Surgicopathological classification of hepatic space-occupying lesions: A single-center experience with literature review

Wen-Ming Cong, Hui Dong, Lu Tan, Xu-Xu Sun, Meng-Chao Wu

Wen-Ming Cong, Hui Dong, Lu Tan, Xu-Xu Sun, Department of Pathology, Eastern Hepatobiliary Surgery Hospital, Second Military Medical University, Shanghai 200438, China
Meng-Chao Wu, Department of Surgery, Eastern Hepatobiliary Surgery Hospital, Second Military Medical University, 225 Changhai Road, Shanghai 200438, China

Author contributions: Cong WM and Wu MC designed the study; all authors generated the ideas and contributed to the writing of this manuscript.

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Correspondence to: Wen-Ming Cong, MD, PhD, Professor and Director, Department of Pathology, Eastern Hepatobiliary Surgery Hospital, Second Military Medical University, 225 Changhai Road, Shanghai 200438, China. wmcong@gmail.com
Telephone: +86-21-81875191 Fax: +86-21-81875191
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Abstract
Accompanying rapid developments in hepatic surgery, the number of surgeries and identifications of histological types of primary hepatic space-occupying lesions (PHSOLs) have increased dramatically. This has led to many changes in the surgicopathological spectrum of PHSOLs, and has contributed to a theoretical basis for modern hepatic surgery and oncological pathology. Between 1982 and 2009 at the Eastern Hepatobiliary Surgery Hospital (EHBH) in Shanghai, 31 901 patients underwent surgery and were diagnosed as having a PHSOL. In this paper, we present an analysis of the PHSOL cases at the EHBH for this time period, along with results from a systematic literature review. We describe a surgicopathological spectrum comprising more than 100 types of PHSOLs that can be stratified into three types: tumor-like, benign, and malignant. We also stratified the PHSOLs into six subtypes derived from hepatocytes; cholangiocytes; vascular, lymphoid and hematopoietic tissues; muscular, fibrous and adipose tissues; neural and neuroendocrine tissues; and miscellaneous tissues. The present study provides a new classification system that can be used as a current reference for clinicians and pathologists to make correct diagnoses and differential diagnoses among various PHSOLs.

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Key words: Liver tumors; Tumor-like lesions; Pathology; Immunohistochemistry; Classification

Peer reviewers: Kuniya Tanaka, MD, PhD, Professor, Department of Gastroenterological Surgery, Yokohama City University, 3-9 Fukuura, Kanazawaku, Yokohama, Ktrj 112, Japan; Toshifumi Wakai, MD, PhD, Division of Digestive and General Surgery, Niigata University Graduate School of Medical and Dental Sciences, 1-757 Asahimachi-dori, Chu-o-ku, Niigata City 951-8510, Japan

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INTRODUCTION
Liver neoplasms are one of the most common tumors worldwide, especially in China and other developing countries. Rapid developments in liver surgery and liver pathology have led to many new types of primary hepatic space-occupying lesions (PHSOLs) being surgically resected and pathologically diagnosed, which has greatly increased the surgicopathological spectrum of PHSOLs. Indeed, insights into tumor pathological characteristics have illuminated the need for an improved practical guide for oncological clinicians and pathologists to make correct diagnoses and differential diagnoses among PH-
SOLs\textsuperscript{[3]}. However, to the best of our knowledge, there is no report in the English literature that thoroughly assesses the whole spectrum of PHSOLs.

During the period from January 1982 to December 2009, 31901 surgically resected PHSOLs were deposited in the archives of the Department of Pathology, Eastern Hepatobiliary Surgery Hospital (EHBH) in Shanghai. In this paper, we present an analysis of the above 31901 PHSOL cases, along with results from a systematic literature review. To the best of our knowledge, this is the largest series of PHSOLs presented from a single center. Based on the EHBH archival data and literature reviews using MEDLINE and PUBMED, more than 100 types of PHSOLs have been described. In this article, we suggest a surgicopathological classification of PHSOLs comprising three types: tumor-like PHSOLs, benign PHSOLs, and malignant PHSOLs. We also stratified the PHSOLs into six subtypes: lesions derived from hepatocytes; cholangiocytes; vascular, lymphoid and hemopoietic tissues; muscular, fibrous and adipose tissues; neural and neuroendocrine tissues; and miscellaneous tissues.

### TUMOR-LIKE PHSOLs

Tumor-like PHSOLs are usually a type of space-occupying lesion within the hepatic parenchyma or intrahepatic bile ducts, but without a truly neoplastic nature. At least 31 kinds of tumor-like PHSOLs have been reported, as summarized in Table 1\textsuperscript{[2-23]}. In the EHBH series, tumor-like PHSOLs accounted for 4.3% ($n = 1370$) of the 31901 cases. Of the tumor-like PHSOLs, focal nodular hyperplasia (FNH) accounted for 51.5% ($n = 705$), solitary necrotic nodules accounted for 19.6% ($n = 269$), and hepatic inflammatory pseudotumors (HIP) accounted for 12.0% ($n = 165$). These are the three most common tumor-like PHSOLs.

