# **Clinical Profile of Liver Abscess**

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Abstract: Liver abscess is fairly common in developing countries like India due to inadequate sanitation, overcrowding and poor nutrition. yet, there is limited data on the clinical profile and presentation at the medical wards. The liver is the common site of abscess of all the visceral abscesses which may arise from hematogenous spread of bacteria or from local spread from contagious sites like ruptured appendix and pyelonephritis [1]. Liver abscess can be of amebic and pyogenic liver abscess. Liver abscess presents diagnostic and therapeautic challenges to the physicians and if left untreated, these lesions are invariably fatal [2] and the mortality rate still remained at 60-80% [3], despite the more aggressive approach to treatment. So to reduce the morbidity and mortality, early diagnosis, early intiation of treatment is important. Hence an attempt has been made to study the various clinical manifestations, biochemical changes, radiological features, sonographic changes and complications of liver abscesses in 50 patients who were admitted in the medical wards of Government General Hospital, Guntur. The results showed that more males are affected than females and amebic liver abscess is commoner than pyogenic liver abscess and fever, jaundice, mass in abdomen are the commonest features with ultrasonogram is the most dependable, noninvasive, economical investigation used in diagnosis.

Keywords: Amebic liver abscess, pyogenic liver abscess, jaundice, ultrasonogram,

#### I. Introduction

In the present days of rapidly developing modern medicine, with the advent of many investigative procedures in diagnosis of abdominal diseases, the liver is still the main focus of attention for many abdominal problems. In the abdominal infections and intra abdominal abscesses, the main attention of the physician also should be the liver as the liver is the organ most subject to the development of abscess as it is 48% of all the visceral abscesses. Liver abscess may arise from hematogenous spread of bacteria or from local spread from contagious sites like ruptured appendix and pyelonephritis [1]. Liver abscess is broadly divided into amebic Liver abscess(ALA) and pyogenic liver abscess(PLA). It also presents diagnostic and therapeautic challenges to the physicians and if left untreated, these lesions are invariably fatal<sup>[2]</sup>.

In a developing country like India, many bacterial and parasitic infections cause liver abscess, due to inadequate sanitation, overcrowding and poor nutrition. Bacterial abscess of the liver is relatively rare. It has been described since the time of Hippocrates (400 BC), with the first published review by Bright appearing in 1936. In 1938, Ochsner's classic review heralded surgical drainage as the definitive therapy; however, despite the more aggressive approach to treatment, the mortality rate remained at 60-80%. [3]

At this juncture, high index of suspicion is required for diagnosing all liver abscesses. With the advent of non invasive diagnostic measures like ultrasonography, and CT scan, early diagnosis and effective management of liver abscess has become possible. The main aim of management of liver abscess should be to reduce the hospital stay and its complications, thereby reducing the morbidity and mortality.

Hence an attempt has been made to study the various clinical manifestations, biochemical changes, radiological features, sonographic changes and complications of liver abscesses in those patients who were admitted in the medical wards of Government General Hospital, Guntur.

### II. Materials And Methods

This study comprises 50 cases of liver abscess admitted in the medical wards of Government General Hospital, Guntur. The diagnosis of liver abscess was made according to the World health organization (WHO) criteria, 1969.

- 1. Enlarged and tender liver.
- 2. Hematological findings of leucocytosis and raised ESR.
- 3. Radiological findings of elevated right dome of diaphragm.
- 4. Ultrasonographic evidence of abscess in liver.
- 5. Aspiration of anchovy sauce pus from liver in amebic liver abscess and yellow coloured pus from pyogenic liver abscess.
- 6. Demonstration of trophozoites in the aspirated pus in amebic liver abscess, culture and sensitivity positive for pyogenic liver abscess.

- 7. Altered liver function tests(LFT) mainly increased serum bilirubin and transaminases and alkaline phosphatase levels.
- 8. Response to specific antimicrobial, antiamebic and antifungal therapy.

