Case report

Recurrent non classic focal nodular hyperplasia after extended right hepatectomy; a case report and review of literature

***Make H. Fayadh**

ABSTRACT

A case of a 16 years old female with right upper quadrant pain.
Imaging showed multiple right lobe focal liver lesions,the biopsy was inconclusive.
The atypical presentation and pain necessitated an extended right hepatectomy.
The histologic diagnosis was focal nodular hyperplasia (FNH).2 years after surgery she pres ented with upper abdominal pain with multiple focal lesions in the remaining liver.EUS guided FNA aspirate was suggestive of an atypical non classic focal nodular hyperplasia FNH.
Clinical presentation,and the difficulties facing the clinician in reaching the diagnosis in the atypical cases of FNH is presented.

Background

Focal nodular hyperplasia (FNH) is the most common non-malignant hepatic tumor that is not of vascular origin.In recent series of MRI evaluation,23 percent of benign lesions were FNH,and Of the non-hemangiomatous benign lesions, 86 percent were FNH [1
].FNH is seen in both sexes and throughout the age spectrum, although it is found predominantly in women (in a ratio of 8 or 9:1) between the ages of 20 and 50 years [3
]. FNH comprises up to 2 percent of liver tumors in children [4].

Case presentation

A 16 years old Palestinian female
patient was hospitalized in Feb.2003 with one month history of abdominal pain mainly right hypochondrium,l

- Laboratory investigations showed mildly elevated liver enzymes ALT 65(n 40).Alk.Phosphatase 120(n 100),ESR 40 WBC 9600 60% Lymphocytes.l
- Initial ultrasound and CT scan with no contrast were reported as negative.l
- CT scan with contrast showed mild hepatomegaly with perfusion defect altering the right hepatic lobe enhancement with apparent portosystemic shunting ,possibilities raised were,Splenic vien thrombosis,storage disease,lymphoma
  (fig 1)l
- MRI liver showed normal size liver with large well defined mass 10x7 cm,bilobed,well encapsulated with well defined internal septum,possibilities were adenoma or hemangioma(fig2)

Ultra sound guided Liver biopsy 2004 reported (normal architecture of periportal hepatocytes,fibrosis of portal tracts with some bile duct proliferation and incomplete septa.no chlestasis,no storage disease.)

Slide reviewed by another pathologist reported (morphologic features of the hepatocytes and their relation to the biliary system are similar to the normal liver pattern which favor the diagnosis of focal nodular hyperplasia).
Because of thr elevated ESR,Lymphocytosis,the possibilities raised by the treating physicians at that time were? (Lymphoma, hepatic adenoma,hemangioma,focal nodular hyperplasia (FNH),liver cirrhosis.)
Because of the symptoms her family asked for another opinion in Lebanon .

- Re investigated in Lebanon
- Triphasic CTscan showed 3 well defined lesions segments 5, 6, 7 occupying the right lobe,caudate and one in the left lobe segment 2, a diagnosis of adenoma was made .(fig3)
- Surgery was advised to remove the masses.
- A staging laparascopy was done which confirmed the lesions and biopsy from normal appearing liver was normal.
- An extended right hepatectomy and partial left hepatectomy and cholecystectomy donel
- Histopathology demonstrated nodular pattern with 3 well defined circumscribed lesions with lack of capsule, the large nodule has thin fibrous bands traversing it and dividing it into 3 lobules,

*Department of medicine,Gastroenterology unit,GDC Hospital-Abu Dhabi,UAE

- Microscopic examination showed focal nodular hyperplasia FNH.
- 3 years after operation Feb.2007 presented with recurrent abdominal pain, amenorrhea, acne,mood changes,general weakness and sleepiness.
- Laboratory tests showed mildly raised alkaline phosphatase 130 (N 110)and ammonia level 70 (N 50) with normal transaminases.
- CT scan 2 well demarcated left focal liver lesions(fig4)
- MRI multiple focal lesions in the remaining liver(fig5)
- Upper GIT endoscopy-no esophageal varices .
- She was presented in a meeting during an EUS workshop on Endoscopic Ultra Sound(E.U.S) done in may 2008 showed multiple focal lesions in the remaining liver.EUS guided biopsy from the lesions was confusing and reported as:(Primary hepatocytic parenchymal lesion- hyperplastic or adenomatous with absence of duct epithelium may suggest liver cell adenoma, may be difficult to differentiate from well differentiated (HCC) hepatocelluar carcinoma??
  diagnostic dilemma-because it is an aspirate)
  Slides reviewed by a different pathologist,and reported as
- (the preservation of reticulin pattern favor benign nature)
- At this time the family was advised to continue on conservative management and the question of liver transplant was raised if future deterioration occur.Management
- She was given lactulose and her Sleepiness and altered mood improved.
- The blood ammonia level decreased and is working in a company.

Discussion:

:The initial diagnostic work-up, together with the liver biopsy was suggestive of either an adenoma or an atypical FNH and because of the symptoms she was operated upon with extended right hepatectomy with cholecystectomy.The gross section and histopathology of the resected liver was typical of FNH but the follow up after 2 years showed multiple focal lesions in the remaining liver with elevation of the blood ammonia level and sleepiness which suggested mild hepatic impairment either due to the liver resection.

The re appearance of nodules in the remaining liver raised concerns and so she was re investigated.

Repeated imaging was not helpful.

EUS was performed , aspirates from the lesions were not conclusive because it was an aspirate.

Liver transplant was suggested by experts during a workshop in endosonography 2008 but the patient improved on lactulose and currently back to a normal life.We presume that the cause of the rapid development of these FNHs is due to the growth spurt caused by hormonal influences which we hope will gradually decrease over the coming years.The difficulties faced in the diagnosis and management of these lesions are discussed below

Focal nodular hyperplasia:

Focal nodular hyperplasia (FNH) is the most common non-malignant hepatic tumor that is not of vascular origin, 8 percent of non-hemangiomatous lesions were FNH, 66 percent of all benign non-hemangiomatous lesions seen between 1918 and 1982 [1].

In a more recent series of MRI evaluation,23 percent of benign lesions were FNH,and Of the non-hemangiomatous benign lesions, 86 percent were FNH [2].

FNH is seen in both sexes and throughout the age spectrum, although it is found predominantly in women (in a ratio of 8 or 9:1) between the ages of 20 and 50 years [3].

FNH comprises up to 2 percent of liver tumors in children [4].

PATHOGENESISThe International Working Party of the World Congresses of Gastroenterology proposed a standardized nomenclature in 1994, which placed FNH in the group of regenerative nodules, as opposed to dysplastic or neoplastic nodules [5].

FNH is now generally accepted to be a hyperplastic (regenerative) response to hyperperfusion by the characteristic anomalous arteries found in the center of these nodules [3,8,9].

