MAK HILL Publications International Journal of Tropical Medicine

Case Report
doi: 10.59218/makijtm.2024.2.17.20

OPEN ACCESS

Key Words
Acute pancreatitis, alcohol use, mumps, cytomegalovirus, parasites, coxsackievirus

Corresponding Author
Nayan Paul,
Department of General Medicine, KPC Medical College and Hospital, 1F, Raja S. C. Mullick Road, Jadavpur, Kolkata, West Bengal, 700032, India
nayanpaulmd28@gmail.com

Author Designation
[1]1st year Post-Graduate Trainee
[2]2nd year Post-Graduate Trainee
[3]3rd year Post-Graduate Trainee
[4]Associate Professor
[5]Senior Resident

Received: 31 January 2024
Accepted: 15 February 2024
Published: 22 February 2024

Citation: Nabanita Mondal, Nayan Paul, Amit Karmakar, Kausik Munsi and Puja Mahato, 2024. Acute Pancreatitis Secondary to Icteric Leptospirosis: A Case Report. Int. J. Trop. Med., 18: 338-341, doi: 10.59218/makijtm.2024.2.17.20

Acute Pancreatitis Secondary to Icteric Leptospirosis: A Case Report

[1]Nabanita Mondal, [2]Nayan Paul, [3]Amit Karmakar, [4]Kausik Munsi and [5]Puja Mahato

[1-5]Department of General Medicine, KPC Medical College and Hospital, 1F, Raja S. C. Mullick Road, Jadavpur, Kolkata, West Bengal, 700032, India

ABSTRACT
Acute pancreatitis is one of the most common causes of hospitalisation globally, posing a significant health risk that results in higher rates of mortality and morbidity. Consequently, prompt diagnosis and treatment are imperative. Acute pancreatitis can be caused by various factors, with gallstones and alcohol use being the most common ones. Infections such as mumps, cytomegalovirus, coxsackievirus, and parasites are rare causes of pancreatitis, which may occasionally be the causative agent of acute pancreatitis. In this cbeen diagnosed with Icteric leptospirosis and developed acute pancreatitis subsequently. But fortunately, the patient had fully recovered after receiving timely treatment and intervention.

INTRODUCTION

Leptospirosis is a zoonotic disease which is primarily prevalent in tropical regions, is typically transmitted through the bite of an infected animal or contact with water or soil contaminated with the urine of an infected animal[1]. It is characterised by a wide range of symptoms, varying from mild and asymptomatic to severe multi-organ dysfunction. Severe leptospirosis, also known as icteric leptospirosis, can manifest with acute kidney injury (AKI), bleeding symptoms, and hepatitis (icterohemorrhagic fever or Weil's Disease)[2]. But pancreatitis is a very rare presentation and is also more common in children rather in adult[3]. The spirochete itself and the cytokines generated by this infection may contribute to premature trypsin activation, resulting in the proteolytic digestion of the pancreas and subsequent development of acute pancreatitis. This report presents a case of leptospirosis that resulted in pancreatitis and its related consequences.

Case Description: The patient, a 55-year-old postman, came to our emergency with several complaints like fever and severe headaches for 9 days, muscle cramps and knee pain for 8 days, nausea, vomiting, and loose stools for 7 days, abdominal pain for 5 days, and significant difficulty in breathing for 3 days. He was then admitted to our intensive care unit (ICU) with a diagnosis of multiple organ dysfunction syndrome (MODS) and sepsis (diagnosed prior to admission at our institution). He was previously hospitalised at another facility centre due to symptoms including fever, vomiting, myalgia, loose stool, and headache, which occurred 6 days ago. He experienced abdominal pain and difficulty in breathing, which worsened one day ago. As a result, he was referred to a more advanced medical facility. Upon admission to our institution, this patient's physical examination revealed several symptoms and findings: pallor, icterus, subconjunctival haemorrhage (Fig. 1), elevated jugular venous pressure, bilateral pitting pedal edoema, a temperature of 99.8°F, blood pressure of 60/40, heart rate of 110 beats per minute, blood glucose level of 266 mg dL^{-1}, respiratory rate of 32 breaths per minute. Systemic examination revealed audible S1 and S2 heart sounds with an additional S3 gallop, fine crepitations in both lung bases, decreased breath sounds at the base of the lungs, abdominal tenderness, and a Glasgow Coma Scale score of 15/15. The patient was experiencing warm septic shock. Upon reviewing the prior reports, we found the following:

Day 1 of Admission: Hb- 9.9. TLC 7720(ANC 7450). Platelet 88,000. Albumin 3.2. SGOT 53, SGPT 74.

Procalcitonin 13.19, CRP 30.943. Urea 74, creatinine 3.18, Na 126, k 4.1. Dengue NS1, MPDA, Typhoid IgM-Non Reactive. Blood culture: negative.