A detailed history was taken, a thorough physical examination was done and noted in separate case sheets. All cases were subjected to various laboratory tests, LFT, radiological and USG examination.

- 2.1. The fever is noted with thermometer [ DOCTOR company].
- 2.2 ESR, Serum bilirubin, SGOT, SGPT, Alkaline phosphatase were done at Department of biochemistry, Guntur medical college, Guntur. The ESR estimation is done with westergreen method, Serum bilirubin withDiazo method (manual), SGOT with kinetic method and SGPT with kinetic method (rapid kit).
- 2.3. Blood culture, pus culture and stool examination for cysts and trophozoites were done at Department of microbiology, Guntur medical college, guntur.
- 2.4. Total leucocyte count was done at Department of pathology, Guntur medical college, guntur.
- 2.5. Radiographs and Ultrasonography were done by experienced radiologist at Government General Hospital, Guntur.

Normal ranges of parameters:-

2.6. Total leucocyte count: - 7000-11000 cells/cumm.

2.7. Serum bilirubin :- <1mg. 2.8. SGOT :- 5 - 45 i.u. 2.9. SGPT :- 5 - 35 i.u.

2.10. Alkaline phosphatase: - 40 - 100 i.u./litre

The chi-square test is applied for all parameters with degree of freedom (df) of '1' and 'P' value as 0.05 (p = 0.05) and the level of significance as x2 > 3.84.

Limitations of study:-The no. of cases were not sufficient to draw conclusions as the phenomenon of iceberg exists. The lack of serology is also limiting the study. The fungal liver abscess were not present in the study due to the sample size is less.

### **Case Sheet Proforma**

Name: Age/Sex: Address: IP No.:

Occupation: Socioeconomic status:

DOA: DOD:

Complaints / Duration:

present/absent 1. Fever 2. Pain abdomen/distension present/absent 3. Nausea and vomitting present/absent 4. Yellow discoloration sclera present/absent 5. Dysentry present/absent 6. Cough with expectoration present/absent 7. Right lower chest pain present/absent 8. Breathlessness present/absent

9. Others: -Mass per abdomen

-Referred pain to right shoulder -Loss of weight and appetite

-Altered sensation

### Past history:-

Similar complaints in past present/absent
 H/o recurrent diarrhoea/dysentry present/absent
 H/o DM/HTN/IHD/PT/HIV present/absent

Family history:-

Similar complaints in the family present/absent

### Personal history:-

- 1. Alcohol :-Type of alcohol/ amount/duration
- 2. Diet:- Veg/non veg
- 3. Smoker
- 4. Drug abuse-IV

General exasmination:

1.built & nourishment2. Anemia3.jaundice4.clubbing5.cyanosis5.edema of feet

Vital data:-

1.pulse rate 2.blood pressure 3.respiratory rate 4.temperature

### **Systemic Examination:-**

Abdomen:-Inspection:-

Shape of the abdomen: 
 Position of umbilicus
 Moments of the abdominal wall
 Pulsations/peristalsis
 Dilated veins over abdomen
 Swelling in the abdomen

 Hernial orifices
 Normal/distended

 Normal/diminished
 present/absent
 present/absent

 normal/full

### Palpation:-

1.Tenderness/rigidity/guarding present/absent

In the right hypochondriam

Hepatomegaly-tender/nontender present/absent
 splenomegaly present/absent
 right lower intercostal tenderness present/absent

#### Percussion:-

1.extent of liver dullness in cms.
2.Free fluid present/absent

#### Auscultation:-

Bowel sounds present/absent
 Bruit/venous hum./hepatic rub present/absent

# Respiratory system:-

| 1) | Respiratory moments | normal/diminished           | right/left |
|----|---------------------|-----------------------------|------------|
| 2) | Percussion          | resonant/dull               | right/left |
| 3) | Brath sounds        | normal/diminished           | right/left |
| 4) | Abnormasl sounds    | crackles/wheeze/pleural rub | right/left |

