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Original Research

Histopathological Assessment of Cases of Hepatoblastoma

Dr. Ishani Gupta[1], Dr. Anuradha Sarmal[2]

[1]Senior resident, [2]Junior resident, Department of Pathology, GMC Jammu, J & K, India

ABSTRACT:

Background: Primary hepatic malignancies in children comprise about 0.5–2% of all solid tumors in children. The present study was conducted to assess cases of hepatoblastoma histopathologically. **Materials & Methods:** 48 cases of hepatoblastoma of both genders were recorded. PRETEXT staging was assigned using contrast computed tomography (CT). **Results:** Out of 48 cases, males were 28 and females were 20. Common complaints of patients was vomiting in 20, abdominal distension in 38, jaundice in 16 and abdomen mass in 14 cases. Out of 48 cases, epithelial type was seen in 32 and mixed epithelial and mesenchymal in 16. Epithelial type 8 was pure fetal in 18, fetal mitotically active in 6, embryonal in 4 and mixed epithelial in 4 cases. The difference was significant (P< 0.05). **Conclusion:** Maximum cases were epithelial type as compared to mixed epithelial and mesenchymal and females predominance was observed.

Key words: Hepatoblastoma, Mitotical, Embryonal

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Corresponding author: Dr. Anuradha Sarmal, Junior resident, Department of Pathology, GMC Jammu, J & K, India

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INTRODUCTION

Primary hepatic malignancies in children comprise about 0.5–2% of all solid tumors in children of which hepatoblastoma is the most common with an incidence of 1.5 cases per million children less than 18 years of age worldwide, followed by hepatocellular carcinoma. It mostly occurs in children less than 5 years of age with a slight preponderance to males.[1] Factors such as prematurity, low-birth weight, maternal smoking, alcohol, oral contraceptive use, and methods of assisted reproduction are some risk factors. It is also seen to be associated with syndromes such as Beckwith-Wiedemann syndrome, familial adenomatous polyposis, Li-Fraumeni syndrome, trisomy 18, and other metabolic disorders.[2]

The HBs were encapsulated, with a nodular, grey-white outer surface observed in three cases. The cut surface was tan to greywhite and feshy with areas of haemorrhage and necrosis. Translucent areas were noted in one tumour. Haemotoxylin and eosin (H & E)-stained histological sections from the neoplasms were reviewed. The analysis included tumour subtype, necrosis, and mitotic activity. Histological criteria used for diagnosis of HB have been summarised by Weinberg and Finegold.[3]

Histology is very important because it is incorporated as a risk stratification parameter in the Children's oncology group (COG) protocols for planning treatment.[4] It is seen that each of the histological parameters have distinct clinical associations. Very few studies have mentioned proper histologic favorability as per College of American Pathologists (CAPs) protocols, correct Pretreatment extent of tumor (PRETEXT) staging, and the risk status.[5] The present study was conducted to assess cases of hepatoblastoma histopathologically.

MATERIALS & METHODS

The present study was conducted among 48 cases of hepatoblastoma of both genders. All were informed regarding the study and their consent was obtained.

Data such as name, age, gender etc. was recorded. Laboratory parameters such as liver function tests;

alpha fetoprotein levels (APLs); complete blood count, etc. were recorded. PRETEXT staging was assigned using contrast computed tomography (CT). Histological data on biopsy and/or resection specimens and follow-up data on survival/response to therapy were collected and were analyzed. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of cases

Total- 48		
Gender	**Males**	**Females**
Number	28	20

Table I shows that out of 48 cases, males were 28 and females were 20.

Table II Clinical symptoms

Clinical symptoms	Number	P value
Vomiting	20	0.012
Abdominal distension	38	
Jaundice	16	
Abdomen mass	14	

Table II, graph I shows that common complaints of patients was vomiting in 20, abdominal distension in 38, jaundice in 16 and abdomen mass in 14 cases. The difference was significant (P< 0.05).