In the latest edition of the World Health Organization (WHO) classification report (2010 Edition), FNH and HIP were grouped as benign liver tumors\textsuperscript{[30]}. However, most scholars, and the present authors, prefer to regard FNH and HIP as a kind of non-neoplastic lesion or a tumor-like lesion\textsuperscript{[27]}. FNH is a regenerative hepatocellular nodule that is frequently related to factors that stimulate the hyperperfusion of either the artery or the portal vein. Clonal analysis using the human androgen receptor locus test demonstrated the reactive polyclonal nature in 50%-100% of the FNH cases. Genetic analysis of FNH failed to identify somatic gene mutations that occurred in hepatocellular adenoma (HCA)\textsuperscript{[28]}. Currently, most FNH are considered as polyclonal, and there was neither recurrence nor substantiated malignant transformation in all 705 FNH cases included in the EHBH series after surgery, even though FNH may occasionally coexist with hepatocellular carcinoma (HCC)\textsuperscript{[29]}. Either clinically or pathologically, FNH should be distinguished from other hepatocellular nodules, such as HCA and highly differentiated HCC. Hepatocyte paraffin 1 (Hep Par 1) and polyclonal carcinoembryonic antigen (CEA) are special hepatocellular markers, which cannot, however, differentiate between benign and malignant nature; therefore, we prefer to use CD34 immunostaining to sensitively and specifically outline microvasculatures to differentiate hepatocellular nodules\textsuperscript{[2,6,13,14]}. FNH usually presents in a focal distribution pattern of microvasculatures around fibrous scars (Figure 1A and B), whereas HCA shows a chaotic distribution pattern, usually with thin-walled vascular staining (Figure 1C and D). HCC presents in a diffuse distribution pattern occupying a greater proportion of the lesion area (Figure 1E and F). Although glypican-3 (GPC-3) has recently been reported to be overexpressed in HCC, the lack of GPC-3 immunostaining could not exclude the diagnosis in at least 25%-30% of HCC\textsuperscript{[15]}.

### BENIGN PHSOLs

At least 30 types of benign PHSOLs have been reported, as summarized in Table 2\textsuperscript{[2,23-36]}. In the EHBH series, benign tumors accounted for 12.1% of the cases ($n = 3847$), among which hepatic cavernous hemangioma ($n = 3191$, 82.9%), hepatic angiomylipoma (HAML, $n = 153$, 4.0%), and HCA ($n = 148$, 3.8%) were the most frequent types in this group.

| Table 1 Histological classification of tumor-like primary hepatic space-occupying lesions |
|-----------------------------------------|-------------------|
| **Hepatocellular lesions**              |                    |
| Focal nodular hyperplasia\textsuperscript{[2]} |                    |
| Nodular regenerative hyperplasia\textsuperscript{[2]} |                    |
| Partial nodular transformation\textsuperscript{[3]} |                    |
| Adenomatoid hyperplasia (dysplastic nodules)\textsuperscript{[4]} |                    |
| Compensatory lobar or segmental hyperplasia\textsuperscript{[4]} |                    |
| Focal fatty change\textsuperscript{[4]} |                    |
| Accessory lobe\textsuperscript{[4]} |                    |
| Bile duct lesions                       |                    |
| Biliary microhamartoma (Von Meyenburg complex)\textsuperscript{[2]} |                    |
| Cyst and polycystic liver\textsuperscript{[4]} |                    |
| Ciliated foregut cyst\textsuperscript{[2]} |                    |
| Epidermoid cyst\textsuperscript{[4]} |                    |
| Endometrial cyst\textsuperscript{[4]} |                    |
| Intrahepatic peribiliary gland cyst\textsuperscript{[4]} |                    |
| Mesothelial cyst\textsuperscript{[4]} |                    |
| Cystic echinococcosis\textsuperscript{[11]} |                    |
| Biloma\textsuperscript{[12]} |                    |
| Miscellaneous lesions                   |                    |
| Mesenchymal hamartoma\textsuperscript{[4]} |                    |
| Inflammatory pseudotumor\textsuperscript{[4]} |                    |
| Pseudolymphoma\textsuperscript{[4]} |                    |
| Solitary necrotic nodule\textsuperscript{[4]} |                    |
| Peliosis hepatic\textsuperscript{[13]} |                    |
| Hereditary hemorrhagic telangiectasia\textsuperscript{[16]} |                    |
| Sarcoidosis\textsuperscript{[17]} |                    |
| Nodular extramedullary hematopoiesis\textsuperscript{[18]} |                    |
| Abscess\textsuperscript{[19]} |                    |
| Tuberculosis\textsuperscript{[20]} |                    |
| Botryomyositis\textsuperscript{[21]} |                    |
| Malacoplasia\textsuperscript{[22]} |                    |
| Ectopic tissue\textsuperscript{[20]} and adrenal rest tumor\textsuperscript{[24]} |                    |
| Pseudolipoma\textsuperscript{[24]} |                    |
| Granulomas\textsuperscript{[24]} |                    |
In Western countries, patients with HCA or hepatic adenomatosis are mostly estrogen/androgen dependent types, with a female gender bias (> 90%). Among them, 78% have a history of taking contraceptive drugs, and 4% to 4.7% may develop HCC[4,5]. It has also been reported that 4% to 17.6% of HCA patients with HCA may have differences in etiology, genetic, and HCA-related HCC risk, compared to Western patients. The above research suggests that the detection of molecular biological or immunohistochemical markers before or after surgery is essential for providing an active radical radiotherapy cure. In addition, more attention should be paid to the careful follow-up of patients with a high potential for transformation of β-catenin activated HCA to prevent HCA transformation or recurrence[26]. Thus, the treatment roadmap based on HCA molecular characteristics has also been described[59].

