

Fine Needle Aspiration Cytology Study and Histopathology Correlation of Liver Diseases - Study of 80 Cases In Ateritiary Care Centre

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ABSTRACT:-

AIM:. Fine needle aspiration (FNAC) is a widely accepted, rapid diagnostic procedure and has been routinely used for all palpable masses. FNAC of liver is extremely safe procedure, simple and almost non- invasive procedure for diagnostic as well as prognostic purposes^{1,2}. With the introduction of ultrasonography (US) and computerized axial tomographic (CAT) scanning many space occupying lesions (SOL) of the liver are accurately localized and have made FNA from the targeted site easier. Primary carcinomas of liver either as a single mass or multiple mass have facilitated several passes (4-6) at the same sitting and good cellular smears which can be studied by routine May Grunwald Geimsa (MGG) stain, Haematoxylin-eosin and Papanicolaou's stain and any other special stain if necessary.^{4,6} The purposive sampling of 80 cases of FNA of the liver lesions were done at Siddhartha government general hospital, Vijayawada from June 2016 to July 2018. In the present study of 80 cases of FNAC liver. 45 cases were non-neoplastic lesions. 29 cases were male and 16 cases were females. Age group varied from 11 years to 74 years and males were nearly twice more frequently affected than females. The study is supported by needle biopsies and cell block studies are analysed with the review of literature.

KEYWORDS:- Fine needle aspiration -FNAC liver lesions, , benign and malignant

I. INTRODUCTION

Fine needle aspiration (FNA) technique has been used as a means of a simple and rapid method of diagnosis since 1927 by Dudgeon and Patrick in United Kingdom, Martin in 1934 at Memorial Hospital in USA. Their study of categorizing began with the most commonly found cytologic feature of HCC continued with the least common of the variables. commonest cytological features were:

Polygonal cells with central nucleus-94.3%

1. Malignant cells separated by capillaries-94.3%
2. Granular, well defined cytoplasm-91.4%
3. Larger nucleoli-85.7%
4. Endothelial cells surrounding tumour cells-77.1%
5. Intranuclear cytoplasmic inclusions -71.4%
6. Polymorphonuclear leukocytes (PMN)-68.6%
7. Small cytoplasmic vacuoles-68.6%
8. Multinucleated tumour giant cells-45.7%
9. Large cytoplasmic vacuoles-37.1%
10. Intracytoplasmic bile-34.3%
11. Eosinophilic intra cytoplasmic inclusions-14.3%
12. Basophilic intracytoplasmic inclusion-11.4%

Out of thirteen cytologic variable the presence of polygonal cells with central nuclei, malignant cells separated by capillaries and intracytoplasmic bile were the most likely criteria to distinguish HCC from metastatic tumours.

