CASE REPORT

Community Acquired Pyogenic Liver Abscess Caused by A Nosocomial Organism: A Case Report

Shirley N **CHUKWURAH**[1]
Simon N **USHIE**[2]
Felix E **EZENEKWE**[3]
Ejeye O **UGWUNZE**[1]
Chinyelu U **UFOAROH**[1]
Nkechi P **MADUEKWE**[2]

[1]*Department of Medicine*
[2]*Department of Medical Microbiology & Parasitology*
[3]*Department of Radiology*

Nnamdi Azikiwe University/ Nnamdi Azikiwe University Teaching Hospital Nnewi, Anambra State NIGERIA

Author for Correspondence
Dr Shirley N ***CHUKWURAH***
Department of Medicine
Nnamdi Azikiwe University
Teaching Hospital
Nnewi, Anambra State NIGERIA.

Phone: +234 803 700 1715
Email: nasfic2@gmail.com.

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ABSTRACT

Pyogenic liver abscess (PLA) is a potentially life-threatening disease affecting patients in many parts of the world, especially Asia and the other third world countries, Nigeria inclusive. Knowing the aetiology of PLA, where possible, plays a significant role in the successful treatment of the patients. Recently, the prevalence of *Klebsiella pneumonia* induced PLA (KP-PLA) has become an emerging public health challenge all over the world, however, other rare organisms are also implicated as shown in our case report. *Acinetobacter baumannii* is a multi-drug resistant nosocomial organism that may also be responsible for some cases of community acquired infections as seen in this index case.

Key words: *Acinetobacter baumannii, Ciprofloxacin, Hospital acquired infections, Community associated infections, Immune deficiency, Abscess.*

INTRODUCTION

Pyogenic liver abscess (PLA) is a pocket of pus that forms in the liver due to bacterial infection. It accounts for about 80% of liver abscesses with non-pyogenic (fungal and amoebic) liver abscesses accounting for 20%.[2] The introduction of antibiotics and advances in bacteriology and other diagnostic techniques has generally improved the diagnosis and management outcomes. A basic requirement for effective therapy is early diagnosis.[3] New imaging techniques such as ultrasound, CT scan and MRI have made the differential diagnosis easier but

cannot always definitively rule out parasitic abscesses.[4,5] Diagnosis can be missed even intra- operatively.[6]

CASE SUMMARY

A 42-year-old married man and a trader who deals on clothing, presented with fever of 3 weeks' duration and abdominal pain of 2 weeks' duration. The fever was insidious in onset, continuous, high grade and associated with chills and rigor; and was temporarily relieved by intake of acetaminophen. There were associated headache, generalized body weakness and loss of appetite. A week into the illness, he developed abdominal pain which was localized to the right upper quadrant, dull, not radiating, worsened by deep inspiration and has no known relieving factors. There were no changes in bowel habit and no history of abdominal swelling or jaundice. There was no history of cough, neck stiffness, ear pain or discharge or pain on urination.

He has a history of significant alcohol consumption of about 120g of alcohol/week for years and occasionally took water based herbal preparations for febrile illnesses. He patronises food vendors for his meals. He is not a known diabetic nor hypertensive patient. His Human Immuno-deficiency Viral(HIV) status was negative but he was diagnosed of hepatitis B virus infection in the course of this illness. He has not been treated for tuberculosis in the past.

Following the above symptoms, he was admitted in a missionary hospital for 5 days where he was managed for acute viral hepatitis with intravenous fluids, analgesics and antibiotics (names not known). With the persistence of symptoms and the ultrasound finding of a liver abscess, he was referred to the Gastroenterology unit of Nnamdi Azikiwe University Teaching Hospital (NAUTH) Nnewi for further evaluation and management.

On examination he was pale, anxious looking and febrile (37.7^0C). The abdomen was full and moved with respiration, there was right hypochondrial tenderness, liver was 6cm palpable, tender, firm with blunt edge; spleen was not palpable and kidneys were not ballotable. The liver span was 14cm. There was no demonstrable ascites and bowel sounds were normal.

