene rearrangements is a frequent alteration in IMTs. ALK plays a significant role in the regulation of cell proliferation in several cell lineages and is the

partner of several fusion proteins in distinct chromosomal translocations (reviews: Ladanyi 2000; Palmer et al. 2009; Schonherr et al. 2012). ALK is a receptor tyrosine kinase that is highly related to leukocyte tyrosine kinase (Morris et al. 1997). ALK was expressed in 47.1 % of cases analyzed by fine-needle aspiration cytology (Stoll and Li 2011). In a study of 73 IMTs, immunohistochemical ALK positivity was detected in 60 % of cases. In contrast, all cases of nodular fasciitis, desmoid fibromatosis, and gastrointestinal stromal tumors were ALK negative (Cook et al. 2001). Among sixteen children (age range 1–15 years), aberrant ALK expression was detected in only 18.8 % of patients (Mergan et al. 2005). In one study, ALK-negative IMTs occurred in older patients and had a greater nuclear pleomorphism, atypia, and atypical mitoses, and all metastatic IMTs were ALK-negative, suggesting that ALK negativity is an indicator of an aggressive course and metastasizing disease (Coffin et al. 2007). The significance of ALK in the biology of IMT is highlighted by the observation that the ALK inhibitor, crizotinib, has led to a sustained partial response of IMT (Butrynski et al. 2010). Another myofibroblastic neoplasm that expresses ALK with a nuclear membrane or perinuclear expression pattern is epithelioid inflammatory myofibroblastic sarcoma, a predominantly intra-abdominal variant of IMT (Mariño-Enriquez et al. 2011).

Molecular characterizations have identified ALK fusions involving tropomyosins 3 and 4 (TPM-3 and TPM-4; Lawrence et al. 2000), clathrin heavy chain (CLTC; Bridge et al. 2001), cysteinyl-tRNA synthase (CARS), and Ran-binding protein 2 genes as fusion partners. IMTs with Ran-binding protein 2 (RANBP2/Nup358) fused with ALK are rare. The N-terminal 867 residues of RANBP2 are fused to the cytoplasmic segment of ALK in the RANBP2-ALK chimeric protein, and this chimerization causes ALK to be localized to the nuclear membrane (Ma et al. 2003). RANBP2 is a large (358 kDa) nucleopore protein (nucleoporin) localized at the cytoplasmic side of the nuclear pore complex. It is mutated in familial or recurrent acute necrotizing encephalopathy (Neilson et al. 2010; Loh and Appleton 2010), and the major component of the cytoplasmic filaments of the nuclear pore complex has a critical function in capturing recycling RanGTP-importin-beta complexes at cytoplasmic fibrils to allow for adequate classical nuclear localization signal-mediated cargo import (Hamada et al. 2011). The mediation of cargo processes is also related to the association of RANBP2, through its kinesin-binding domain, with kinesin-1, and RANBP2 is a positive allosteric activator of the kin2718esin-1 system (Cho et al. 2009). The protein is involved in the distribution of SR splicing factors enriched in nuclear speckles or interchromatin granule clusters and plays a role in the speckled distribution of phosphorylated pre-mRNA processing factors (Saitoh et al. 2012). RanBP2 contains a domain that catalyzes E3 ligase activity and forms a stable complex with SUMO-modified RanGAP1 and UBC9 at the nuclear pore complex (Gareau et al. 2012).

Epstein-Barr Virus-Positive Inflammatory Pseudotumor/ Inflammatory Pseudotumor-Like Follicular Dendritic Cell Tumor

Introduction

Lesions histologically resembling inflammatory pseudotumors have been found to be infected with Epstein-Barr virus (EBV), which is thought to play a role in the pathogenesis of these lesions. 18 inflammatory pseudotumor specimens (from lymph nodes, spleen, and liver) from 17 patients were studied by in situ hybridization for EBV RNA, and EBV virus RNA expression was detected in 41.2 % of cases, including one from the liver. Two morphologically different EBV-positive cell types, spindled and round cells, were evident, whereby the EBV-positive spindled cells were present exclusively in the extranodal lesions. Part of the EBV-positive spindled cells co-expressed smooth muscle actin, while others had immunohistochemical features of follicular dendritic cells (Arber et al. 1995). The presence of EBV virus in inflammatory