Imaging study, using ultrasound and dynamic CT, found that 23 percent of FNH patients had associated hemangiomas [14].

FNH with similar clinical and radiographic features has been documented in identical twins supporting a role of congenital vascular anomalies in its pathogenesis and a possible genetic predisposition to the disease [16].

PATHOLOGYFNH is most often solitary (80 to 95 percent), and usually less than 5 cm in diameter. Only 3 percent are larger than 10 cm, although FNH as large as 19 cm have been reported [1,12,17].

It has a sharp margin with no capsule and may be pedunculated. The contain bile ductular proliferation. They almost always lack the characteristic central scar [12].

Three variants have been recognized:

- The most common of these, the telangiectatic type, often presents with multiple FNH. In addition to the lack of a central scar, the mass is characterized by the absence of nodular architecture and the presence of single, quite regular plates of hepatocytes separated by sinusoids fed directly by anomalous arteries [12,19].

The risk of bleeding appears to be similar to the risk observed in patients with hepatic adenoma [20].

- Our patient fits this type of multiple FNH.
- A mixed hyperplastic and adenomatous form may be difficult to distinguish from HA due to its subtle vascular and bile ductular findings [12,19].
- A third histologic variant consisting of FNH with cytologic atypia resembling dysplasia of large cell type has been proposed [12].

A comprehensive pathological study of 305 lesions from Hospital Beaujon failed to identify a macroscopic central stellate scar in 50 percent and noted non-classical histology in 20 percent of the lesions, most showing a telangiectatic variant [12]. The surprisingly high number of lesions without a central scar was almost exclusively due to the large number of masses that had non-classical histology. Ninety-five percent of those with non-classical histology did not have a scar, whereas only 18 percent of those with classical histology lacked a scar [12]. The overall prevalence and clinical significance of these variants remains to be determined.DIAGNOSISThe diagnosis of FNH is usually made by demonstrating its characteristic features on imaging tests and excluding other lesions.

The differential diagnosis includes hepatic adenoma, hepatocellular carcinoma, fibrolamellar carcinoma, cirrhosis, large regenerative nodules, hemangioma, and hypervascular metastases. SymptomsThe majority of reports have found that symptoms and signs directly attributable to FNH are infrequent. Two-thirds to three-fourths of patients are identified incidentally [17], with the mass noted at the time of surgery, on an abdominal imaging study, or at autopsy. Unlike hepatic adenomas, FNH rarely presents with acute onset of hemorrhage, necrosis, or infarction [21,22].

However, symptomatic presentations have been described. In one series, for example, abdominal discomfort or a palpable liver mass was observed in 25 percent of 41 patients [23]. Another series that included 168 patient found that 60 percent had abdominal pain and 4 percent had an abdominal mass [12]. The high number of symptomatic patients in the second report probably reflects selection bias since all of the patients were identified from pathology specimens obtained at the time of surgical resection [12].

Laboratory testsLiver tests are most often normal although minor elevations in aspartate and alanine aminotransferase, alkaline phosphatase and gamma glutamyl transpeptidase levels may be seen [12,13,23].

The alpha-fetoprotein is normal.

Imaging testsA confident diagnosis can usually be made through a combination of imaging modalities; tissue diagnosis is usually not required.

In our patient initial imaging were reported normal include CT scan as these lesions contain normal liver tissue and are are sodense.UltrasoundAlthough often first identified on ultrasound examination, FNH is variably hyper, hypo, or isoechoic [23] and US is able to identify the central scar in only 20 percent of cases [24]. The ultrasound characteristics are difficult to distinguish from an adenoma or malignant lesions. Power Doppler ultrasound may help differentiate the arterial flow in FNH from the venous flow in HA [23,25,26].CT scanA properly timed dynamic, triphasic, helical CT scan performed without contrast, and with contrast during the hepatic arterial and portal venous phases, will often be highly suggestive of the diagnosis [27,28]. The lesion may be hypo or isodense on non-contrast imaging with the central scar identified in one-third of patients. The lesion becomes hyperdense during the hepatic arterial phase due to the arterial origin of its blood supply . FNH is generally isode
nse during the portal venous phase, although the central scar may become hyperdense as contrast diffuses into the scar. While characteristic of FNH, a central scar may be present in the fibrolamellar variant of HCC.

The initial CT scan was reported as normal but when repeated with contrast it demonstrated the lesions but the radiologist was suggesting a vascular anomaly or lymphoma.

Technetium sulfur colloid scanningA characteristic of FNH is that it usually contains Kupffer cells. Thus, 80 percent of lesions will show active uptake of technetium sulfur colloid on nuclear medicine scannings, whereas HA, which lack Kupffer cells, generally will not [27,29-31]. One study suggested that the presence of a "hot spot" on sulfur colloid scanning was comparable to or more sensitive for the diagnosis of FNH than CT or MRI (92 versus 84 percent) [32]. Unfortunately, because occasional HA will also show uptake, a positive nuclear medicine scan is not sufficient for a definitive diagnosis of FNH. In many centers, nuclear imaging has been largely replaced by Gd-BOPTA-enhanced MRI or dynamic multi-phase CT angiography.

MRIThere may be little to distinguish FNH from normal liver on standard MRI, since it is composed of the same elements as normal liver. An isointense lesion is noted on T1-weighted images, while an isointense to slightly hyperintense mass appears on T2-weighted images [33]. The scar typically shows high signal intensity on T2-weighted images due to vessels or edema in the scar ([34]. Gadolinium infusion produces rapid enhancement of the FNH mass due to its arterial blood supply,

producing a hyperintense lesion on early films . On delayed images it becomes more isointense with respect to normal liver. The central scar enhances on delayed imaging as contrast gradually diffuses into the fibrous center of the mass [35-38]. In one study, gadolinium enhanced MRI had a sensitivity and specificity of 70 and 98 percent, respectively [23].A relatively new MR contrast agent has been introduced into clinical use. Unlike currently used gadolinium-based contrast agents for MRI, this agent, a Gd-BOPTA chelate of Godobenate Dimeglumine, has a dual route of elimination, through both renal and hepatobiliary excretion . Thus, it can be useful for distinguishing hepatic adenomas from focal nodular hyperplasia. AngiographyAlthough angiography may reveal the diagnostic "spoked wheel" appearance of FNH, its use is rarely indicated [27,30,31].

ROLE OF ORAL CONTRACEPTIVESFNH was first described in the early 1900s, long before the advent of oral contraceptives (OCPs).

It is seen in men and children who do not use OCPs and its incidence remained steady after the introduction of OCPs in 1960, in sharp contrast to the dramatic rise in the incidence of HA with the widespread use of OCPs. Thus use of OCPs is not required for the development of FNH [39-41].

