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Giant hepatocellular adenoma- A rare case report

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Abstract: *The authors describe a case of a large hepatocellular adenoma diagnosed in a 22 year old female who came to us complaining of acute pain in upper abdominal quadrants. The patient had been taking oral contraceptive pills for the last 5 years. We present the clinical presentation and the diagnostic work up of the patient.*

Keywords: *Large hepatocellular adenoma,Oral contraceptive pills.*

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I. Introduction

Hepatic adenomas, or hepatocellular adenomas are rare benign hepatic neoplasms that typically affect young women of child bearing age. They are frequently located in right hemiliver and solitary in up to 80 %. Since the introduction of oral contraceptive pills in early 1960s, it has become increasingly apparent that long term use of OCPs is associated with a 30 fold increase in hepatic adenoma incidence as illustrated by numerous epidemiological studies[1]. With the advent of newer low dose estrogen and /or progesterone formulations, the incidence of hepatic adenomas is declining again. Adenomatosis refers to the presence of more than 10 hepatic adenomas and represents a distant disease entity because there is no relationship with hormone exposure.

II. Case presentation

A 22 years female presented in surgery OPD of RIMS, Ranchi with complaint of frequent attacks of pain abdomen mainly in left upper abdomen along with nausea and vomiting after having meals on several occasions. The patient had no history of abdominal disease and reported that she had been taking a contraceptive pill for the last 5 years.Clinical examination of the abdomen revealed a painful, palpable mass of 6-8 cm in diameter in left upper abdomen.Laboratory tests at hospital admittance showed a slight increase in serum transaminases (AST 56 U/L, ALT 73 U/L) whereas Hb, GGT , total and fractionated bilirubin, glycaemia were within normal limits. Markers for Hepatitis B and C were negative.X- ray Abdomen did not have any significant finding, ultrasound revealed a neoformation of about 20 cms in left hepatic lobe, with clear margins, the mass presented a dyshomogeneous echo, with hypoechogenic areas alternating with hyperechogenic zones.

An abdominal CT scan using a contrast medium and triphasic techniques confirmed the presence of a nodular lesion of about 20 cms in diameter in left lobe of the liver. The lesion showed a dyshomogeneous density with irregular enhancement in arterial phase and late washout. No parenchymal or vascular infiltrations were seen.The diagnostic workup , together with the medical history of long term use of OCPs, led us to suspect a large HCA.Ultrasound guided fine needle aspiration cytology was done from multiple sites (taking all aseptic and precautionary measures), which showed moderately cellular smear showing polygonal hepatocytes, arranged in clusters , having abundant cytoplasm and mild to moderate pleomorphism and having round nuclei with regular border suggestive of hepatocellular adenoma.

A liver biopsy for histopathological examination was performed which showed a shaft of hepatic tissue containing a few vacuolated hepatocytes within an area of widespread necrosis, no portal or biliary structures were present. Morphological examination of the specimen suggested a diagnosis of hepatic adenoma, although this could not be considered as conclusive.As the tumor was extremely large and causing considerable pain, most probably due to distension of Glissons capsule, the patient underwent left hepatectomy. Intra operative ultrasound did not reveal any further lesions. There were no post operative complications and patient was discharged seventh post operative day.The anatomopathological examination showed a mass of 20 cms with a smooth, regular external surface and well defined margins, the walls were of yellowish colour. There was a

wide area of necrotic, haemorrhagic tissue extending as far as Glissons capsule. Microscopic examination showed presence of mature, vacuolated hepatocytes with absence of portal and biliary structures which confirmed the diagnosis of hepatocellular adenoma.