### Cardiovascular system:-

| 1) | JVP                                                                     | normal/elevated |
|----|-------------------------------------------------------------------------|-----------------|
| 2) | Heart soundsS <sub>1</sub> S <sub>2</sub> S <sub>3</sub> S <sub>4</sub> | present/absent  |
| 3) | Heart borders on percussion                                             | normal/widened  |
| 4) | Murmurs/pericardial rub                                                 | present/absent  |

### Central nervous system:-

1) Level of conciousness concious/ unconcious

2) Size of pupil in mm.3) Signs of meningeal irritation present/absent

4) Plantar reflex flexor/extensor

# Investigations:-

1) Blood -TC,DC,ESR,Hb%

2) Urine -albumin, sugar, microscopy

3) Stool - bile salts/pigments,urobilinogen.pus cells

4) Blood sugar - blood urea

5) LFT -s.bilirubin(direct,indirect),SGOT,SGPT,Alk.phosphatase,

-s.proteins, Albumin, Globulin, A/G ratio

### Radiological examination:-

a) Xray chest P/A view :- raised dome of diaphragm,

pleuropulmonary complications

- b) Xray chest lateral view:- bulge of diaphragm
- c) Ultrasonography -

1.site of the abscess
3.No. of abscess
4. Amount of fluid

d) CT scan:-

1.site of the abscess 2.size of the abscess 3.No. of abscess 4. Amount of fluid

### Response to treatment:-

- 1) Drug therapy
- 2) Aspiration of pus colour, quantity, no. of times of aspiration

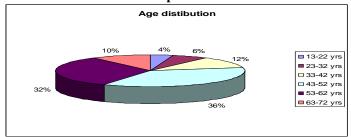
Recovery of trophozoites from the pus Culture of the pus for microorganisms.

- 3)duration of hospital stay
- 4) outcome
- 5) follow up.

### III. Results

### Distribution of cases according to age:

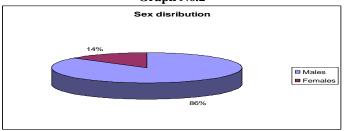




Fifty patients were enrolled into the study. Out of 50 patients,  $4\%^{(n=2)}$  were belonged to 13-22 years,  $6\%^{(n=3)}$  were belonged to 23-32years,  $12\%^{(n=6)}$  belonged to 33-42 years,  $36\%^{(n=18)}$  were belonged to 43-52 years,  $32\%^{(n=16)}$  were belonged to 53-62 years and  $10\%^{(n=5)}$  belonged to 63-72 years. Maximum number of patients i.e.  $36\%^{(n=18)}$  and  $32\%^{(n=16)}$  belonged to 44-53 years and 54-63 years respectively.

# Distribution of cases according to gender:

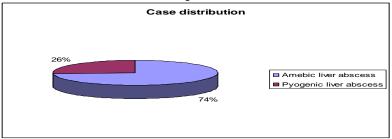
#### Graph No.2



Out of 50 patients,  $86\%^{(n=43)}$  were males and  $14\%^{(n=7)}$  were females. So more no. of males were affected with liver abscess.

### Distribution of cases by type of abscess:

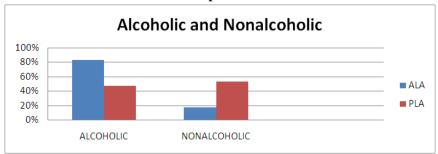
### **Graph No.3**



Out of 50 patients, 74% <sup>(n=37)</sup> suffered from amebic liver abscess(ALA) and 23% <sup>(n=13)</sup> sufferd from pyogenic liver abscess(PLA) and in this study no fungal liver abscess was present.