Graph I Clinical symptoms

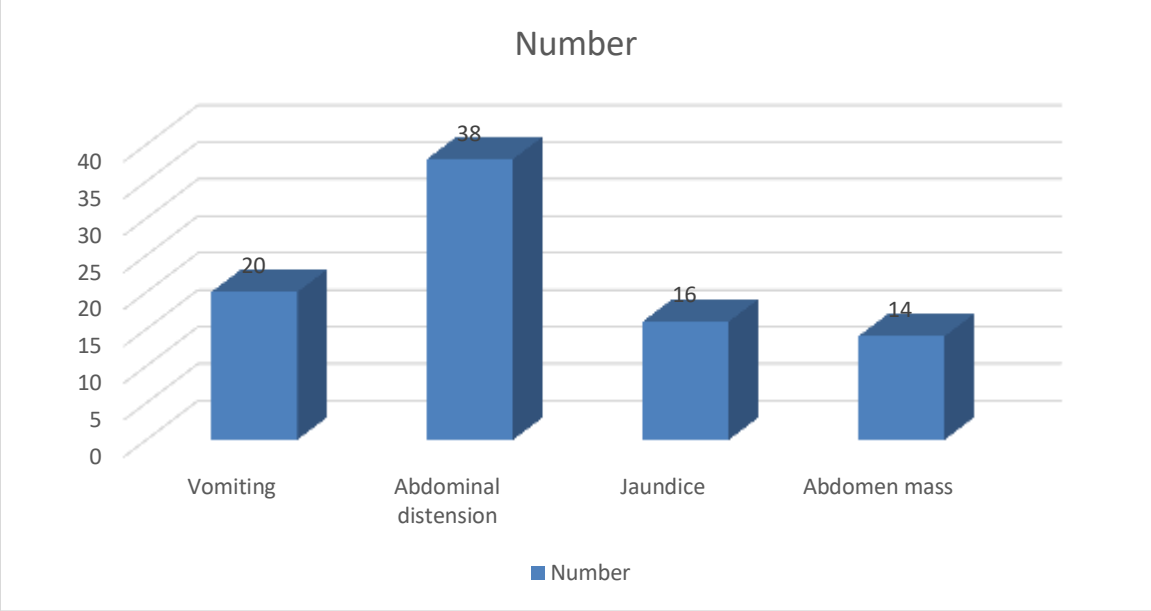
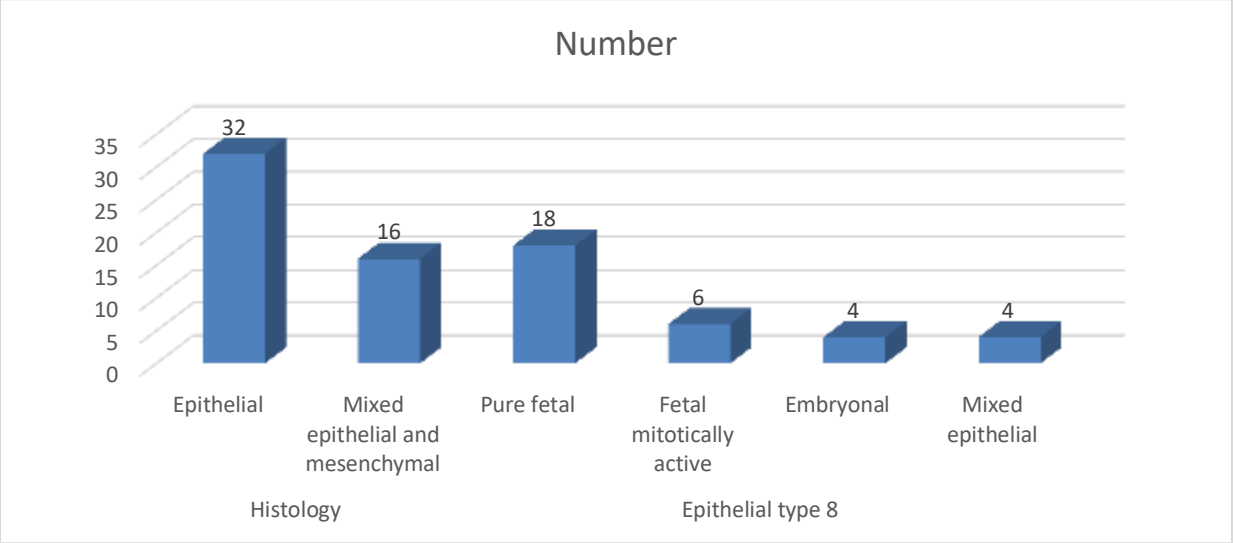


Table III Histopathological features

Variables	Parameters	Number	P value
Histology	Epithelial	32	0.04
	Mixed epithelial and mesenchymal	16	
Epithelial type 8	Pure fetal	18	0.12
	Fetal mitotically active	6	
	Embryonal	4	
	Mixed epithelial	4	

Table III, graph I shows that out of 48 cases, epithelial type was seen in 32 and mixed epithelial and mesenchymal in 16. Epithelial type 8 was pure fetal in 18, fetal mitotically active in 6, embryonal in 4 and mixed epithelial in 4 cases. The difference was significant (P< 0.05).

Graph I Histopathological features



DISCUSSION

According to the Los Angeles Children's oncology group (LACOG) liver tumors symposium consensus, mitotically active fetal type necessitates chemotherapy, and the small cell type requires extensive therapy. Small cell histological type has further been classified into INI positive/negative.[6] INI1 positive tumors are associated with a good prognosis. Histological favorability is also to be incorporated in the final report as favorable histology corresponds to a good prognosis compared to less favorable and unfavorable histological grade. Tumors of the liver, although relatively infrequent in childhood, pose a considerable therapeutic and diagnostic challenge.[7] Primary neoplasms of the liver comprise 0.5% to 2% of all pediatric malignancies, and are the tenth most frequent tumours in children. However, 15% of all abdominal tumours in childhood are primary liver tumours; 66% of these are malignant, the commonest being hepatoblastoma (HB). Complete surgical resection of the tumour with adjuvant chemotherapy is associated with good survival.[8] The present study was conducted to assess cases of hepatoblastoma histopathologically.