On the other hand, FNH may be responsive to estrogens [10]. Patients taking OCPs tend to have larger, more vascular tumors, have more symptoms, and reports of hemorrhage or rupture in patients with FNH have all occurred in patients taking OCPs [42-45]. In our patient ,the increase in number of the lesions may be explained by the growth spurt and the hormonal influences.

MANAGEMENTThe natural history of FNH is one of stability and lack of complications. Lesions generally do not change over time, although they occasionally become smaller [46,48-51]. However, as mentioned above, enlargement of FNH in the setting of OCPs and during pregnancy have been reported [52]. There is no evidence for malignant transformation of FNH [12,23,53,54

].In our patient the occurrence durind the growth spurt may explain the occurrence of lesions after resection.

Patients who are suspected of having FNH based upon the evaluation described above should be managed conservatively [23,34,46,48,49,51,55,56]. If a diagnosis remains unclear, a liver biopsy may be helpful, but may also be misleading since only resection will be definitive [57]. Follow-up studies at three and six months will often be sufficient to confirm the stability of the lesion and its benign nature, after which no long-term follow-up is required routinely. Surgery should be reserved for the rare, very symptomatic FNH lesion, and the highly suspicious lesion, which has eluded diagnosis by all other modalities.

Conclusion:

Ayoung 16 years old female with multiple FNH that recurred after major liver resection showed the problems of dealing with focal liver lesions in children.

Difficulties may arise in interpretations of imaging &histopathologic samples.

The reason for the rapid recurrence was probably because of hormonal influences and the growth spurt.

Focal liver lesions need a b team approach to minimize the difficulties in interpretations.

A second opinion in both radiology and histopathology can be of great importance.

Competing interests

The author declare that he has no competing interests.

Authors' contributions

This patient was investigated in many hospitals including UAE- and the operation was done in Lebanon.

Acknowledgements

The Author thanks the patient and her family involved for permitting the publication of the case report.

References:

1. John, TG, Greig, JD, Crosbie, JL, et al. Superior Staging of Liver Tumors with Laparoscopy and Laparoscopic Ultrasound. Ann Surg 1994; 220:711.

2. Wanless, IR, Mawdsley, C, Adams, R. On the Pathogenesis of Focal Nodular Hyperplasia of the Liver. Hepatology 1985; 5:1194.

3. Reymond, D, Plaschkes, J, Luthy, AR, et al. Focal nodular hyperplasia of the liver in children: Review of follow-up and outcome. J Pediatr Surg 1995; 30:1590.

4. Terminology of nodular hepatocellular lesions. International Working Party. Hepatology 1995; 22:983.

5. Paradis, V, Laurent, A, Flejou, JF, et al. Evidence for the polyclonal nature of focal nodular hyperplasia of the liver by the study of X-chromosome inactivation. Hepatology 1997; 26:891.

6. Gaffey, MJ, Iezzoni, JC, Weiss, LM. Clonal analysis of focal nodular hyperplasia of the liver. Am J Pathol 1996; 148:1089.

7. Fukukura, Y, Nakashima, O, Kusaba, A, et al. Angioarchitecture and blood circulation in focal nodular hyperplasia of the liver. J Hepatol 1998; 29:470.

8. Kondo, F, Nagao, T, Sato, T, et al. Etiological analysis of focal nodular hyperplasia of the liver, with emphasis on similar abnormal vasculatures to nodular regenerative hyperplasia and idiopathic portal hypertension. Pathol Res Pract 1998; 194:487.

9. Nakanuma, Y. Non-neoplastic nodular lesions in the liver. Pathol Int 1995; 45:703.

10. Wanless, IR, Gryfe, A. Nodular transformation of the liver in hereditary hemorrhagic telangiectasia. Arch Pathol Lab Med 1986; 110:331.

11. Nguyen, BN, Flejou, JF, Terris, B, et al. Focal nodular hyperplasia of the liver: A comprehensive pathologic study of 305 lesions and recognition of new histologic forms. Am J Surg Pathol 1999; 23:1441.

12. Ishak, KG, Rabin, L. Benign Tumors of the Liver. Med Clin North Am 1975; 59:995.

13. Mathieu, D, Zafrani, ES, Anglade, MC, Dhumeaux, D. Association of focal nodular hyperplasia and hepatic hemangioma. Gastroenterology 1989; 97:154.

14. Haber, M, Reuben, A, Burrell, M, et al. Multiple focal nodular hyperplasia of the liver associated with hemihypertrophy and vascular malformations. Gastroenterology 1995; 108:1256.

15. Mindikoglu, AL, Regev, A, Levi, JU, et al. Focal nodular hyperplasia in identical twins. Am J Gastroenterol 2005; 100:1616.

16. Goodman, ZD. Benign Tumors of the Liver. In: Okuda, K, Ishak, KD (Eds), Neoplasms of the Liver, Springer Tokyo 1987. P.105.

17. Klatskin, G, Conn, H. Neoplasms of the Liver and Intrahepatic Bile Ducts In Histopathology of the Liver. Oxford University Press, New York 1993. P.367.

18. Wanless, IR, Albrecht, S, Bilbao, J, et al. Multiple focal nodular hyperplasia of the liver associated with vascular malformations of various organs and neoplasia of the brain: A new syndrome. Mod Pathol 1989; 2:456.

19. Bioulac-Sage, P, Rebouissou, S, Sa Cunha, A, et al. Clinical, morphologic, and molecular features defining so-called telangiectatic focal nodular hyperplasias of the liver. Gastroenterology 2005; 128:1211.

20. Brunt, EM, Flye, MW. Infarction in focal nodular hyperplasia of the liver. A case report. Am J Clin Pathol 1991; 95:503.

21. Lee, MJ, Saini, S, Hamm, B, et al. Focal nodular hyperplasia of the liver: MR findings in 35 proved cases. AJR Am J Roentgenol 1991; 156:317.

22. Cherqui, D, Rahmouni, A, Charlotte, F, et al. Management of focal nodular hyperplasia and hepatocellular adenoma in young women: A series of 41 patients with clinical, radiological, and pathological correlations. Hepatology 1995; 22:1674.

23. Shamsi, K, De Schepper, A, Degryse, H, Deckers, F. Focal nodular hyperplasia of the liver: Radiologic findings. Abdom Imaging 1993; 18:32.

24. Bartolozzi, C, Lencioni, R, Paolicchi, A, et al. Differentiation of hepatocellular adenoma and focal nodular hyperplasia of the liver: Comparison of power Doppler imaging and conventional color Doppler sonography. Eur Radiol 1997; 7:1410.

25. Wang, LY, Wang, JH, Lin, ZY, et al. Hepatic focal nodular hyperplasia: Findings on color Doppler ultrasound. Abdom Imaging 1997; 22:178.

26. Mergo, PJ, Ros, PR. Benign Lesions of the Liver In The Radiologic Clinics of North America 36(2). W.B. Saunders, Philadelphia 1998 p.319.