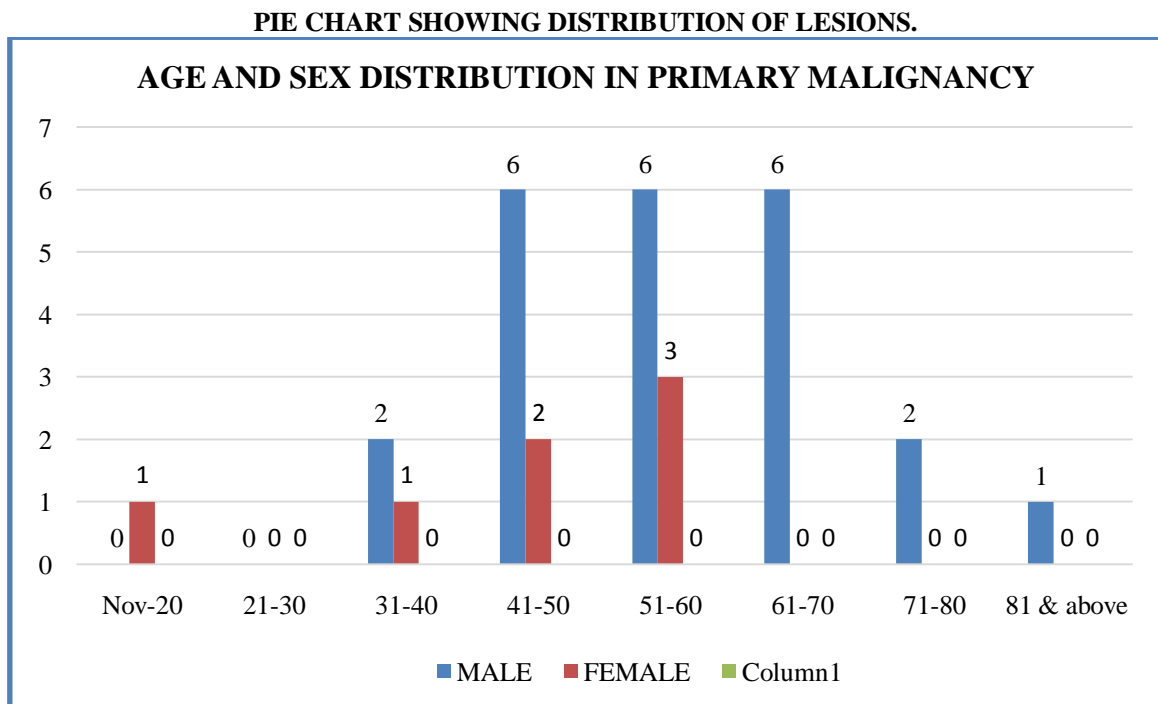
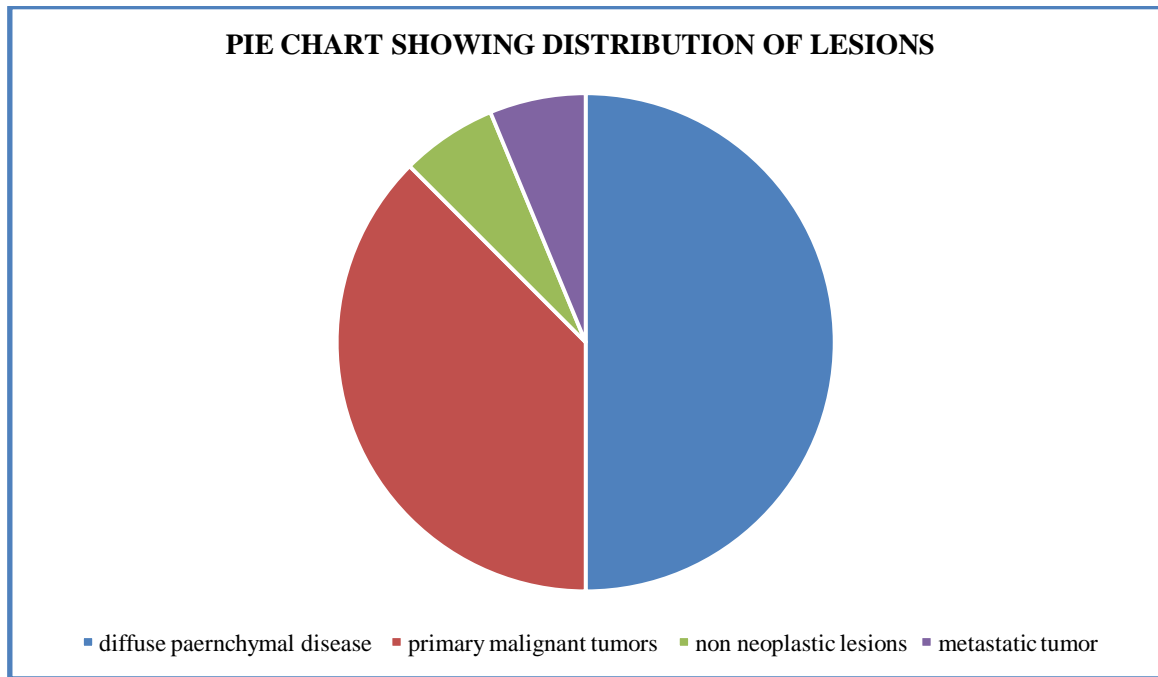
II. MATERIALS & METHODS:

FNAC study of eighty cases of liver disease were undertaken in the patients admitted to government general hospital, Vijayawada from June 2016 to July 2018. A prospective and retrospective study of these cases who showed signs of jaundice, other hepatocellular failure, enlarged liver including the patients who needed ultrasonography/scanning were selected for a purposive sampling by FNA/guided FNAC. Forty cases of chronic

liver disease like chronic hepatitis, cirrhosis, fatty change and unexplained hepatosplenomegaly were studied by blind aspirations. Forty-one FNAs of solitary or multiple space occupying lesion in liver were carried out through US guidance and four CT guided aspirations for deep seated lesions.

For aspiration technique 23G disposable needles of varying length depending on the depth of the lesion, connected to 10ml disposable syringe were used. The aspirations smeared on clean glass slides. A minimum of 4-6 smears were prepared and stained with H&E stain and 2 air dried for MGG stain.