In present study, out of 48 cases, males were 28 and females were 20. We found that common complaints of patients was vomiting in 20, abdominal distension in 38, jaundice in 16 and abdomen mass in 14 cases. Archana et al[9] in their retrospective study on 10 children diagnosed with hepatoblastoma, the median age of these children at diagnosis was 11 months, and only 1 child was premature at birth. Most children were presented with abdominal distension. One child had lung metastasis at presentation. Elevated alpha fetoprotein levels were present in 90% of the children. The histological types were fetal, embryonal, macrotrabecular, and mixed epithelial-mesenchymal types. SIOPEL risk stratification was done, which showed 40% of the children to be of high risk. Three children had PRETEXT 1, 2, and 4, respectively.

We found that out of 48 cases, epithelial type was seen in 32 and mixed epithelial and mesenchymal in 16. Epithelial type 8 was pure fetal in 18, fetal mitotically active in 6, embryonal in 4 and mixed epithelial in 4 cases. Bhattacharya et al[10] observed nine cases between 1976 and 1995, of which eight were hepatoblastomas and one a mesenchymal hamartoma. A male-to-female ratio of 2:1 was noted; 78% of the cases occurred in children below 2 years of age. Five hepatoblastomas were of the pure epithelial type; mesenchymal components encountered in the mixed type were cartilage, bone, and spindle sarcomatous cells.

HB is known to be associated with disorders such as Beckwith-Wiedemann syndrome, familial adenomatous polyposis, and congenital anomalies like hemihypertrophy and cleft palate, Wilms' tumour, and glycogen storage diseases. Isosexual precocity resulting from human chorionic gonadotropin (HCG) elaboration by the tumour is seen in some cases.[11] The known association between HB and Beckwith-Wiedemann syndrome has indicated a role for abnormalities on chromosome 11, and loss of heterozygosity on chromosome 11p has been documented in tumour tissue. The increased incidence of HB in families with

familial adenomatous polyposis indicates a possible significance for abnormalities of chromosome 5q. HB has also been reported in association with maternal ingestion of contraceptives and gonadotropins and in the fetal alcohol syndrome.[12]
The shortcoming of the study is small sample size.

CONCLUSION

Authors found that maximum cases were epithelial type as compared to mixed epithelial and mesenchymal and females predominance was observed.

REFERENCES

1. Reynolds P, Urayama KY, Von Behren J, Feusner J. Birth characteristics and hepatoblastoma risk in young children. Cancer 2004;100:1070-6.
2. Roebuck DJ, Aronson D, Clapuyt P, Czauderna P, de Ville de Goyet J, Gauthier F, *et al* 2005 PRETEXT: A revised staging system for primary malignant liver tumours of childhood developed by the SIOPEL group. Pediatr Radiol 2007;37:123-32.
3. Herzog CE, Andrassy RJ, Eftekhari F. Childhood cancers: hepatoblastoma. Oncologist. AlphaMed Press; 2000;5:445-53.
4. López-Terrada D, Alaggio R, de Dávila MT, Czauderna P, Hiyama E, Katzenstein H, *et al*. Towards an international pediatric liver tumor consensus classification: Proceedings of the Los Angeles COG liver tumors symposium. Mod Pathol. Nature Publishing Group 2013;27:472.
5. Stocker JT. Hepatic tumors in children. Clin Liver Dis 2001;5:259-81.
6. Fasano M, Theise ND, Nalesnik M, Goswami S, Garcia de Davila MT, Finegold MJ, *et al*. Immunohistochemical evaluation of hepatoblastomas with use of the hepatocyte-specific marker, hepatocyte paraffin 1, and the polyclonal anti-carcinoembryonic antigen. Mod Pathol 1998;11:934-8.
7. Arora RS, Ramandeep D, Arora S. Outcomes of Hepatoblastoma in the Indian Context. Indian Pediatrics 2012;49:307-9.
8. Mesquita MC de O, Ferreira AR, Veloso LF, Roquete MLV, Lima AS de, Pimenta JR, *et al*. Pediatric liver transplantation: 10 years of experience at a single center in Brazil. J Pediatr (Rio J);84:395-402.
9. Archana B, Thanka J, Sneha LM, Xavier Scott J J, Arunan M, Agarwal P. Clinicopathological profile of hepatoblastoma: An experience from a tertiary care center in India. Indian J Pathol Microbiol 2019;62:556-60.
10. Bhattacharya S, Lobo FD, Pai PK, Pai GK. Hepatic neoplasms in childhood–a clinicopathologic study. Pediatric surgery international. 1998 Nov 1;14(1-2):51-4.
11. Schnater JM, Aronson DC, Plaschkes J, Perilongo G, Brown J, Otte J-B, et al. Surgical view of the treatment of patients with hepatoblastoma: Results from the first prospective trial of the International Society of Pediatric Oncology Liver Tumor Study Group. Cancer 2002;94:1111-20.
12. Luo J-H, Ren B, Keryanov S, Tseng GC, Rao UNM, Monga SP, et al. Transcriptomic and genomic analysis of human hepatocellular carcinomas and hepatoblastomas. Hepatology. Wiley-Blackwell; 2006;44:1012-24.