### Distribution of cases by Alcoholism:

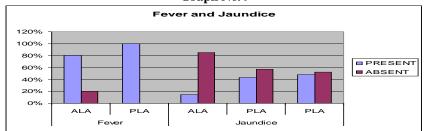
**Graph No.4** 



Out of  $74\%^{(n=37)}$  of ALA cases,  $83\%^{(n=31)}$  were alcoholics and  $17\%^{(n=6)}$  were non alcoholics and out of  $26\%^{(n=13)}$  of PLA cases,  $47\%^{(n=6)}$  were alcoholics and  $53\%^{(n=7)}$  were non alcoholics. Significantly more no. of patients who were alcoholics suffered from amebic liver abscess.

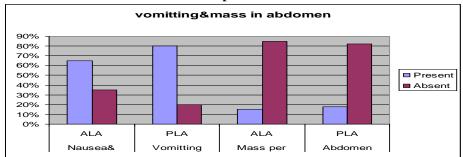
#### Clinical symptoms:-

**Graph No.4** 



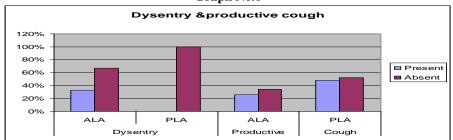
out of 37 ALA patients,  $80\%^{(n=29)}$  had fever,  $15\%^{(n=60)}$  had jaundice, Out of the 13 PLA patients ,  $100\%^{(n=13)}$  had fever-,  $57\%^{(n=7)}$  had jaundice,

Graph No 5



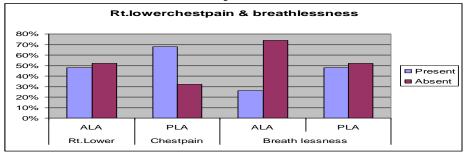
Out of the 37 ALA patients  $65\%^{(n=24)}$  had nausea and vomitting,  $16\%^{(n=6)}$  had mass per abdomen, Out of the 13 PLA patients ,  $80\%^{(n=10)}$  had nausea and vomitting,  $18\%^{(n=2)}$  had mass per abdomen

### Graph No.6



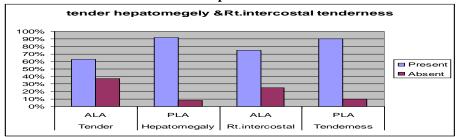
Out of the 37 ALA patients  $33\%^{(n=12)}$  had dysentry,  $26\%^{(n=9)}$  hadproductive cough, Out of the 13 PLA patients , none had dysentry,  $48\%^{(n=6)}$  had productive cough

Graph No.7



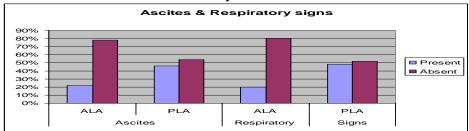
Out of the 37 ALA patients ,  $48\%^{(n=18)}$  had right lower chest pain,  $26\%^{(n=9)}$  had breathlessness and Out of the 13 PLA patients ,  $68\%^{(n=9)}$  had right lower chest pain, and  $48\%^{(n=6)}$  had breathlessness. Significantly more no. of patients with PLA had jaundice(57%)<sup>(n=7)</sup>, more no. of patients with ALA had dysentry(33%)<sup>(n=12)</sup> and other clinical symptoms were statistically insignificant.

**Graph NO.8** 



Regarding clinical signs,  $63\%^{(n=23)}$  of ALA patients had tender hepatmegaly, $75\%^{(n=28)}$  had right intercostal tenderness, and  $92\%^{(n=12)}$  of PLA patients had tender hepatmegaly, $90\%^{(n=11)}$  had right intercostal tenderness,

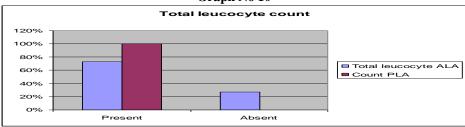
**Graph No.9** 



out of 37 ALA patients ,  $22\%^{(n=8)}$  had ascites,  $20\%^{(n=7)}$  had respiratory signs, out of 13 PLA patients  $46\%^{(n=6)}$  had ascites,  $48\%^{(n=6)}$  had respiratory signs. Significantly more no. of PLA patients had respiratory signs and others were statistically insignificant.