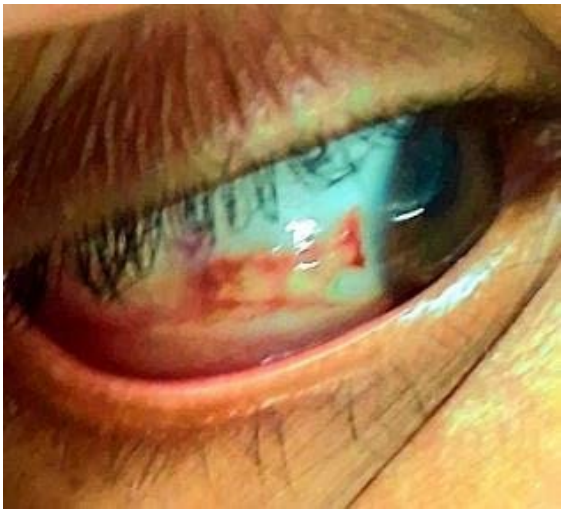

Fig. 1: Subconjunctival hemorrhage

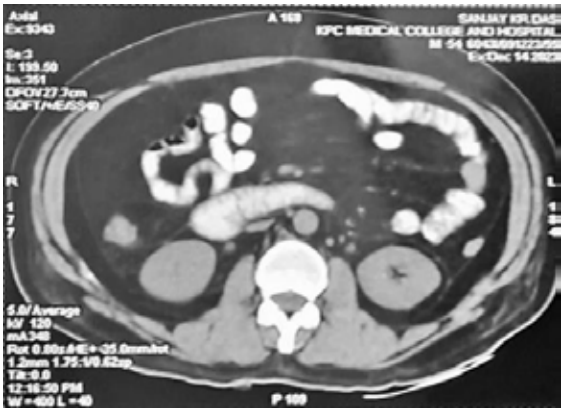

Fig. 2: Chest xray: features of ARDS

Day 3 of Admission: Amylase 113, Lipase 73, calcium 8.2. Lipid profile: Triglyceride 284, VLDL 59, LDL 18, HDL 18.

Day 4 of Admission: SGOT 5664, SGPT 2662, AMYLASE 509, LIPASE 1457. Hepatitis A igM, hep E IgM, anti HCV, hbsAg were all Negative. The patient received IV Meropenem treatment outdoors, but his condition worsened with severe respiratory distress. He was then referred to a higher-level medical facility for further management, with a diagnosed of GI Sepsis, hepatitis, hyponatremia, thrombocytopenia, pancreatitis, and pleural effusion. Subsequently, he was transferred to our hospital. Upon further assessment, it was determined that the patient is suffering from Sepsis accompanied by Acute Kidney Injury (AKI), moderate Acute Respiratory Distress Syndrome (ARDS) (Fig. 2) and heart failure with preserved ejection fraction. In order to determine the aetiology and foci of Sepsis, upon conducting another round of normal blood tests, we discovered:

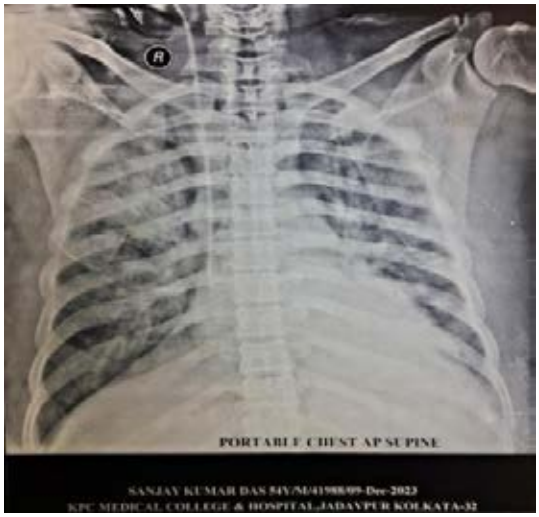

Fig. 3: CECT Whole abdomen: bulky and heterogeneous pancreas suggestive of acute pancreatitis

Table 1: Pathology and Biochemical reports of the patient during Hospital stay and follow up

Investigations	DAY 1	DAY 4	DAY 7	Day 14	Day 21	Day 35
Total Leucocyte count	7720	9950	21210	10990	7500	6540
SGOT	63	5664	564	112	38	34
SGPT	74	2662	562	401	26	28
AMYLASE	113	509	308	440	235	87
LIPASE	73	1457	684	418	70	44
Urea	74	158	172	50	20	18
Creatinine	3.18	4.12	3.5	1.1	0.8	0.8

Day 7 of 1st Hospital Admission: The laboratory results are as follows: TLC- 21210, NT proBNP 6398, urea 172, creatinine 3.5, Na 131, Lipase 687, Amylase 308. Blood culture: negative for bacterial growth. The serology test results indicate a negative response. An abdominal ultrasound scan shows an enlarged and irregular pancreas, which is indicative of pancreatitis. Acute pancreatitis can account for the occurrence of ARDS. Here we have used widely recognised scoring methods to categorise and predict the seriousness of pancreatitis. 1) Revised Atlanta criteria: severe pancreatitis, 2) Modified Marshall Score -> 1. Respiratory: PaO_2/fiO_2: 240: score 2, 2. Renal: Creatinine 3.5 mg dL^{-1}: score 2 3. cardiovascular: SBP 80 which was responsive to fluid: score 1. 3) BISAP Score: 3 indicating 5-20% mortality (Table 1).