The vital signs were normal and chest examination revealed reduced breath sounds in the right lower lung zone.

An initial diagnosis of poorly treated hepatic abscess in a patient with hepatitis B virus infection was made.

Patient Management and Outcome

Full blood count showed relative neutrophilia and anaemia, Liver function tests revealed mild elevations of liver enzymes with normal serum protein and albumin and PT/INR, kidney function was normal. Abdominal ultrasound revealed a well loculated abscess in the right lobe of the liver with a volume of 204.5cm^3.

HBV DNA load was 8,540 iu/ml and panel test showed HBeAg negative. His laboratory investigations were as shown in Table 1. He had drainage of abscess under ultrasound guidance with 150ml of pus drained. An estimated 50ml could not be drained.

The microscopy, culture and sensitivity (M/C/S) results revealed numerous pus cells, Gram variable coccobacillary organism identified with the help of Microbact 12 A (Oxoid, UK) [showed 99.99%]as *Acinetobacter baumannii* susceptible to Meropenem, Amikacin, Ciprofloxacin, Levofloxacin,

Piperacillin- tazobactam, but was resistant to Gentamycin, Amoxicillin clavulanic acid and Ceftriaxone using Kirby Bauer disc diffusion method.[1] He was subsequently commenced on tabs levofloxacin 500mg twice daily for 2 weeks. He was observed and discharged after 5 days. During his follow up visit, patient had no symptoms, he was gaining weight and repeat abdominal ultrasound showed no more collections. The treatment for chronic hepatitis B was commenced.

Table 1. Results of investigations

Investigations	Results	Reference interval
Full blood count + ESR	ESR= 110mm/hr	
	HB- 7.2g/dl	11-17g/dl
	WBC- 6 X 10^3/UL	4-12 X 10^3/UL
	Neutrophils- 61.8%	50-80%
	Lymphocytes- 33.5%	25-50%
	Monocytes- 4.7%	2-10%
	Platelets- 371 x 10^3	150-400 x 10^3
Alpha fetoprotein	4.9ng/ml	<8.5ng/ml
Total protein	70g/l	62-80g/l
Albumin	39g/l	28-40g/l
Liver Function tests	Total bilirubin- 46.7umol/l	3.4-17umol/l
	Conj. Bilirubin- 36.5umol/l	0.4-3.4umol/l
	AST-22IU/L	5-18IU/L
	ALT- 21IU/L	3-25IU/L
	ALP- 222IU/L	21-92IU/L
Anti- HCV	Negative	
HIV I &II	Negative	
HBSAg	Positive	
E/U/Cr:	Creatinine-77umol/l	76-127umol/l
	Na^+- 137mmol/l	134-145mmol/l
	K^+- 4mmol/l	3.5-5.5mmol/l
	Cl^-98mmol/l	96-106mmol/l
	HCO_3- 27mmol/l	21-31mmol/l
	Urea- 2.5umol/l	1.7-9.1umol/l
PT	13.4secs	11-16secs
INR	0.96	0.8-1.2
Abdominal USS	Cranio-caudal span of the liver was 19cm with a huge multiseptated cystic mass measuring 170mm x 94 mm and an estimated volume of 204.5cm^3 located on segments IV& V of the right lobe of the liver. It showed no colour Doppler on imaging	
HBV DNA viral load	8,540 iu/ml	
Panel test	HBSAg-positive	
	HBSAb- negative	
	HBeAg- negative	
	HBeAb-positive	
	HBcAb- positive	

DISCUSSION

Pyogenic abscess is more common among males. as in the index patient.[7] The patient also presented with the classic triad of fever, malaise and right upper abdominal discomfort common in patients with pyogenic liver abscess. The patient had no obvious risk factor for pyogenic liver abscess and can be classified as cryptogenic.

Pyogenic liver abscess may be poly-microbial.[8] *Escherichia coli* is usually the most common cause followed by *K. pneumonia*. In the index patient, *Acinetobacter baumannii*, a rare cause of PLA was the implicated organism.