In 1993, we reported the first case of primary HAML in China. During the last 3 years of the study period, 85 cases of primary HAML and 66 cases of HCA were surgically resected at the EBHB. HAML is generally considered as a miscellaneous benign tumor; however, we find that some cases of HAML can show doubtful growth patterns, such as multi-focus, boundary infiltration along the sinusoids (Figure 2A and B), or even intravascular aggregation of conspicuous HMB45 positive cells (Figure 2C and D), which are similar to malignant behaviors. However, none of the 153 cases of HAML in the EBHB series showed evidence of malignant transformation or postoperative recurrence up to the time of the termination of this study.

The presence of malignant HAML or malignant transformation of HAML[60,61] indicates that surgical excision should be considered as a preferred therapeutic, and a long-term follow-up after liver surgery is needed.

**MALIGNANT PHSOLS**

At least 41 malignant PHSOLs were reported, as summarized in Table 3[6,26]. In the EBHB series, malignant PHSOLs accounted for 83.6% (n = 26,684) of the cases, among which, HCC (n = 24,075, 90.2%) and intrahepatic cholangiocarcinoma (ICC, n = 2,188, 8.2%) were the two most common malignant tumors. In contrast, undifferentiated embryonal sarcoma (UES, n = 34, 0.1%) and hepatoblastoma (HB, n = 33, 0.1%) ranked third, with a similar incidence. Histopathologically, HCC, which comprises more than 10 histological varieties[6], is always the central point of differentiated diagnoses among PHSOLs and metastatic tumors. We propose that CD34 immunostaining is one of the most effective methods to distinguish well-differentiated HCC from benign hepatocellular tumors (Figure 1)[6,26]. When HCC appears as a tubular-like arrangement, with solid nest structures and a pseudoglandular pattern, it is difficult to distinguish from ICC or metastatic adenocarcinomas. Based on scanning a panel of immunohistochemical markers, we propose that, for the diagnosis of HCC, Hep Par 1, CD34, and polyclonal CEA are first-line antibodies, and CK19 and MUC-1 are first-line antibodies for ICC[6,31].

UES is a unique hepatic malignant tumor that usually affects the pediatric population. To the best of our knowledge, only 70 cases of UES in adults have been reported worldwide[6,93]. Histologically, UES is characterized by a huge hemorrhagic mass and is composed of pleomorphic cells with eosinophilic cytoplasmic globules entrapped in a loose myxoid stroma[78]. Among 34 cases of UES in

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**Table 2** Histological classification of benign primary hepatic space-occupying lesions

| Category | Subcategory |
|----------|-------------|
| Hepatocellular tumors | Hepatocellular adenoma, and hepatic adenomatosis |
| Intrahepatic bile duct tumors | Bile duct cystadenoma, and intraductal papillary neoplasms |
| Vascular and lymphoid tumors | Cavernous hemangioma, and perivascular epithelioid cell tumor |
| Muscle, fibrous and adipose tumors | Angiomyolipoma, leiomyoma, solitary fibrous tumor, and lipoma |
| Neuronal and neuroendocrine tumors | Neurilemmoma, paraganglioma, and plexiform neurofibromatosis |
| Neuronal and neuroendocrine tumors | Neuroendocrine tumors |
| Miscellaneous tumors | Myxoma, spongiotic pericytoma, and plasmacytoma |
| Muscle, fibrous and adipose tumors | Myxoma, spongiotic pericytoma, and plasmacytoma |
| Muscle, fibrous and adipose tumors | Myxoma, spongiotic pericytoma, and plasmacytoma |
| Muscle, fibrous and adipose tumors | Myxoma, spongiotic pericytoma, and plasmacytoma |