After excluding the contraindications for liver biopsy procedure such as bleeding diathesis by coagulation profile , gross ascites, chronic passive congestion, haemangioma of liver and hydatid cyst, biopsy procedure was carried out in 16 diffuse liver pathology and 7 HCC where cytologic diagnosis was inconclusive. Biopsy study undertaken for a confirmative diagnosis.



III. OBSERVATIONS

In the present study of 80 cases of FNAC liver. 45 cases were non-neoplastic lesions. 29 cases were male and 16 cases were females. Age group varied from 11 years to 74 years and males were nearly twice more frequently affected than females. Out of 30 primary malignant tumours 23 were males, 7 were females varied in the age group from 14 years to 82 years with the peak incidence of 41 years to 71 years.

Five cases of metastatic lesions, the age group affected was between 30 years to 65 years and all were males. Blind aspirations are carried out in 40 diffuse parenchymatous liver disease cytological diagnosis and histological confirmation is tabulated.

SL.NO	DISEASE	FNAC	CELL BLOCK	BIOPSY
1.	No significant pathology	02	-	-
2.	Chronic hepatitis	08	-	05
3.	Tuberculosis*	01	-	01*
4.	Fatty change	07	-	03
5.	Alcoholic liver disease	06	-	02
6.	Cirrhosis**	10	-	04**
7.	Myeloid Metaplasia	01	-	-
SL.NO	DISEASES	FNAC	CELL BLOCK	BIOPSY
8.	Dysplasia	04	-	02
9.	Amyloidosis*	01	01*	-
	Total	40	01	16

NON NEOPLASTIC CYSTIC SPACE OCCUPYING LESIONS

SL.NO	DISEASES	FNAC	BIOPSY
1.	Pyemic abscess	03	-
2.	Hydatid disease	02	-
	Total	05	-

**Biopsy not done because of danger of fulminant complications.

SPACE OCCUPYING LESIONS -PRIMARY TUMORS

SL.NO	DISEASE	FNAC	CELL BLOCK	BIOPSY
1.	Hepatocellular carcinoma(HCC)			
	Well differentiated HCC	13	04	02
	Moderately differentiated HCC	10	03	01
	Poorly differentiated HCC	04	-	02
	Clear cell variant HCC	01	-	01
	Small cell variant HCC	01	-	01
2.	Angiosarcoma	01	-	-
	Total	30	07	

Among cases cited above the diagnosis of well differentiated HCC were difficult to differentiate from cirrhotic regenerating nodule cytologically in 3 cases. Biopsy procedure was carried out which showed the features of well differentiated HCC, with cells arranged in thick trabecular and acinar pattern and these cells showed increased nuclear-cytoplasmic ratio with prominent nucleoli and intracytoplasmic bile.

SPACE OCCUPYING LESIONS- METASTATIC TUMORS

SL.NO	DISEASES	FNAC	CELL BLOCK	BIOPSY
1.	Metastatic adenocarcinoma			
	Git	03	02	-
	pancreas	01	-	-
2.	Lymphoma	01	01	-
	Total	05	03	

Since the lesion was very deep seated in the liver with pancreatic mass, biopsy could not be done.

IV. DISCUSSION:

A total of eighty liver aspirates were performed from June 2016 to July 2018. In present series 40 cases of chronic liver diseases with palpable / enlarged liver were preferred and repeat aspirations were done in 8 cases. Among diffused parenchymatous lesions cytological diagnosis was possible in 24 out of 40 cases giving a positive rate of 60 percent.

The diffused parenchymatous lesions like chronic hepatitis (3 cases), tuberculosis (1 cases), fatty change(4 cases),alcoholic liver disease (4 cases) cirrhosis (6 cases), dysplasia (2) ,Myeloid metaplasia(1cases), amyloid (1 cases), and with no significant pathology (2 cases) were accurately diagnosed cytologically gave the cytological positivity for non-neoplastic diffused lesions.16 needle biopsy study of inconclusive cytological study gave a final histological diagnosis in chronic hepatitis (5 cases), fatty change (3 cases), alcoholic liver disease (2 cases), cirrhosis (4 cases), dysplasia (2 cases). 1 case of tuberculosis and amyloidosis liver also showed the same lesions on biopsy and cell block study respectively, confirming the cytological study.5 cases of non -neoplastic space occupying lesions, diagnosed by US guidance and on FNAC study showed 3 cases of pyemic abscess and 2 cases of hydatid disease giving a cytological positivity of 100 %. Biopsy not needed as cases are contraindicated for study.From the study of 35 malignant tumours of the liver, a few diagnostic difficulties were encountered while reporting FNAC of the liver.