III. Figure

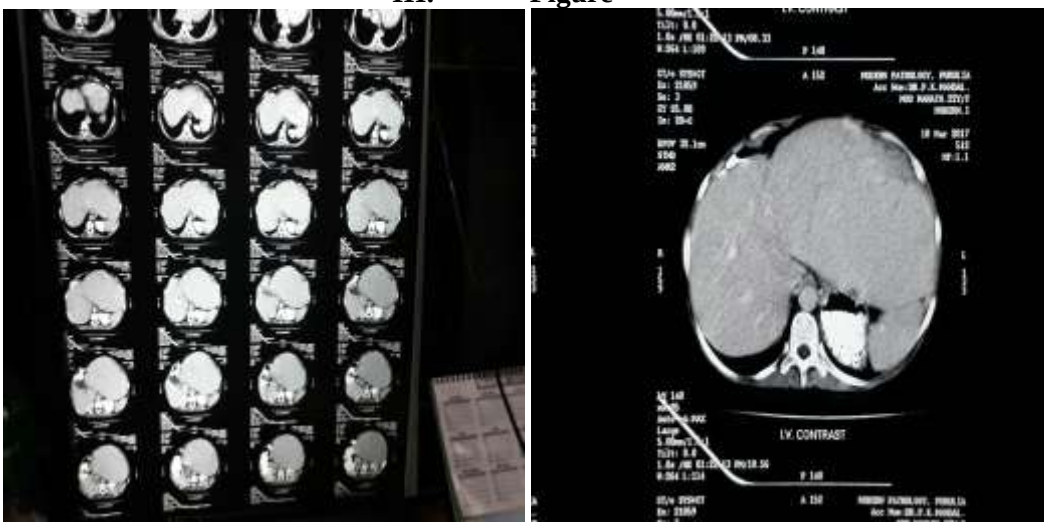

Fig 1 and Fig 2- Triple phase contrast enhanced CT showed nodular growth in left lobe of liver with enhancement in arterial phase and late washout.

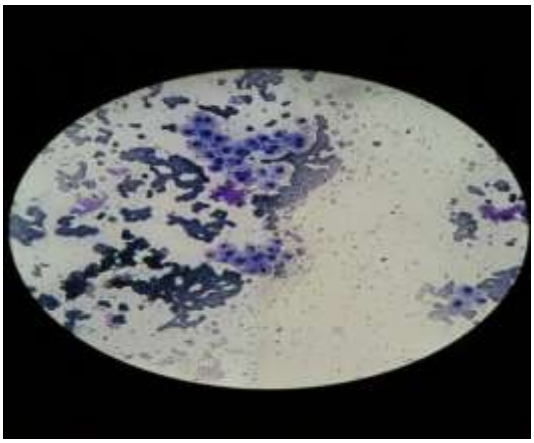

Fig 3 showing moderately cellular smear ,hepatocytes having abundant cytoplasm.

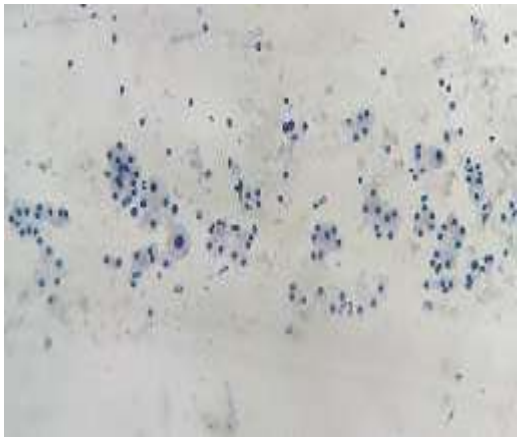

Fig 4 H&E of cytology slide showing mild to moderate pleomorphism and round regular nuclei.