We proceeded to treat the patient conservatively with the help of intravenous antibiotics, carefully controlling hydration, and implementing other necessary supportive measures. In this clinical background, there was a strong suspicion of leptospirosis. The leptospira IgM test was conducted on the 8th day of hospital admission, and the result was positive. Managing the disease was challenging due to the patient's simultaneous presence of ARDS, heart failure and septic shock. Nevertheless, his condition gradually improved and once the levels of urea creatinine stabilised, a contrast-enhanced computed tomography (CECT) scan of the entire abdomen was performed on the 13th day, which showed signs of acute pancreatitis (Fig. 3). The computed tomography (CT) severity score was 6.

The patient received conservative management in the ICU, which involved close observation, intravenous fluid administration, intravenous antibiotics, and other supportive measures. Over time, the patient's acute respiratory distress syndrome (ARDS) improved, acute kidney injury (AKI) resolved, hepatic enzymes normalised, pancreatic enzymes decreased, and the patient's clinical condition improved. Eventually, the patient was discharged in a stable hemodynamic state with oral Doxycycline. The patient was advised to follow up in the outpatient department (OPD) once fully recovered from all complications.

After 2 weeks, he visited the outpatient department (OPD) for a follow-up. His levels of Amylase, Lipase, SGOT, SGPT, Urea, and Creatinine were all within the normal range. Currently, he has recovered fully and leading a normal lifestyle.

DISCUSSION

This report describes a case of Leptospirosis characterised by the presence of acute hepatitis, Acute Kidney Injury, Septic Shock, Pleural effusion, and notably acute pancreatitis. The patient was non-alcoholic, had a triglyceride level that was close to normal, low calcium levels, absence of gallstones, no history of trauma, and no prolonged use of any specific medication. The patient exhibited a significantly elevated total leucocyte count, along with higher levels of C-reactive protein (CRP) and Procalcitonin. However, the origin of the illness remained unidentified. During a thorough examination of his medical history, we discovered that he had sustained an abrasion on his foot. Despite this injury, he chose to continue working. During the monsoon season in tropical climates, the stagnant rainwater that is contaminated and blocked might serve as a potential cause of infection. Given that all other reports, such as the viral Bio-fire, Blood culture, urine culture, and infective hepatitis were negative, it is reasonable to assume that the pancreatitis was caused by leptospira itself, but the possibility of coinfection cannot be completely eliminated. Leptospirosis seldom causes pancreatitis which has been documented worldwide[4-6]. Pancreatitis is an unusual but dreadful presentation of Leptospirosis.

CONCLUSION

In the background of sepsis with pancreatitis presenting with fever, jaundice, pain abdomen and renal dysfunction, a rare possibility of leptospirosis should be considered as early diagnosis and intervention can lead to favourable outcome in such patients with complete recovery and less mortality.

REFERENCES

1. Ding, G., X. Li, D. Fang, H. Hao, X. Li and L. Zhou, 2021. Etiology and ureteral reconstruction strategy for iatrogenic ureteral injuries: A retrospective single-center experience. Urol. Internat., 105: 470-476.
2. Hartskeerl, R.A., M.C. Pereira and W.A. Ellis, 2011. Emergence, control and re-emerging leptospirosis: Dynamics of infection in the changing world. Clin. MicroBiol. Infec., 17: 494-501.
3. O'brien, M.M., J.M. Vincent, D.A. Person and B.A. Cook, 1998. Leptospirosis and pancreatitis: A report of ten cases. Pediatr. Infect. Dis. J., 17: 436-438.
4. Spichler, A., E. Spichler, M. Moock, J.M. Vinetz and J.A.D. Leake, 2007. Acute pancreatitis in fatal anicteric leptospirosis. Am. J. Trop. Med. Hyg., 76: 886-887.
5. Kaya, E., 2005. Acute pancreatitis caused by leptospirosis: Report of two cases. World J. Gastroenterol., Vol. 11, No. 28. 10.3748/wjg.v11.i28.4447.
6. Pai, N.D. and P. Adhikari, 2002. Painless pancreatitis: A rare manifestation of leptospirosis. J. Assoc. Physicians India, 50: 1318-1319.