V. CONCLUSION:

FNAC of liver is an extremely simple and safe procedure, only one case developed haematoma which was drained through US guidance. For diffused enlargement of liver FNA was diagnostic in 24 out of 40 cases giving an accuracy rate of 60 percent. 16 (40 percent) biopsies were needed for confirmative diagnosis in doubtful and inconclusive cytological diagnosis. Both the techniques together gave a specificity of 100 percent. There were 5 non neoplastic cystic lesions in the liver like pyemic abscess(3 cases) and hydatid cyst (2 cases) diagnosed cytologically.30 primary malignant lesions were cytologically diagnosed, differentiated and graded cytologically. biopsy was done in two cases to differentiate well differentiated HCC from regenerating cirrhotic nodules as both resembled remarkably and thus biopsy was complimentary for a final diagnosis of HCC. The malignant lesions gave a better cytological sensitivity of 80 percent and biopsy confirmation was needed in 20 percent. FNAC study a histological confirmation together gave a specificity of 100 percent.Five cases of metastatic tumours were cytologically diagnosed and they presented with localized lesions at the time of hospitalization.

It is not necessary to perform both FNAC and biopsy procedures, though histopathological study is scientific bases of FNAC “LIVER BIOPSY AND CYTOLOGICAL STUDY ARE COMPLIMENTARY TO EACH OTHER”.

REFERENCES

[1]. Millward-Sadler GH . Cirrhosis. In: MacSween RNM, Anthony PP, Scheuer PJ, Burt AD, Portmann BC, editors. *Pathology of the liver*. 3rd ed. London:Churchill Livingstone; 1994. p. 397–424.

[2]. Nevens F, Staessen D, Sciote R, Van Damme B, Desmet V, Fevery J, *et al*. Clinical aspects of incomplete septal cirrhosis in comparison with macronodular cirrhosis. *Gastroenterology* 1994; **106**: 459–63.

[3]. Baptista A, Bianchi L, De Groote J, Desmet V, Gedigk P, Korb G, *et al*. Alcoholic liver disease: morphological manifestations. *Lancet* 1981; **1**: 707–11

[4]. Lewis J, Mullick F, Ishak K, Ranard R, Ragsdale B, Perse R, *et al*. Histopathologic analysis of suspected amiodarone hepatotoxicity. *Hum Pathol* 1990; **21**: 59–67.

[5]. Babany G, Uzzan F, Larrey D, Degott C, Bourgeois P, Rene E, *et al*. Alcoholic-like liver lesions induced by nifedipine. *J Hepatol*1989; **9**: 252–5.