This condition is a life-threatening disease if left untreated and data from different sources place the incidence rate at 1.1 to 17.6/1,000,000 individuals.[9] The other most common pathogens associated with PLA in addition to *Escherichia coli* and *Klebsiella pneumoniae*, are *Bacteroides, Enterococci, Streptococci, and Staphylococci* .[10] This patient had PLA caused by *A. baumannii* which is not very common.

Acinetobacter species were considered as low pathogen five decades ago but with the introduction of powerful new antibiotics in clinical practice and agriculture, coupled with the use of invasive procedures in hospital intensive care units (ICUs), drug resistant-related community and hospital-acquired *Acinetobacter* infections have emerged with increasing frequency.[11] the organism is responsible for the following infections within the hospital: bloodstream infections, pneumonia, meningitis, wound and surgical site infections, including necrotizing fasciitis and urinary tract infections especially in catheterized patients.

Among its species, *A. baumannii* has emerged as of a greater clinical importance and is associated with hospital outbreaks. But infections due to other species like *A. lwoffii* have also been reported in hospitals and community settings.[12,13] *Acinetobacter baumannii* is a non-fermentative aerobic Gram-negative or Gram variable bacillus and it is known that 25% of the healthy individuals harbour it as a commensal in the oropharynx and skin. Infection results if the host's first line of immunity is compromised. Due to its ubiquitous nature, it is a potential opportunistic pathogen in individuals with impaired immune systems, and it has been identified as a cause of nosocomial and community acquired infections.

Some rare cases of community acquired infections like pneumonia and bacteraemia caused by *Acinetobacter* species have also been reported. Direct Gram's stain showed gram variable bacteria along with the pus cells. Moreover, *Acinetobacter* species tends to survive in dry conditions and in a wide range of temperature and are resistant to many disinfectants, irradiation and desiccation.[14] These conditions may favour colonization of the stomach by *Acinetobacter spp*. in the hypochlorhydric or achlorhydric stomach.[14]

Weak immune status of the patient from the combined effect of chronic alcoholism and Hepatitis B infection, possibility of poor nutrition as indicated by his petty trading business and virulence potential of the pathogen may have been the factors which led to the spread of the bacteria to the liver from the gut via the portal system and eventually to the formation of liver abscess. Though the cultured strain from the index patient was found to be sensitive to almost all the tested antibiotics, many studies have reported high rates of antibiotic resistance in

Acinetobacter species. Nakwen *et al.* showed good susceptibility to netilmicin, imipenem, cefoperazone/sulbactam, while resistance to amikacin, gentamycin, ceftazidime, ceftriaxone, cefepime, and ciprofloxacin, clindamycin in neonatal septicaemia.[15] Mittal *et al*. reported high resistance to imipenem (57%), cotrimoxazole (57%), gentamycin (82%), piperacillin + tazobactum (61%) in a species of Acinetobacter called *A. iwofii* as compared to other non-baumanii acinetobacter spp in nosocomial infections.[12]

Acinetobacter species has been known to produce a variety of beta–lactamases which confer resistance to aminopenicillins, ureidopenicillins, narrow-spectrum and expanded-spectrum cephalosporin and cephamycin.[16] Partial susceptibility is retained for some relatively new antibiotics such as broad-spectrum cephalosporin (cefotaxime, ceftazidime, and cefepime), tobramycin, imipenem, amikacin, and fluoroquinolones as is noticed in our patient.[16] Since our patient did not recall any previous history of prolonged hospital stay or prolonged antibiotic intake; and the strain was sensitive to most of the antibiotics used in the test, we presumed it to be a community acquired infection through ingestion of food contaminated with *A. baumannii.*

CONCLUSION

This to the best of our knowledge is the first case report of community acquired pyogenic liver abscess in our centre, caused by *A. baumannii*. Apart from *A. baumannii* other species are also emerging in hospital and community settings. Therefore, there is need for a high index of suspicion to be able to identify *A. baumannii* associated infections when they occur either in the hospital environment as hospital acquired infection or in the community as community associated infections. Measures such as improved infection prevention and control practices, diligent environmental cleaning, antibiotics stewardship practices and education of HCWs on regular hand hygiene is important to be able to control the spread of the organism.