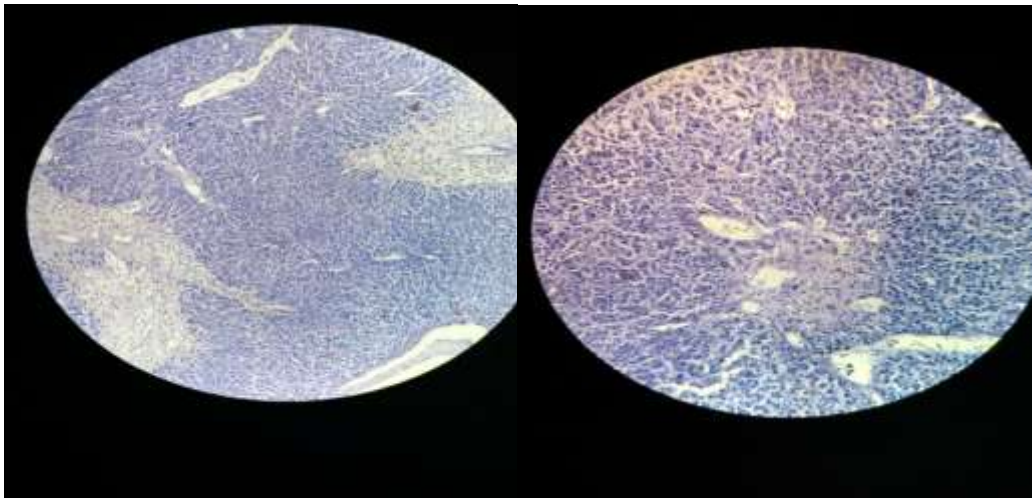

Fig 6 showing necrosis **Fig 7** showing vacuolated hepatocytes with

areas of necrosis.

IV. Conclusion

Hepatocellular adenomas are solitary, sometimes pedunculated , with size ranging from few millimetres to 30 cms. On cut sections, the tumor is soft, white to brown and well delineated with little or no fibrous capsule. Heterogeneous areas of necrosis or haemorrhage may be observed, usually in tumors of large size. Histologically, HCA consists of proliferation of benign hepatocytes of normal size or slightly enlarged with normal nuclear/cytoplaasmic ratio. Hepatocytes are arranged in a trabecular pattern without any residual portal tracts. Small, thin and unpaired vessels without other portal tract elements are found throughout the tumor. The cytoplasm of hepatocytes may be either normal,clear, glycogen rich , or fatty. Compared with FNH, patients with HCA are more likely to be seen with symptoms such as spontaneous bleeding and haemorrhage, with an increased risk according to the size for tumors larger than 5 cm in diameter[2]. The risk of malignant transformation of HCA ranges between 4% and 10 %, with a higher rate in males and in large HCA[2,3]. Recent evidences suggest that metabolic syndrome may favour development of HCC in a pre existing HCA[4]. Increased incidence of metabolic syndrome may partly explain the rising incidence of malignant transformation of HCA, particularly in male population[5].

Patients with multiple HCAs are predominantly females, but the use of OCPs appears to be less prevalent[6]. Patients with type 1 glycogen storage diseases are also at risk for multiple HCAs[7]. These tumors share the same clinical and imaging characteristics independently of their number[2]. A recent study supports that the risk of complications, including bleeding and malignant transformations, is similar to that in patients with solitary HCA, and is not influenced by the number of tumors[2,3].Three main morphological patterns of liver adenomatosis have been described: the steatotic form, the peliotic/telangoectatic form and the mixed form[8]. The proportion of steatotic form is higher, and the presence of microadenomatous foci in the "nontumoral liver" is more often observed in patients with liver adenomatosis[2].

HCAs are subdivided into three main types according to phenotypic and molecular features: hepatocyte nuclear factor 1 alpha mutated steatotic, telangiectatic/inflammatory and beta catenin mutated subtypes[4,9]. A group of HCAs remain unclassified because they do not display any specific morphological or genotypical features. The first group of HCA displays biallelic mutations of the transcription factor 1 (TCF 1) gene inactivating the HNF1 alpha transcription factor. This group is phenotypically characterized by marked steatosis , absence of cytological abnormalities and inflammatory infiltrates[10].The second group of HCAs displays beta catenin activating mutations and is characterized by increased risk of malignant transformation into HCC. These HCAs are mostly found in male patients and frequently show significant cell atypias and pseudoglandular formations[11.]

The third group , the telangiectatic/ inflammatory group are well delineated, unencapsulated tumors with areas of vascular changes without any fibrous scar[3,4,11]. Histologically, the hepatocellular proliferations contain small clusters of arteries embedded in collagen associated with an inflammatory infiltrate of lymphocytes and macrophages and occasionally ductular proliferation. Mild or significant steatosis may be present. These group of HCAs are often seen in patients with increases body mass index associated with inflammatory syndromes[4]. The cardinal feature is activation of Janus Kinase (JAK)/ Signal transducer and activator of transcription pathways, resulting in inflammatory phenotype[12]. The most frequent mutations are observed in interleukin 6 signal transducer (IL6ST) gene, which encodes for signalling coreceptor gp130[12,13].
The fourth group contains HCAs without any characteristic morphological features or genetic abnormalities previously described.

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