- [6]. Snover D. The liver in systemic disease. In: *Biopsy diagnosis of liver disease*. 1st ed. Baltimore: Williams & Wilkins; 1992. p. 192–4.
- [7]. Arcidi J, Moore G, Hutchins G. Hepatic morphology in cardiac dysfunction: a clinico-pathologic study of 1000 subjects at autopsy. *Am J Pathol* 1981; **104**: 159–66.
- [8]. Ruoslahti E, Seppala M, Vuopio P, Saksela E, Peltokallio P. Radioimmunoassay of alpha-fetoprotein in primary and secondary cancer of the liver. *J Natl Cancer Inst* 1972, 49: 623–630.
- [9]. Trevisani F, D’Intino PE, Grazi GL, Caraceni P, Gasbarrini A, Colantoni A, Stefanini GH, Mazziotti A, Gozzetti G, Gasbarrini G, Barnardi M. Clinical and pathologic features of hepatocellular carcinoma in young and older Italian patients. *Cancer* 1996, 77: 2223–2232.
- [10]. Akagi G, Furuya K, Kanamura A, Chihara T, Otsuka H. Liver cell dysplasia and hepatitis B surface antigen in liver cirrhosis and hepatocellular carcinoma. *Cancer* 1984, 54: 315–318.
- [11]. Alpert ME, Davidson CS. Mycotoxins. A possible cause of primary carcinoma of the liver. *Am J Med* 1969, 46: 325–327.
- [12]. Anthony PP. Hepatocellular carcinoma: an overview. *Histopathology* 2001, 39: 109–118. Anthony PP, Vogel CL, Barker LF. Liver cell dysplasia. A premalignant condition. *J Clin Pathol* 1973, 26: 217–223.
- [13]. Aterman K. Hepatic neoplasia. Reflections and ruminations. *Virchows Arch* 1995, 427: 1–18.
- [14]. Balazs M. Primary hepatocellular tumours during long-term androgenic steroid therapy. A light and electron microscopic study of 11 cases with emphasis on microvasculature of the tumours. *Acta Morphol Hung* 1991, 39: 201–216.
- [15]. Boyd PR, Mark GJ. Multiple hepatic adenomas and a hepatocellular carcinoma in a man on oral methyl testosterone for eleven years. *Cancer* 1977, 40: 1765–1770.
- [16]. Brambilla C, Tackney C, Hirschman SZ, Colombo M, Dioguardi ML, Donato MF, Paronetto F. Varying nuclear staining intensity of hepatitis B virus DNA in human liver. A model tumor. A review. *Am J Pathol* 1974, 74: 179–200.
- [17]. Kojiro M, Kawabata K, Kawano Y, Shirai F, Takemoto N, Nakashima T. Hepatocellular carcinoma presenting as intrabiliary duct tumor growth. A clinicopathologic study of 24 cases. *Cancer* 1982, 49: 2144–2147.
- [18]. Kojiro M, Nakahara H, Sugihara S, Murakami T, Nakashima T, Kawasaki H. Hepatocellular carcinoma with intra-atrial tumor growth. A clinicopathologic study of 18 autopsy cases. *Arch Pathol Lab Med* 1984, 108: 989–992.
- [19]. Kondo Y, Wada K. Intrahepatic metastasis of hepatocellular carcinoma. A histopathologic study. *Hum Pathol* 1991, 22: 125–130.
- [20]. Liaw CC, Ng KT, Chen TJ, Liaw YF. Hepatocellular carcinoma presenting as bone metastasis. *Cancer* 1989, 64: 1753–1757.
- [21]. Linder GT, Crook JN, Cohn I Jr. Primary liver carcinoma. *Cancer* 1974, 33: 1624–1629.
- [22]. Okazaki N, Yoshino M, Yoshida T, Hirohashi S, Kishi K, Shimosato Y. Bone metastasis in hepatocellular carcinoma. *Cancer* 1985, 55: 1991–1994.
- [23]. Young RH, Gersell DJ, Clement PB, Scully RE. Hepatocellular carcinoma metastatic to the ovary. A report of three cases discovered during life with discussion of the differential diagnosis of hepatoid tumors of the ovary. *Hum Pathol* 1992, 23: 574–580.
- [24]. Yuki K, Hirohashi S, Sakamoto M, Kanai T, Shimosato Y. Growth and spread of hepatocellular carcinoma. A review of 240 consecutive autopsy cases. *Cancer* 1990, 66: 2174–2179.
- [25]. Pappo O, Yunis E, Jordan J, Jaffe R, Mateo R, Fung J, *et al*. Recurrent and de novo giant cell hepatitis after orthotopic liver transplantation. *Am J Surg Pathol* 1994; **18**: 804–13.
- [26]. Devaney K, Goodman Z, Ishak K. Postinfantile giant-cell transformation in hepatitis. *Hepatology* 1992; **16**: 327–33.
- [27]. Chen M-F, Jan Y-Y, Jeng L-B, Hwang T-L, Wang C-S, Chen S-C. Obstructive jaundice secondary to ruptured hepatocellular carcinoma into the common bile duct. Surgical experiences of 20 cases. *Cancer* 1994, 73: 1335–1340.
- [28]. Cong WM, Wu MC, Zhang XH, Chen H, Yuan JY. Primary hepatocellular carcinoma in women of mainland China. A clinicopathologic analysis of 104 patients. *Cancer* 1993, 71: 2941–2945.
- [29]. El-Serag HB, Mason AC. Rising incidence of hepatocellular carcinoma in the United States. *N Engl J Med* 1999, 340: 745–750.
- [30]. Farhi DC, Shikes RH, Murari PJ, Silverberg SG. Hepatocellular carcinoma in young people. *Cancer* 1983, 52: 1516–1525.
- [31]. Ferrucci JT. Liver tumor imaging. *Cancer* 1991, (Suppl 4): 1189–1195.
- [32]. Higgingson J. The epidemiology of primary carcinoma of the liver. In Pack GT, Islami AH (eds): *Tumors of the liver*. Vol. 26 of Recent results in cancer research. Heidelberg, 1970, Springer-Verlag.

- [33]. Ikeda T, Tozuka S, Hasumura Y, Takeuchi J. Prostaglandin E-producing hepatocellular carcinoma with hypercalcemia. *Cancer* 1988, 61: 1813–1814.
- [34]. Klein WM, Molmenti EP, Colombani PM, Grover DS, Schwarz KB, Boitnott J, Torbenson MS. Primary liver carcinoma arising in people younger than 30 years. *Am J Clin Pathol* 2005, 124: 512–518.

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