*Indian Journal of Medical Case Reports ISSN: 2319–3832(Online)*
*An Open Access, Online International Journal Available at http://www.cibtech.org/jcr.htm*
*2018 Vol.7 (3) July-September, pp. 1-3/Tarun et al.*
***Case Report***

# CIKPLA SYNDROME

***Tarun S[1], Jacob Mathews[2] and Indira Menon[2]**
*[1]D-1302, Footprints, Chokkanahalli, Thannisandra Main Road, Bangalore-560066*
*[2]Bangalore Baptist Hospital, Bellary Road, Hebbal, Bangalore-560024*
**Author for Correspondence: drtarunrao@hotmail.com*

## ABSTRACT
CIKPLA (Cryptogenic invasive *K. pneumoniae*-associated liver abscess) syndrome has not been reported in India. We report a laboratory-confirmed case of CIKPLA syndrome in 50 yr old patient of Indian origin presenting with community acquired, invasive liver abscess. Case was studied at Bangalore Baptist Hospital, Bangalore.

## INTRODUCTION
Cryptogenic invasive *K. pneumoniae*-associated liver abscess (CIKPLA) syndrome has distinct clinical and epidemiologic features. It is typically community acquired. We report a case of CIKPLA syndrome in 50 yr old patient of Indian origin.
Cryptogenic invasive *K. pneumoniae*-associated liver abscess (CIKPLA) syndrome is typically community-acquired and is highly associated with distant septic seeding. The syndrome appears to target populations of Asian descent with underlying disorders reflecting an impaired immunity.

***Keywords:****, Abscess, Antibiotics, Community acquired, Invasive*

## CASE
A 50-yrs-old male patient came to our hospital with complaints of breathlessness since 7 days and high-grade continuous fever, without chills or rigors since 7 days. Along with pain in right hypochondrium, which was dull aching type, more on coughing and was associated with catch in inspiration. He also had cough with scanty whitish expectoration, non-foul smelling and with no aggravating or relieving factors. There was no history of chest pain, burning micturition, altered bowel habits, or any major illness in the past. He was a known diabetic on medications since 2 years. On admission, he was averagely built and nourished with a toxic look, restless, pulse-110/min, BP-140/70 mmHg, and respiratory rate- 26 cycles/min. On systemic examination, cardiovascular and central nervous systems were normal. Respiratory system examination revealed reduced breath sounds on right side with bilateral extensive coarse crepitations, more on the right side. His abdomen examination revealed tenderness in right hypochondrium. His laboratory reports were as follows:
Hb- 11G%; TLC- 7700/cmm; Platelet count- 1.43 lakh; PT(INR)- 1.4; Random blood sugar- >500 mg/dl; serum acetone-negative, blood urea- 29mg/dl; serum creatinine- 0.8 mg/dl; serum sodium- 119.8 mg/dl; serum potassium- 4.3mg/dl; urine (routine and microscopy)- 20-25 pus cells.
His initial chest X-ray showed bilateral non homogenous opacities. His breathlessness continued to worsen. CT chest was done which showed bilateral multiple patchy alveolar opacities with bilateral moderate pleural effusion and a possibility of septic emboli in the right lower lobe (figure 1). He was immediately started on injectable ceftriaxone 1 gm IV q 12 $^{th}$ hourly and oral Azithromycin 500 mg once a day. He was managed for his hyperglycemia with insulin infusion and controlled USG abdomen and pelvis –two ill defined necrotic lesions of 4.3x2.6 cms and 3.2x2.5cms in segment5/6-suggesting evolving abscess, bilateral mild pleural effusion and (figure2 ). His breathlessness rapidly worsened and requiring ventilator support. Also, he required vasopressors. His antibiotics were immediately hiked up to Meropenam 1g iv q 8$^{th}$ hourly.
Meanwhile, USG-guided aspiration of liver abscess was performed on emergency basis, and 100 ml of anchovy-sauce aspirate was removed and sent for analysis sent for culture and sensitivity. Meropenam

was continued. Meanwhile, the culture report of the aspirated pus as well as the blood culture showed the growth of *Klebsiella* sensitive to Meropenam, Piperacillin/Tazobactum ,ciprofloxacin, amikacin, and cefotaxim. Meanwhile, sputum was also sent for culture and sensitivity studies, which showed normal flora. As his general condition was sick and as he was on mechanical ventilator, the antibiotics were not de-escalated and it was continued. However his hypotension was refractory in spite of the vasopressors. Finally he succumbed to the illness on the 14th day of his hospital stay.