27. Carlson, SK, Johnson, CD, Bender, CE, Welch, TJ. CT of focal nodular hyperplasia of the liver. AJR Am J Roentgenol 2000; 174:705.

28. Drane, WE, Krasicky, GA, Johnson, DA. Radionuclide imaging of primary tumors and tumor-like conditions of the liver. Clin Nucl Med 1987; 12:569.

29. Welch, TJ, Sheedy, PF 2d, Johnson, CM, et al. Focal Nodular Hyperplasia and Hepatic Adenoma: Comparison of Angiography, CT, US, and Scintigraphy. Radiology 1985; 156:593.

30. Rogers, JV, Mack, LA, Freeny, PC, et al. Hepatic Focal Nodular Hyperplasia: Angiography, CT, Sonography & Scintigraphy. AJR Am J Roentgenol 1981; 137:983.

31. Boulahdour, H, Cherqui, D, Charlotte, F, et al. The hot spot hepatobiliary scan in focal nodular hyperplasia. J Nucl Med 1993; 34:2105.

32. Mattison, GR, Glazer, GM, Quint, LE, et al. MR imaging of hepatic focal nodular hyperplasia: Characterization and distinction from primary malignant hepatic tumors. AJR Am J Roentgenol 1987; 148:711.

33.Buetow, PC, Pantongrag-Brown, L, Buck, JL, et al. Focal nodular hyperplasia of the liver: Radiologic-pathologic correlation [published erratum appears in Radiographics 1997 Mar-Apr;17(2):524]. Radiographics 1996; 16:369.

34.Irie, H, Honda, H, Kaneko, K, et al. MR imaging of focal nodular hyperplasia of the liver: Value of contrast-enhanced dynamic study. Radiat Med 1997; 15:29.

35.Mahfouz, AE, Hamm, B, Taupitz, M, Wolf, KJ. Hypervascular liver lesions: Differentiation of focal nodular hyperplasia from malignant tumors with dynamic gadolinium-enhanced MR imaging. Radiology 1993; 186:133.

36.Rummeny, E, Weissleder, R, Sironi, S, et al. Central scars in primary liver tumors: MR features, specificity, and pathologic correlation. Radiology 1989; 171:323.

37.Mathieu, D, Rahmouni, A, Anglade, MC, et al. Focal nodular hyperplasia of the liver: Assessment with contrast-enhanced TurboFLASH MR imaging. Radiology 1991; 180:25.

38.Fechner, RE. Benign hepatic lesions and orally administered contraceptives. A report of seven cases and a critical analysis of the literature. Hum Pathol 1977; 8:255.

39.Ishak, KG. Hepatic Neoplasms Associated with Contraceptive and Anabolic Steroids in Carcinogenic Hormones. In: Lingeman CH ed. Recent Results in Cancer Research, New York: Springer-Verlag, 1979; 73.

40.Geders, JM, Haque, S, Tesi, RJ, et al. A young man with a solitary hepatic mass [clinical conference]. Hepatology 1995; 22:655.

41.Nime, F, Pickren, JW, Vana, J, et al. The histology of liver tumors in oral contraceptive users observed during a national survey by the American College of Surgeons Commission on Cancer. Cancer 1979; 44:1481.

42.Aldinger, K, Ben-Menachem, Y, Whalen, G. Focal nodular hyperplasia of the liver associated with high-dosage estrogens. Arch Intern Med 1977; 137:357.

43.Klatskin, G. Hepatic Tumors: Possible Relationship to Use of Oral Contraceptives. Gastroenterology 1977; 73:386.

44.Shortell, CK, Schwartz, SI. Hepatic adenoma and focal nodular hyperplasia. Surg Gynecol Obstet 1991; 173:426.

45.Mathieu, D, Kobeiter, H, Maison, P, et al. Oral Contraceptive Use and Focal Nodular Hyperplasia of the Liver. Gastroenterology 2000; 118:560.

46.Scalori, A, Tavani, A, Gallus, S, et al. Oral contraceptives and the risk of focal nodular hyperplasia of the liver: A case-control study. Am J Obstet Gynecol 2002; 186:195.

47.Weimann, A, Ringe, B, Klempnauer, J, et al. Benign Liver Tumors: Differential Diagnosis and Indications for Surgery. World J Surg 1997; 21:983.

48.Heinemann, LA, Weimann, A, Gerken, G, et al. Modern oral contraceptive use and benign liver tumors: The German Benign Liver Tumor Case-Control Study. Eur J Contracept Reprod Health Care 1998; 3:194.

49.Di Stasi, M, Caturelli, E, De Sio, I, et al. Natural history of focal nodular hyperplasia of the liver: an ultrasound study. J Clin Ultrasound 1996; 24:345.

50.Leconte, I, Van Beers, BE, Lacrosse, M, et al. Focal nodular hyperplasia: Natural course observed with CT and MRI. J Comput Assist Tomogr 2000; 24:61.

51.Scott, LD, Katz, AR, Duke, JH, et al. Oral Contraceptives, pregnancy and focal nodular hyperplasia of the Liver. JAMA 1984; 251:1461.

52.Wanless, IR. Nodular regenerative hyperplasia, dysplasia, and hepatocellular carcinoma [editorial; comment]. Am J Gastroenterol 1996; 91:836.

53.Rubin, RA, Mitchell, DG. Evaluation of the Solid Hepatic Mass. Med Clin North Am 1996; 80:907.

54.Belghiti, J, Pateron, D, Panis, Y, et al. Resection of presumed benign liver tumours. Br J Surg 1993; 80:380.

55.De Carlis, L, Pirotta, V, Rondinara, GF, et al. Hepatic adenoma and focal nodular hyperplasia: Diagnosis and criteria for treatment. Liver Transpl Surg 1997; 3:160.

56.Fabre, A, Audet, P, Vilgrain, V, et al. Histologic scoring of liver biopsy in focal nodular hyperplasia with atypical presentation. Hepatology 2002; 35:414.

57.Weimann, A, Mössinger, M, Fronhoff, K, et al. Pregnancy in Women with Observed Focal Nodular Hyperplasia of the Liver. Lancet 1998; 351:1251.