### Laboratory investigations:-

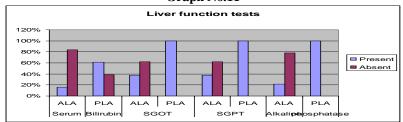
### Graph No 10



In laboratory investigations, 73%<sup>(n=24)</sup> of ALA had raised TLC, and among PLA patients 100%<sup>(n=13)</sup> had raised TLC,

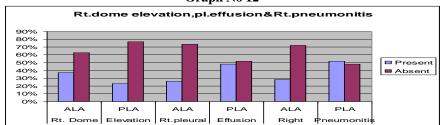
#### Liver functional tests:-

#### **Graph No.11**



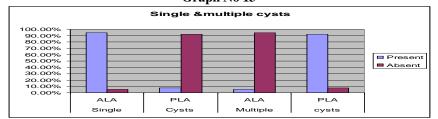
Out of 37 ALA cases,16% <sup>(n=6)</sup> had raised s.bilirubin, 37.8% <sup>(n=14)</sup> had raised SGOT, 37.8% <sup>(n=14)</sup> had raised SGPT, 21.6% <sup>(n=8)</sup> had raised alkaline phaosphatase and out of 13 PLA cases,61.5% <sup>(n=8)</sup> had raised s.bilirubin, 100% <sup>(n=13)</sup> had raised SGOT, 100% <sup>(n=13)</sup> had raised SGPT, 100% <sup>(n=13)</sup> had raised alkaline phaosphatase. X-Ray and USG findings:-

Graph No 12



In the X-ray and USG findings, among 37 ALA patients,  $37\%^{(n=14)}$  had Right dome elevation,  $26\%^{(n=9)}$  had right pleural effusion,  $28\%^{(n=10)}$  had right pneumonitis and Among 13 PLA patients,  $23\%^{(n=3)}$  had Right dome elevation,  $48\%^{(n=6)}$  had right pleural effusion,  $56\%^{(n=7)}$  had right pneumonitis.

Graph No 13



In the X-ray and USG findings, among 37 ALA patients, 94.5% (n=35) had single cysts and 5.5% (n=02) had multiple cysts. Among 13 PLA patients, 7.7% (n=01) had single cysts 92.3% (n=12) had multiple cysts. Significantly more no. of PLA patients had raised TLC, more no. of PLA patients had raised s.bilirubin, more no. of PLA patients had raised SGOT, more no. of PLA patients had raised SGPT, more no. of PLA patients had raised alkline phoshatase. Significantly more no. of ALA had right dome elevation, more no. of PLA

hadright pleural effusion and more no. of PLA had right pneumon Also significantly more no. of ALA patients had single cysts and more no. of PLA patients had multiple cysts.

# **Some figures:**

Case:2-Two abscesses in rt.lobe -8×6cm,4×5cm

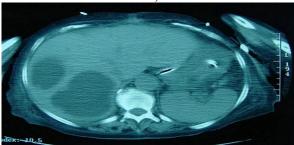


Figure no.01

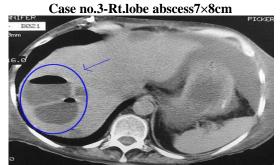


Figure no.02

Case no.4- amebic liver abscess10×12cm



Figure no.03

Case no.7-Multiple liver abscess8×12cm,4×6cm



Figure no.04

Caseno.11 Right lobe amebic liver abscess,10×12cm

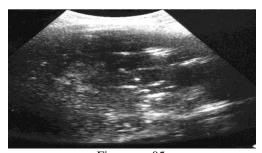


Figure no.05
Caseno.47-pyogenic liver abscess



Figure no.06

Case no.18-pyogenic liver abscess8×6cm,6×7cm

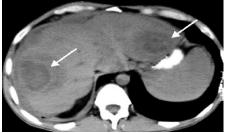


Figure no.07

 $Case\ n\underline{o.24,multiple\ liver\ abscesses,9\times5cms,4\times3cm,4}\times3cm$ 