pseudotumors is an interesting finding regarding the pathogenesis of these lesions and has also been found in rare smooth muscle tumors in immunosuppressed individuals (reviews: Arber et al. 1998; Deyrup 2008). Since 1995, several examples of this distinct lesion have been reported for several organs, including the spleen (Yamaguchi et al. 2000; Kutok et al. 2001; Neuhauser et al. 2001; Lewis et al. 2003; Horigichi et al. 2004; Oz Puyan et al. 2004; Kiryu et al. 2009; Rosenbaum et al. 2009). In splenic lesions, two growth patterns have been noted, viz., a cellular spindle cell pattern and a hypocellular fibrous pattern, and immunohistochemistry revealed the myofibroblastic nature of the spindle cells (Neuhauser et al. 2001). Few examples in intracranial EBV-positive inflammatory pseudotumors are reported, one originating from the trigeminal nerve (Jung et al. 2006) and another showing an intraventricular site (Nishioka et al. 2009). A second splenic lesion that has been shown to express EBV RNA is sclerosing angiomatoid nodular transformation (SANT), a lesion which is thought by some authors to represent a final stage of splenic inflammatory pseudotumor (Weinreb et al. 2007, Kashiwagi et al. 2008).

Recently, the term inflammatory pseudotumor-like follicular dendritic cell tumor (IPL-FDCT) has been proposed to denote such lesions (Cheuk et al. 2001). The authors characterized a distinctive variant of FDCT morphologically mimicking inflammatory pseudotumor based on 11 patients (10 women, 1 man). All tumors occurred in intra-abdominal sites, i.e., liver (seven cases), spleen, and peripancreatic region, and were biologically classified as a low-grade malignant neoplasm. Of the nine patients with follow-up data, six were alive and well, one developed recurrence at 9 years, and two had repeated recurrences over many years. The term has recently been employed to denote splenic lesions formerly termed inflammatory pseudotumor (Horiguchi et al. 2004; Gong et al. 2008; Kiryu et al. 2009). Another term to denote such lesions was follicular dendritic reticulum cell tumor mimicking inflammatory pseudotumor, e.g., of the spleen (Brittig et al. 2004).

Hepatobiliary EBV-Positive Inflammatory Pseudotumors

In about the same frequency as the spleen, EBV-associated inflammatory tumors/IPL-FDCTs develop in the liver. In contrast to FDCTs in extrahepatic and extrasplenic areas, hepatic FDCTs have a strong association with EBV and a stronger inflammatory component and are more prevalent in females. FDCT not associated with EBV also occurs in the liver and is discussed in a separate chapter. The first EBV-positive hepatic inflammatory pseudotumor was described in 1995 (Arber et al. 1995). In the following year, an EBV-related clonal proliferation of follicular dendritic cells was described as follicular dendritic cell tumor of the liver (Shek et al. 1996). This tumor was initially reported as a hepatic inflammatory pseudotumor, but the lesion recurred as two separate tumor masses 30 months after complete resection. Histologically, the tumor contained bland-looking spindle cells amidst a background of lymphocytes and highly pleomorphic tumor cells reactive for CD21, CD35, R4/23, and Ki-M4. Both cell populations were positive for EBV-encoded RNA by in situ hybridization. The tumor cells expressed EBV latent membrane protein (LMP1), but not EBV-encoded nuclear antigen 2/EBNA2 (Shek et al. 1996). In this case, IPL-FDCT may have developed as a neoplastic lineage from an EBV-positive inflammatory pseudotumor. Again in 1996, Selves and coworkers described an "inflammatory pseudotumor" of the liver with a spindle cell component that was shown to be derived from follicular dendritic reticulum cells (FDRC). This cell population expressed the EBV receptor CD21 and contained clonal EBV genomes and EBV RNA transcripts (EBER) and expressed EBV latent membrane protein (Selves et al. 1996). In one young male patient with a large inflammatory pseudotumor of the liver (75 mm diameter) clinically associated with high fever and malaise, only EBV LMP1 was demonstrated within the lesion (Fritzsch et al. 2004). In a 30-year-old female patient, the 6 cm-sized hepatic tumor consisted of an admixture of spindle cells, lymphocytes, plasma cells, and macrophages. The spindle cells

were arranged in a wavy pattern, were immunoreactive for CD21 and CD68, and expressed EBV-encoded nuclear RNAs, leading to the diagnosis of FDCT (Bai et al. 2006). Further reported cases from the liver were identified as IPL-FDCT (Cheuk et al. 2001).