Fig 1 CT scan with contrast showed reported as mild hepatomegaly with perfusion defect altering the right hepatic lobe enhancement with apparent portosystemic shunting, possibilities raised were,Splenic vien thrombosis,storage disease,lymphoma

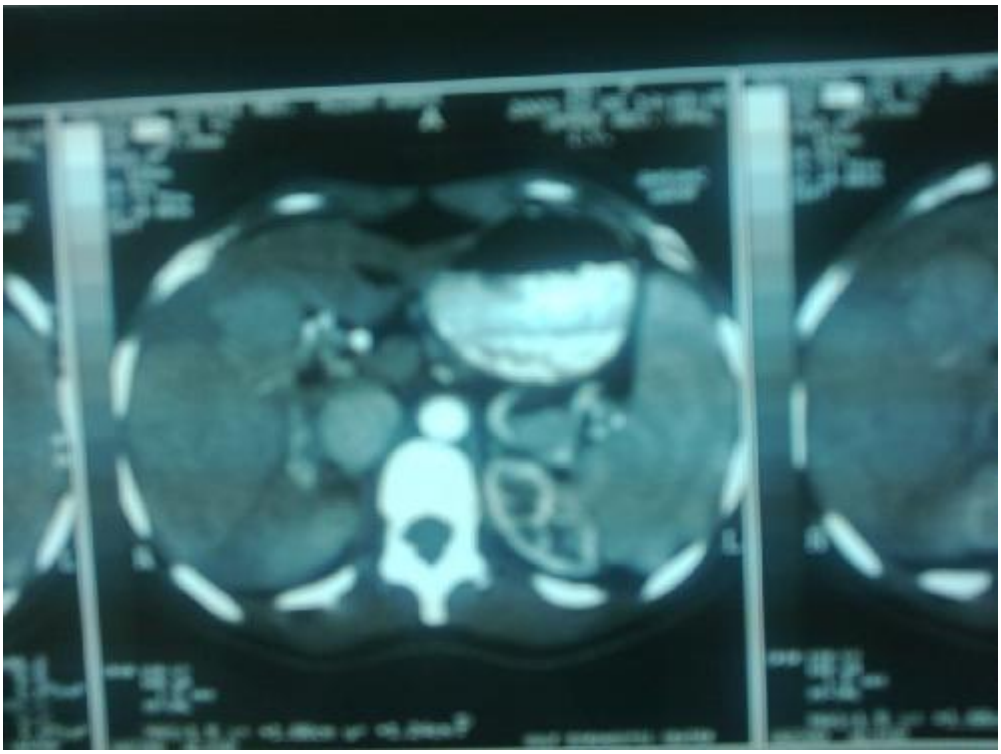


Fig 2 Triphasic Ctscan showed 3 well defined lesions segments 5, 6, 7 occupying the right lobe,caudate and one in the left lobe segment 2, a diagnosis of adenoma was made .

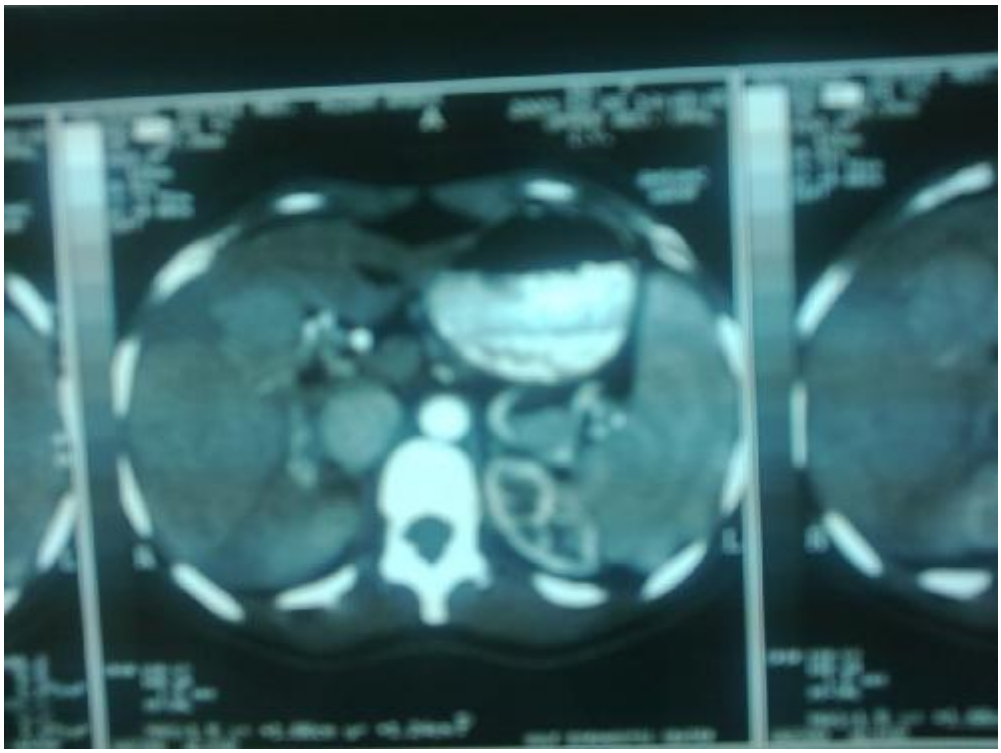


Fig 3 MRI liver showed normal size liver with large well defined mass 10x7 cm,bilobed,well encapsulated with well defined internal septum