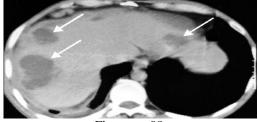


Figure no.08

Case no.28, measuring7×10cm



Figure no.09

Caseno.31, Amebic liver abscess 8×12cm



Figure no.10

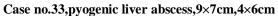




Figure no.11

Case no.34,amebic liver abscess,10×8cms,9×8cm



Figure no.12

Case no.41,pyogenic liver abscess,8×12cm



Figure no.13

Case no.46 Elevated rt.dome of diaphragm due to liver abscess



Figure no.14

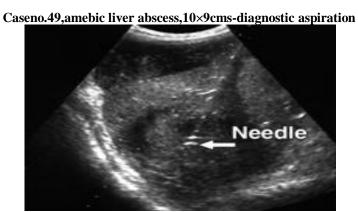
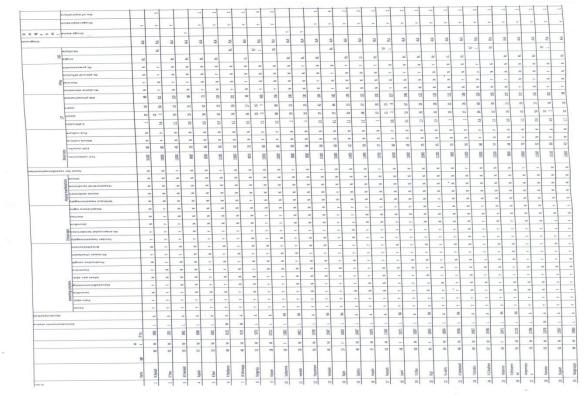
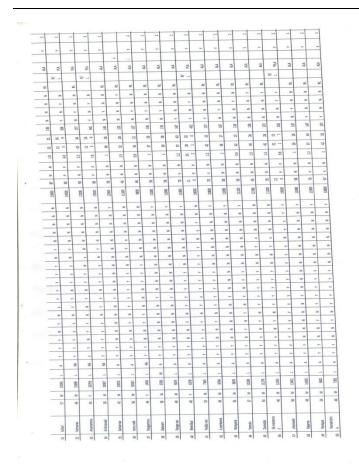


Figure no.15

# **MASTER SHEET OF ALL CASES:**





# IV. Discussion

Liver abscess has been described and treated since hippocrates (400BC) and ochsner (1936) and during those days, mortality rate was very high i.e. upto 70-90% [3]. With the development of new radiologic techniques, improvement in microbiologic identification, advancement of drainage techniques and operative procedures, the mortality rate has come down to 5-30%. Yet the incidence has relatively remained unchanged and with the treatment, the mortality has come down to normal in those patients attending to hospitals. Out of the registered 50 liver abscess cases,  $4\%^{(n=2)}$  cases were admitted with acute abdomen and no deaths occurred during the study. They have been reviewed periodically for any relapses.

Alom siddhique et al<sup>[4]</sup> and Khan et al<sup>[5]</sup> reported peak incidence of age between 21 -50yrs where in this study, peak incidence of age is 44 - 63yrs.

Islam QT et al  $^{[6]}$  and Alom siddhique stated 31% of ALA patients and 83% of PLA patients had association with alcohol consumption whereas in this study,  $83\%^{(n=31)}$  of ALA patients and  $47\%^{(n=6)}$  of PLA had association with alcohol consumption.

Alom siddhique et al<sup>[4]</sup> stated males were predominant 88% than females, whereas this study concurs with above by 86% of males predominant than females.