REFERENCES

1. Peralta R. Liver Abscess. In: Liver Abscess. New York, NY: WebMD. http://emedicine.medscape.com/article/188802. Updated June 20, 2016. [Date accessed: June 4th 2020]
2. Silver S, Weinstein A, Cooperman A Changes in the pathogenesis and detection of intra-hepatic abscess.1979. *Am J Surg*; 137(5):608-610
3. Bari S, Sheikh KA, Ashraf M, Hussain Z, Hamid A, Mufti GN. Ascaris liver abscess in children. *J Gastroenterol* 2007; 42(3):236-40
4. Miedema BW, Dineen P. The diagnosis and treatment of pyogenic liver abscesses. *Ann Surg* 1984; 200:328–335
5. Clinical and Laboratory Standards Institute (CLSI) 24th Informational Supplement, M100-S24. CLSI; Wayne, PA: 2014. Performance standards for antimicrobial susceptibility testing. Available at: https://clsi.org/media/2663/m100ed29_sample. [Date accessed: May 21st 2020]
6. Gyorffy EJ, Frey CF, Silva J Jr, McGahan J. Pyogenic liver abscess. Diagnostic and therapeutic strategies. *Ann Surg* 1987; 206:699–705.
7. Kaplan GG, Gregson DB, Laupland KB. Population-based study of the epidemiology of and the risk factors for pyogenic liver abscess. *Clin Gastroenterol Hepatol* 2004; 2(11): 1032–1038. doi: 10.1016/S1542-3565(04)00459-8.
8. Haishen Kong, Fei YU, Welli Zhang and Xuefen L. Clinical and Microbiological Characteristics of Pyogenic liver abscess in a Tertiary hospital in East China. *Medicine* 2017; 96(37): e8050; PMCID: PMC5604666; PMID 28906397; DOI: 10. 1097/ MD. 0000000000008050

9. Tian LT, Yao K, Zhang XY, Zhang ZD, Liang YJ, Yin DL, et al. Liver Abscess in adult patients with and without diabetes mellitus: An analysis of the clinical characteristics, features of the causative pathogens, outcomes and predictors of fatality: A report based on a large population, retrospective study in China. *Clin Microbiol Infect* 2012; 18:314-330.
10. Meddings L, Myers RP, Hubbard J, Shaheen AA, Laupland KB, Dixon E, et al. A population-based study of pyogenic liver Abscess in the United States: Incidence, mortality, and temporal trends. *Am J Gastroenterol* 2010; 105:117-124.
11. Guardabassi L, Dalsgaard A, Olsen JE. Phenotypic characterization and antibiotic resistance of Acinetobacter spp. isolated from aquatic sources. *J Appl Microbiol* 1999; 87:659-667
12. Mittal S, Sharma M, Yadav A, Bale K, Chaudhary U. Acinetobacter lwoffii. An emerging pathogen in neonatal ICU. *Infectious Disorders – Drug Targets* 2015; 15:184-188.
13. Silva GM, Morais L, Marques L, Senra V. Acinetobacter community-acquired pneumonia in a healthy child. *Rev Port Pneumol* 2012; 18:96-98.
14. Rathinavelu S, Zavros Y, Merchant JL. Acinetobacter lwoffii infection and gastritis. *Microbes Infection* 2003; 5:651–657.
15. Nakwan N, Warnaco J, Nakwan N. Multidrug-resistant Acinetobacter lwoffii infection in neonatal intensive care units. *Res Rep Neonatal* 2011;1: 1-4
16. Rawat D, Nair D. Extended spectrum β-lactamases in gram negative bacteria. *Journal of Global Infectious Diseases* 2010;2(3):263(1-13). Doi:10.4103/0974-777X.68531
17. Tega L, Raieta K, Ottaviani D, Russo G, Blanco G, Carraturo A. Catheter-related Bacteraemia and Multidrug-resistant Acinetobacter lwoffii. *Emerg Infect Dis* 2007;13(2):355-356