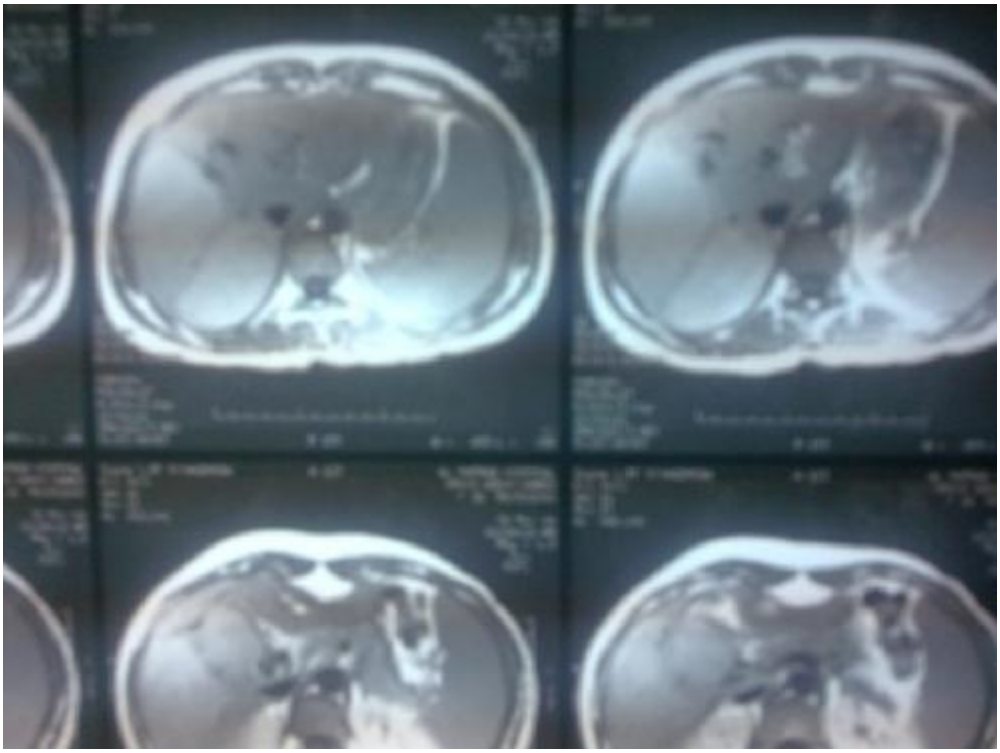
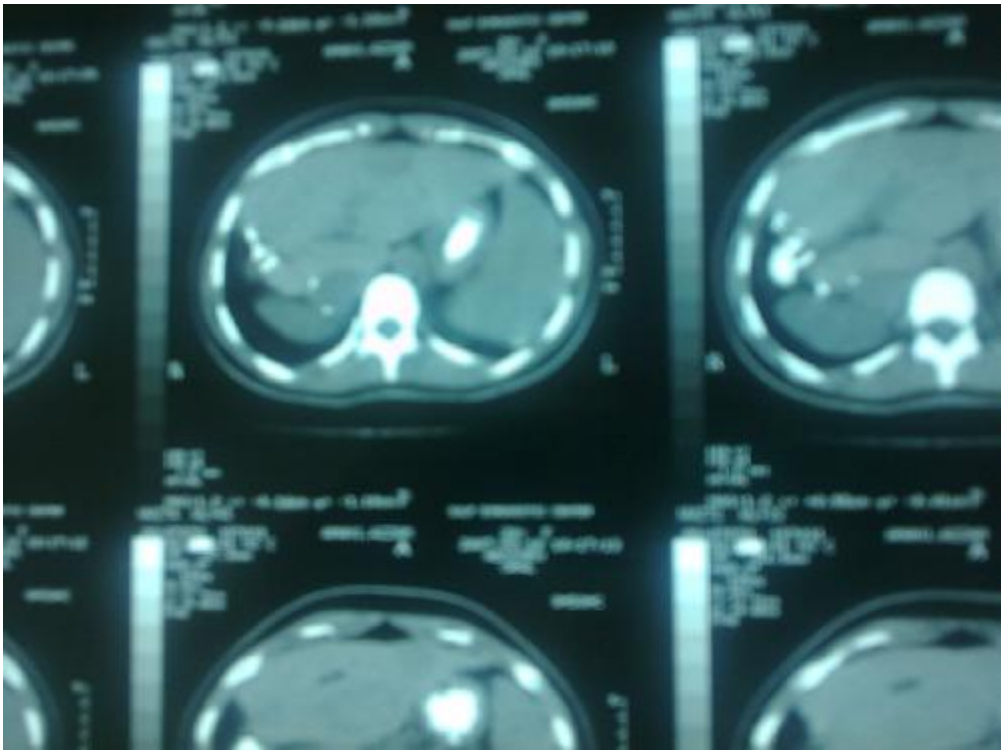


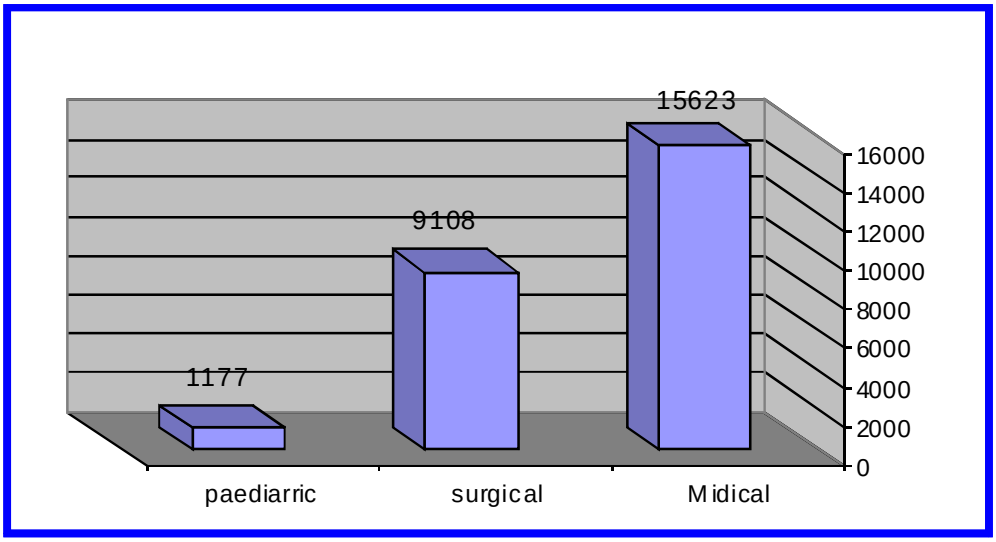
Fig 4-post operative CT scan



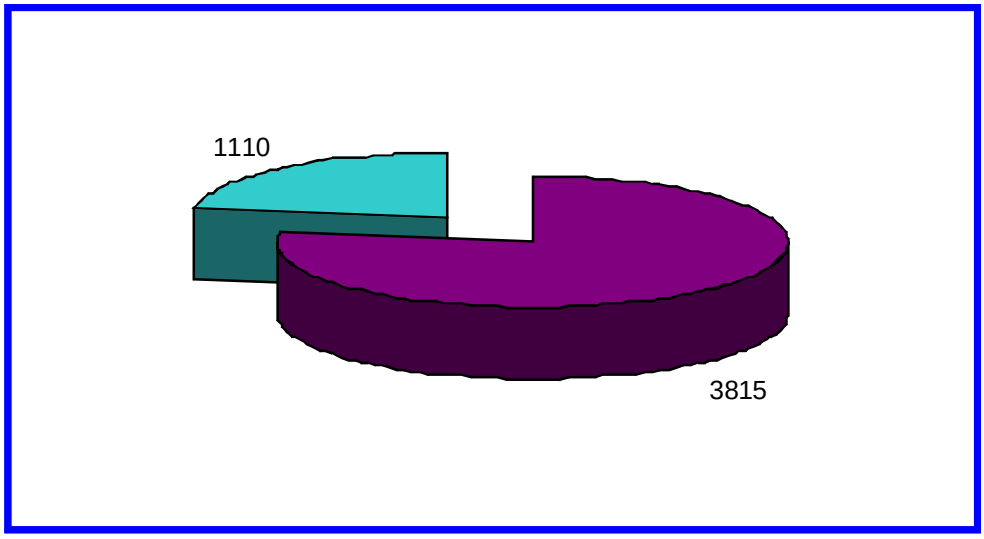
Profile of 2011

***Sana M. Hussein**

No. of attendants of medical & surgical & pediatric clinic 2011		
Paediarric	Surgical	Midical
1177	9108	15623