Alom siddhique et al<sup>[4]</sup> stated presence of fever in 89% and 100%, jaundice in 0% and 8.33%, nausea and vomiting in 39% and 50%, dysentery in 6% and 0%, productive cough in 15% and 0%, lower chest pain in 30% and 66%, breathlessness in 30% and 50% in ALA and PLA respectively.

In this dissertation fever present in  $80\%^{(n=29)}$  and 100%(n=13), jaundice 15% (n=6) and 57%(n=7), nausea and vomiting in 65%(n=24) and 80%(n=10), dysentery in 33%(n=12) and 0%(n=0), productive cough in 26% (n=9) and 48%(n=6), lower chest pain in 48%(n=18) and 68%(n=9), breathlessness in 26%(n=9) and 48%(n=6) in ALA and PLA respectively, which is concurring with the above.

Srivatsava ED et al<sup>[7]</sup> and Alom siddhique et al<sup>[4]</sup> reported tender hepatomegaly in 40% and 89%, ascites in 20% and 55% of ALA and PLA respectively whereas in this discertation, tender hepatomegaly in 63%(n=23) and 92%(n=12), ascites in 22%(n=8) and 46%(n=6) of ALA and PLA respectively.

Alom siddhique et al<sup>[4]</sup>reported amebic cysts in 3% and 0%, trophozoites in 7% and 0% in ALA and PLA respectively and isolated bacteria with E.coli 50%, proteus 25% and pseudomonas 17% in PLA patients. In this dissertation, amebic cysts were found in 25%(n=9) and 0% and trophozoites in 36%(n=13) and 0% in ALA and PLA respectively. The bacterial culture showed E.coli 56%, streptococcal species 37% and proteus

5.5%.Hold Stoch et al  $^{[8]}$  and Alom siddhique et al  $^{[4]}$  reported raise in TLC in 80% and 99%, raised S.bilirubin in 10% and 50%, raise in SGOT in 20% and 90%, raise in SGPT in 25% and 85% and raise in alkaline phosphatase in 30% and 95% in ALA and PLA patients respectively. In this dissertation, raise in TLC in 73%(n=24) and 100%(n=13), raised S.bilirubin in 16%(n=6) and 61.5%(n=8), raise in SGOT in 37.8%(n=14) and 100%(n=13), raise in SGPT in 37.8%(n=14) and 100%(n=13) and raise in alkaline phosphatase in 21.6%(n=8) and 100%(n=13) in ALA and PLA patients respectively.

Mc Donald et al<sup>[9]</sup> and Sherlock S et al<sup>[10]</sup> reported X-ray and USG findings of right dome elevation in 30% and 20%, right pleural effusion in 10% and 30%, single cysts in 80% and 9% and multiple cysts in 5% and 90% of ALA and PLA patients respectively.

Where this study concurs by having right dome elevation in  $37\%^{(n=14)}$  and  $23\%^{(n=3)}$ , right pleural effusion in  $26\%^{(n=9)}$  and  $48\%^{(n=6)}$  right pneumonitis in  $28\%^{(n=10)}$  and  $56\%^{(n=7)}$  single cysts in  $94.5\%^{(n=35)}$  and  $7.7\%^{(n=1)}$  and multiple cysts in  $5.4\%^{(n=20)}$  and  $92.3\%^{(n=12)}$  of ALA and PLA patients respectively. In this study no fungal species were detected.

### V. Summary & Conclusion

This study was conducted at Government General hospital, Guntur on a series of 50 patients. On par with all prior studies the study had similar results in incidence, males had higher disease burden than in women. The fact that why women do not seek medical attention need to be evaluated.

The second most important finding is amebic liver abscess is more common than pyogenic liver abscess. Also contrary to prior studies interestingly, the peak incidence is in the age group of 43-52 years of age.