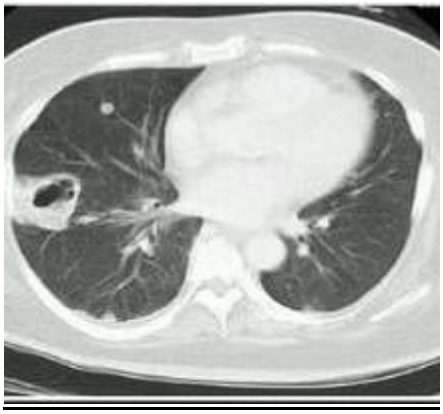

**Figure 1: CT scan showing lung abscess**

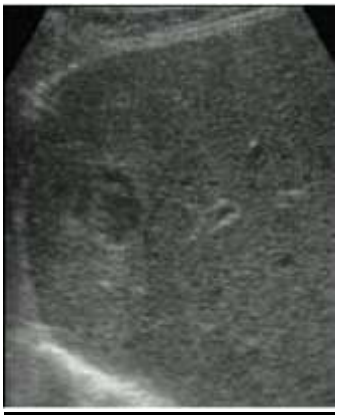

**Figure 2: Ultrasound abdomen showing liver abscess**

**DISCUSSION**

*K. pneumoniae* is an encapsulated Gram-negative bacillus belonging to the family of Enterobacteriaciae. Important geographical differences exist in its clinical presentation in different parts of the world. CIKPLA syndrome is a community-acquired, mostly monomicrobial, solitary liver lesion frequently associated with diabetes mellitus and classically complicated by one or more septic conditions (e.g., meningitis, endophthalmitis, lung abscess, or necrotizing fasciitis). The CIKPLA cases reported have previously only been documented in patients of Asian origin (Braiteh and Golden, 2007). According to Ko *et al*., more than 900 cases of *K. pneumoniae*-associated liver abscess syndrome have been reported from Asian countries in the last decade.

Previous studies in Taiwan have demonstrated that the CIKPLA syndrome appeared to be common in middle-aged men who had concomitant diabetes mellitus (Ko *et al.,* 2002). This has been attributed to the impaired phagocytosis of the capsulated *Klebsiella* organism due to the presence of diabetes mellitus; however, the CIKPLA syndrome has also been documented in patients who were not diabetic, suggesting that there are intrinsic virulence factors in the organism that convey a predilection to escape the host's immune response (Ko *et al.,* 2002) *magA* and *rmpA* (tested by PCR method) are good markers to diagnose liver abscess in *Klebsiella* bacteremia. *K. pneumoniae* serotypes isolated in Taiwanese patients with CIKPLA had a high prevalence of capsular polysaccharide serotypes K1 and K2 and an increased resistance to phagocytosis and intracellular killing. Resistance to phagocytosis and bacterial death in human serum observed in the invasive strains has been linked to the virulence gene *magA*, which encodes the outer membrane protein of a mucoviscous exopolysaccharide web.

A few poor prognostic features have been documented in the CIKPLA syndrome, namely a Glasgow Coma Scale <7 on initiation of treatment (Fang and Chen, 1993) high white blood cell count, thrombocytopenia, and low glucose in the CSF (Huang CR, Lu CH,2002). Although the organism appears to be more virulent, it remains sensitive to a prolonged course of intravenous cephalosporins (three weeks) and drainage of the abscess (Casella *et al.,* 2009). ESBL-producing *K. pneumoniae* is rarely isolated from the aspirates of monomicrobial liver abscesses (invasive or noninvasive), which are commonly community-acquired conditions (4.3%) (Ko *et al.,* 2002). Therefore, the treatment of choice

would be carbapenams and combination cephalosporin-beta-lactamase inhibitor agents are promising (Harris *et al.*, 2015). A high index of suspicion for CIKPLA in any patient with *Klebsiella* bacteremia needs to be considered, as the condition can be fatal if not appropriately managed.

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