Pathology

Macroscopically, the neoplasms have been described as solitary and fleshy lesions with areas of hemorrhage and necrosis. Histologically, spindle cells are the predominant lineage, embedded in a tissue containing an inflammatory infiltrate consisting of lymphocytes, plasma cells, and macrophages. The proportion of spindle cells to lymphoid and plasma cells is variable in different areas, a typical finding in inflammatory pseudotumors. Parts of the spindle cells are bland looking, with a slightly eosinophilic cytoplasm and inconspicuous nuclei. These cells may be confounded with fibroblasts. A significant number of spindle cells show two nuclei, suggesting features of follicular dendritic cells. Sometimes, these large binucleated cells resemble Reed-Sternberg cells. Other spindle cells are atypical, with vesicular nuclei and distinct nucleoli. The spindle cells may be arranged in the form of fascicles, and a storiform pattern may be found. Atypical large ovoid cells are also noted. Immunohistochemically, the neoplastic cells are reactive for follicular dendritic cell markers, i.e., CD21 (C3d receptor), CD35 (C3b receptor), Ki-M4P, CD23, and CNA42, but only a fraction of cells are positive for DRC1 (Selves et al. 1996; Cheuk et al. 2001). The binucleated cells with large vesicular nuclei are consistently negative for CD15 and CD30. Normal-looking and atypical spindle cells express EBV markers, including immunoreactivity for LMP1 and EBNA2, and EBV-encoded RNA is detectable with in situ hybridization. Cells bearing EBV signals were CD21-positive by double immunostaining (Selves et al. 1996). Expression of EBV markers is also seen in those lesions that do not show clear-cut follicular dendritic cell features. Oval cells were negative for CD15, CD20, CD30, ALK protein, Bcl-2, S-100 protein, EMA, CD34, and HHV-8 (Rosenbaum et al. 2009). A small population of binucleated cells show an increased proliferative activity in Ki-67 immunostains. The lymphocytes are predominantly T cells, more CD8+ than CD4+ cells, while B cells are relatively scarce. The plasma-cell population is polytypic. Numerous CD68+ macrophages are commonly present (Selves et al. 1996).

Pathogenic Pathways

There is evidence that IPL-FDCT can develop from EBV-infected cells located in a primary inflammatory pseudotumor without clear frank signs of neoplasia (Shek et al. 1996).

Calcifying Fibrous Tumor (Calcifying Fibrous Pseudotumor) of the Hepatobiliary Tract

Introduction

Calcifying fibrous tumor/pseudotumor (CFT) is a unique, benign tumor or pseudotumor that was described as a childhood lesion in 1988 (Rosenthal and Abdul-Karim 1988) and later again in 1993 by Fetsch and coworkers, who first used the term calcifying fibrous pseudotumor (Fetsch et al. 1993). CFT most commonly occurs in soft tissues of the extremities and in the pleura, with a predilection for young individuals. According to the WHO classification, the lesion is now termed calcifying fibrous tumor. It is histologically characterized by the presence of hyalinized collagen with dystrophic calcifications (sometimes with psammoma bodies) and a mixed lymphocytic and plasmacytic infiltrate, with some contribution of IgG4-positive plasma cells (Kuo et al. 2009; Agaimy et al. 2010). About 10 % of the lesions showed recurrence (Fetsch et al. 1993), and about 10 % of the patients had multiple lesions (Pinkard et al. 1996). It has been discussed whether CFT might represent a late stage of

inflammatory myofibroblastic tumor (Van Dorpe et al. 1999; Sigel et al. 2001). In addition to soft tissues, CFT can also occur in visceral organs and tissues, including lung, heart, esophagus, stomach, omentum, mesentery, small intestine, rectum, fallopian tube, and adrenal gland. CFT also occurs in the gallbladder (Mourra et al. 2004). Calcifying fibrous tumor has been observed as multiple lesions of the peritoneum (Kocova et al. 1997; Farah et al. 2007), in one situation with familial occurrence (Chen 2003). Interestingly, abdominal CFT may be associated with sclerosing angiomatoid nodular transformation of the spleen (SANT). In a study of ten cases of SANT, five cases were associated with disseminated abdominal CFT, and IgG4-positive plasma cells were found in all SANT and CTF (Kuo et al. 2009), suggesting a possible relationship with sclerosing IgG4-related systemic disease.

Whether CFT is a pseudotumor or rather a neoplastic lesion is not yet settled. Based on 15 cases, it was proposed that CFT might represent a benign mesenchymal neoplasm with a low risk of recurrence (Nascimento et al. 2002).

Calcifying Fibrous Tumor of the Liver

CFT has rarely been observed in the liver substance (Jo et al. 2011; Nobili et al. 2011). Liver involvement has been observed in a patient with multiple nodules in several organs (Azam et al. 2014). On CT images, multiple laminated and amorphous calcifications are seen. Macroscopically, the masses are well circumscribed and exhibit a hard consistence due to multiple calcifications. The nodules reveal a fibrous texture and gray-white color and can reach a diameter exceeding 10 cm. The tumor may show a hardness that does not allow insertion of a biopsy needle (Jo et al. 2011). Histologically, the tumors are usually hypocellular and mainly consist of a vascularized collagenous mass with embedded spindle cells. The multiple calcifications are either amorphous or present as psammoma-like bodies. Lymphocytes and plasma cells are predominantly found in peripheral parts of the mass and in the peritumoral tissue.

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