All patients with amebic liver abscess and pyogenic liver abscess had fever 80% and 100% cases, right lower chest pain in 48% and 68% cases, nausea and vomiting in 65% and 80% cases, productive cough in 26% and 48% cases, jaundice in 15% and 43% cases, breathlessness in 26% and 48% cases, mass per abdomen in 15% and 18% cases, dysentery in 33% and 0% cases respectively.

From this study the following conclusions can be drawn that both amebic liver abscess and pyogenic liver abscess have almost similar clinical features. Most of the cases belong to the low income group. Liver abscess has correlation with consumption of indigenous alcohol. Ultrasonogram is an easy, widely available non-invasive, most economical and dependable investigation to diagnose liver abscess. In the absence of sophisticated investigations (e.g. Serum antibody against amoeba) at hand, aspiration of pus is a good guide to confirm and differentiate ALA (by naked eye, microscopic examination and culture/ sensitivity of pus) from PLA as ALA shows anchovy sauce coloured pus and PLA shows yellow coloured pus. The study also showed Amoebic liver abscess is more common than pyogenic liver abscess in our community. Complications like recurrence, pleuro-peritoneal involvement or rupture of the abscess are also common and if left undiagnosed and untreated, mortality will be high which is highly preventable and treatable. With the availability of very efficacious new drugs, medical treatment is giving good results.

### Acknowledgements

We are very thankful to Dr.Bhaskar rao,HOD and professor of Medicine unit-5 and all the teaching and nonteaching faculty of Medicine unit-5,faculty of radiology department,biochemistry department and pathology department. Our utmost thanks to all our patients whithout which this could not be done

### References

- [1]. Miriiam J.Baron, Baron, Dennis L.Kasper, Anthony S.Fauci; Dan L.Longo ; Eugene Braunwald Et Al "Intraabdominal Infections And Abscess" Harrison 'S Principles Of Intenal Medicine , 17<sup>th</sup> Ed, Pg811.
- [2]. Todd A Nicholas ; Brian Red ; Lamar O Mack ; Mohammed Akoad Et Al
- a. Available From URL Http:// Www.Emedicine.Medscape.Com/Article/188802-Overview.
- [3]. Ruben Peratta; Michelle V Lisgaris: Robert A Salata; Sarah C Langenfeld Et Al
- a. Available From URL Http:// Www.Emedicine.Medscape.Com/Article/188802-Overview
- [4]. M N Alom Siddiqui1, M Abdul Ahad2, A R M Saifuddin Ekram3,Q Tarikui Islam4, M Azizul Hoque5, Q A A L Masum Et Al, Clinico-Pathological Profile Of Liver Abscess In A Teaching Hospital The Journal Of Teachers Association RMC, Rajshahi, June 2008; Volume 21 Number 1
- [5]. Khan M, Akhter A, Mamun AA, Mahmud TAK, Ahmad KU. Amoebic Liver Abscess: Clinical Profile And Therapeutic Response. Bang. J. Med. 1991; 2:32-38.
- [6]. Islam N. The Poor Access To Under Land For Housing. In Urban Land Management In Bangladesh. Ministry Of Land, Government Of Bangladesh. 1992; 131-40.
- [7]. Srivasta ED, Mayberry JF. Pyogenic Liver Abscess. A Review Of Actiology, Diagnosis And Intervention. Dig. Dis. 1990; 8: 287
- [8]. Hold Stock G, Balasegaram M, Millward-Sadlergh, Wright R. The Liver In Infection. In: Liver And Biliary Disease. 2nd Edition. Wright R. Millward-Sadler GYH, Alberti KGMM (Eds) WB Saunders Company. London 1987; 1077-1119
- [9]. Mc Donald MI. Pyogenic Liver Abscess: Diagnosis, Bacteriology And Treatment. Eur. J. Clin. Microbiol. 1984; 3: 500-9.
- [10]. Sherlock S, Dooley J. The Liver In Infection. Insherlock S. Dooley J (Ed) Disease Of The Liver And Biliary System. Blackwell Scientific Publications, London. 11th Edition. 2002; 495-8.