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Figure 1  Atypical focal nodular hyperplasia with minimal fibrous septa (A, HE stain, × 200) shows focal microvessels around the periphery of the fibrous septa (B, CD34 immunostaining, × 200). Hepatocellular adenoma is composed of benign-looking hepatocytes with mild steatosis, without a capsule around the periphery (C, HE stain, × 200), and shows a chaotic microvessel distribution pattern with thin-walled vascular staining (D, CD34 immunostaining, × 200). Highly differentiated hepatocellular carcinoma is arranged in a thin trabecular pattern (E, HE stain, × 400) and shows a sinusoidal capillarization pattern (F, CD34 immunostaining, × 200).

Figure 2  Infiltration of neoplastic cells of hepatic angiomyolipoma within the hepatic sinusoid (A, HE stain, × 200) with strong HMB45 positive staining (B, immunostaining, × 200), and within a branch of the portal vein (C, HE stain, × 100) with strong HMB45 positive staining (D, immunostaining, × 200).

the EHBH series, 32.4% (n = 11) and 29.4% (n = 10) occurred in patients less than 12 and older than 50 years of age (range 5-70 years), respectively, and 32.4% (n = 11) had hepatitis B virus (HBV) infection, suggesting a possible causal link between chronic HBV infection and UES development.
The incidence of primary hepatic lymphoma (PHL, 0.09%, $n = 23$) was similar to that of UES and HB (0.1%). It has been reported that hepatitis C virus (HCV) plays a role in the pathogenesis of lymphoma, with an HCV prevalence rate of 9% to 42%, especially in Western countries. In contrast, the prevalence of HCV in our patients with PHL was only 4.3% (1 of 23 cases), whereas 56.5% (13 of 23) were positive for HBV, and three of them underwent surgical resections for simultaneous coexistence of PHL with HCC as two independent masses in the liver. Thus, we hypothesize that HBV, as a kind of lymphotropic virus, may play an important pathogenic role in the development of PHL in China.

### Table 3  Histological classification of malignant primary hepatic space-occupying lesions

| Tumor Type                        | Subtype                                      |
|-----------------------------------|----------------------------------------------|
| Hepatocellular tumors             | Hepatocellular carcinoma[2], Hepatoblastoma[3], Combined hepatocellular and cholangiocarcinoma[2] |
| Intrahepatic bile duct tumors     | Intrahepatic cholangiocarcinoma[2]            |
| Cholangiocarcinoma[2]             | Bile duct cystadenocarcinoma[2]               |
| Biliary rhabdomysosarcoma[26]     | Solid-pseudopapillary tumor[26]               |
| Vascular, lymphoid and haemopoitetic tumors | Angiosarcoma[2], Malignant angiomylipoma/malignant perivascular epithelioid cell tumor[2] |
| Neuronal and neuroendocrine tumors | Carcinoid tumor[2], Malignant neurilemmoma[2] |
| Miscellaneous tumors              | Undifferentiated embryonal sarcoma[73], Undifferentiated carcinoma[2], Carcinosarcoma[2], Lymphoepithelioma-like carcinoma[2], Squamous cell carcinoma[2], Germ cell tumor[2], Choriocarcinoma[2], Yolk sac tumor[2], Immature teratoma[2], Malignant rhabdoid tumor[2], Malignant mesothelioma[2], Synovial sarcoma[2], Epithelial-myoepithelial carcinoma[2], Gastrointestinal stromal tumor[2], Osteosarcoma[2], Osteoclast-like giant cell tumor[2], Desmoplastic small round cell tumor[2], Nested stromal-epithelial tumor[2]/ossifying stromal epithelial tumor[2] |

CONCLUSION

In summary, based on the large number of surgically resected PHSOLs in the EHBH series, we propose a comprehensive surgicopathological classification system that comprises more than 100 kinds of PHSOLs, with three basic types and six subtypes. Our classification system covers all the entities in the new histological classification system generated by the WHO, which included about 30 kinds of PHSOLs, except for microscopic cellular abnormalities. We do not describe details concerning molecular genetics, diagnostic criteria, biological behaviors, treatment strategies, and clinical prognoses for each PHSOL, as they can be found in the given references. Although it is still possible that new types of PHSOLs will be discovered, we think that the above brief summary may provide useful information as a new classification system and current reference for clinicians and pathologists to understand the features of histological spectrum, as well as the differential diagnostic features of PHSOLs.

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