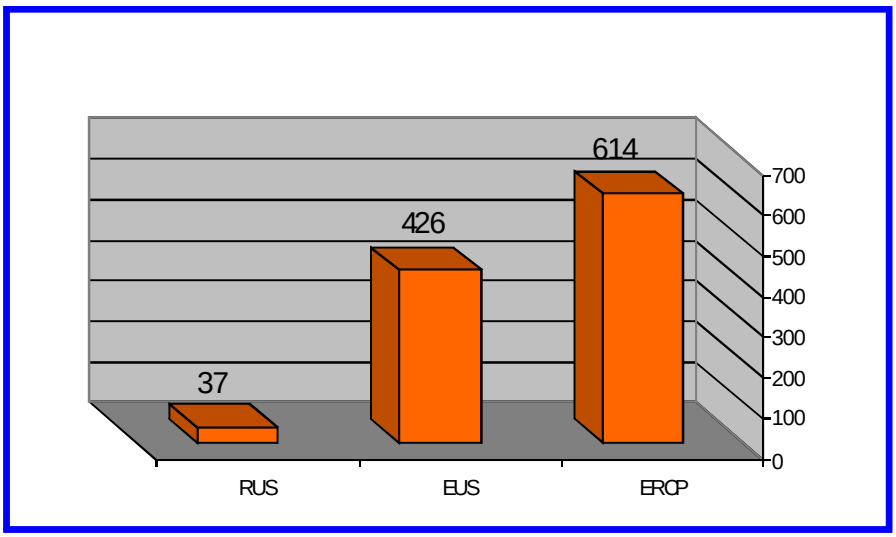


No. of upper & lower endoscopes 2011	
Colonoscopy	Endoscopy (OGD)
1110	3815

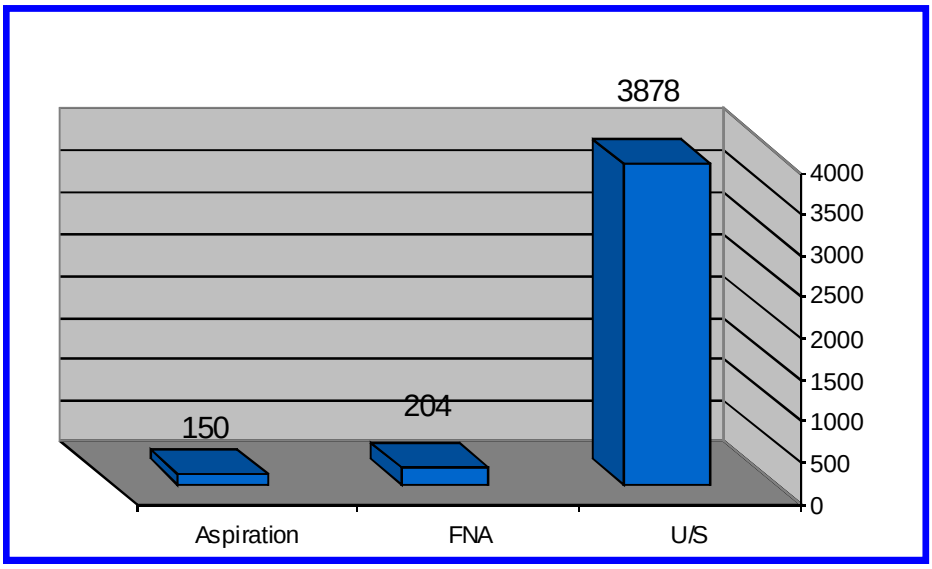


* Statistical Department

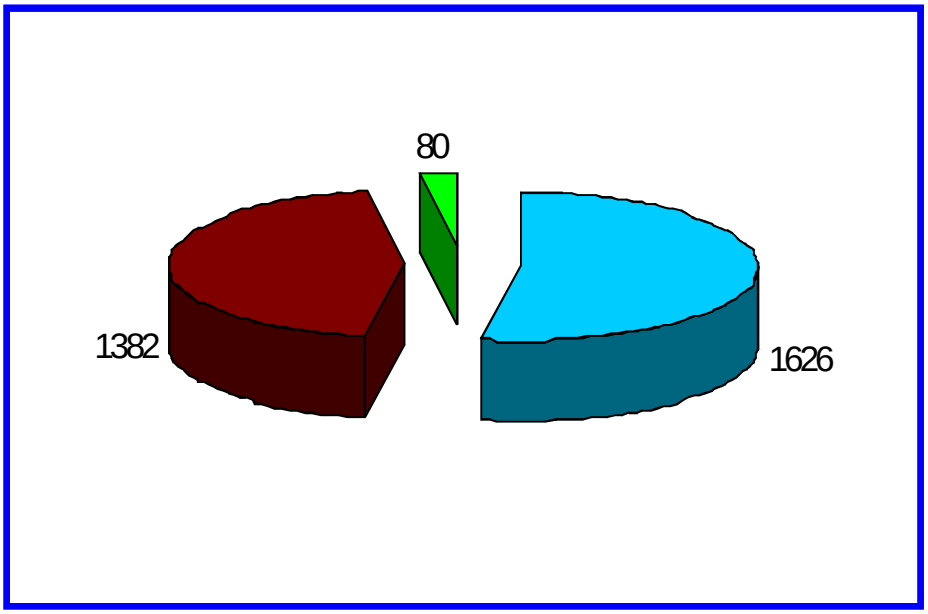
No. of Patients under went RUS,EUS & ERCP 2011		
RUS	EUS	ERCP
37	426	614



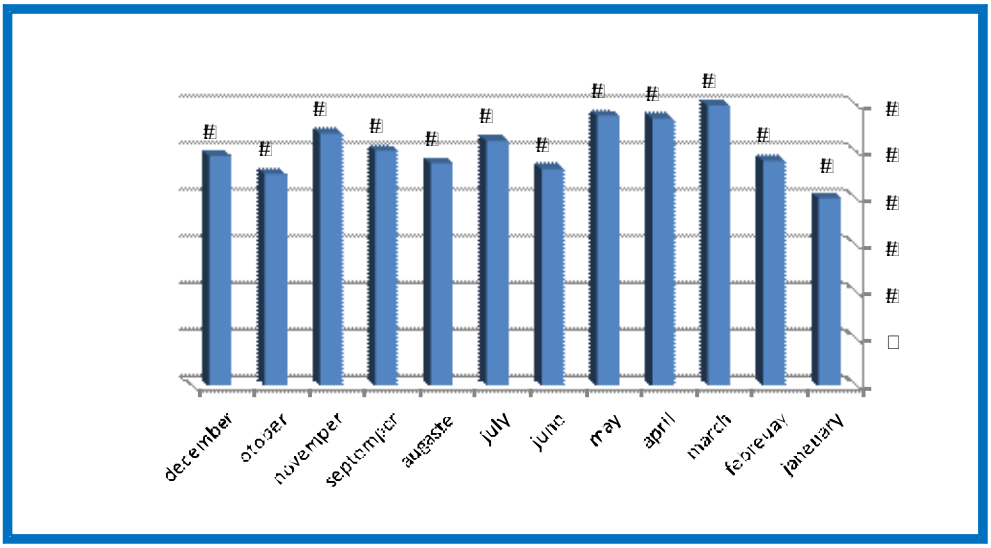
No. of Patient under went abd. U/S, FNA & Aspiration 2011		
Asperation	FNA	Ultra sound
150	204	3878



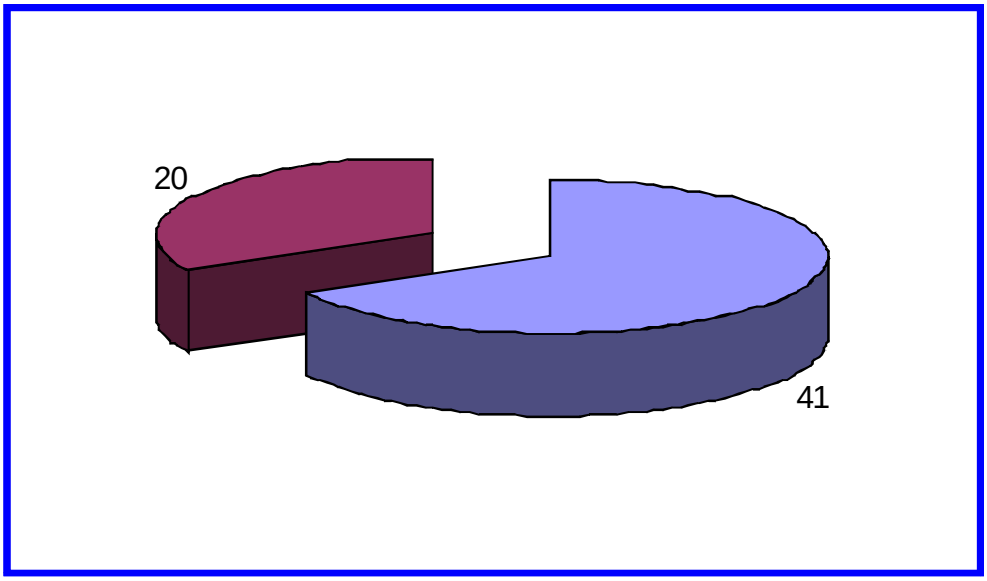
No. of addimision medical & surgical & ICU 2011		
ICU	Surgical	Medical
80	1382	1626



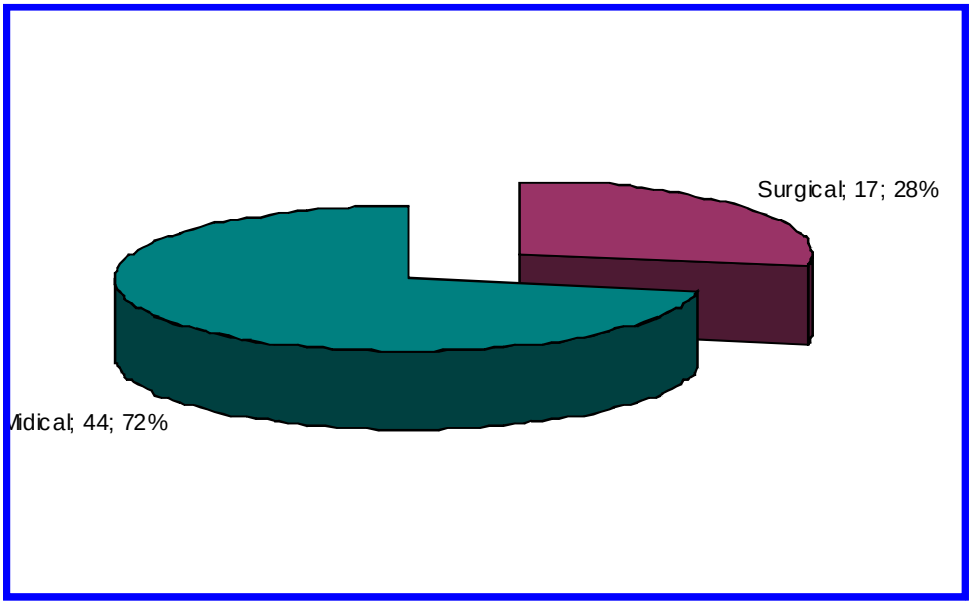
No. of Inpatient 2011



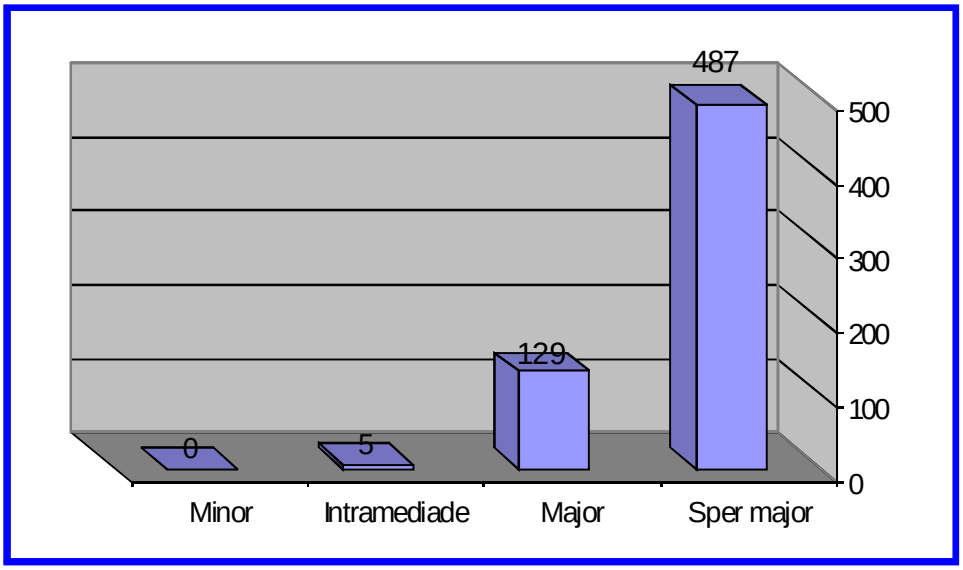
No. of Mortaliaty rate of male & female patient 2011	
Female	Male
20	41



No. of Mortaliaty rate of Midical & Surgical patient 2011	
Midical	Surgical
44	17



No. of Super Major ,Major ,Interameddiade & minor operation 2011			
minor	Interameddiade	Major	Super Major
0	5	129	487



No. of ECG & X - Ray 2011	
ECG	X - Ray
819	3088