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Case Report

Acute cholecystitis secondary to *Haemophilus parainfluenza*: a rare occurrence

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ABSTRACT

This case report is intended to contribute to the medical community's knowledge of the rare occurrence of *Haemophilus parainfluenzae* (HPI) isolated from bile fluid in a 66-year-old male with acute cholecystitis complicated by septic shock. HPI, has predominantly been known as an oropharyngeal commensal bacterium, but it's emergence to be recognized as an opportunistic pathogen may have been under exaggerated. The patient's clinical course included an initial expected presentation of abdominal pain, sudden deteriorating condition complicated by septic shock and complications demanded for flexible yet meticulous clinical management. Clinical course continues with successful percutaneous cholecystostomy tube placement, and close multidisciplinary management involving surgery, infectious disease and cardiology. This case report serves as an opportunity to consider unexpected infectious organisms in biliary infections, a documentation of clinical resilience, and especially in patients with significant interplaying comorbidities. Additionally, the discussion elaborates on the growth requirements of HPI in bile fluid, emphasizing the significance of factor V for its proliferation.

Keywords: Biliary tract, Complicated acute cholecystitis, Haemophilus parainfluenzae, HPI, Percutaneous cholecystostomy tube

INTRODUCTION

Haemophilus parainfluenzae (HPI) is a gram-negative, facultatively anaerobic, coccobacillus commensal bacterium, first recognized in 1922.[1,2] HPI is predominantly found in the oropharynx and is emerging as an opportunistic pathogen, most often seen in children. It is usually associated with upper and lower respiratory tract infections, bacteremia, endocarditis, and meningitis, but rarely causes intra-abdominal infections. Onafowokan et al studied 273 HPI isolates recovered from patients treated at a rural hospital during a two-year period and showed HPI was commonly part of a mixed infection, specifically respiratory tract infections (64.8%). In that study, 13.2% of HPI isolates involved surgical infections (34 patients), with 3 being intra-abdominal, however none were biliary infections.[1] A literature review revealed only 32 intraabdominal infections due to HPI, with just 12 biliary infections.[1] We present a 66-year-old male with acute cholecystitis complicated by septic shock, atrial fibrillation, and pneumonia, requiring a multidisciplinary approach.

CASE REPORT

A 66-year-old male with a history of hypertension, hyperlipidemia, rheumatoid arthritis, gout, and coronary artery disease with angioplasty and stent placements presented with four days of new-onset right upper quadrant abdominal pain associated with altered mental status, without nausea, vomiting, fever, or chills.

On examination, his vitals were: BP 146/84 mmHg, HR 106, RR 19, O_2 sat. 98% on room air, and temperature 98°F, with a positive Murphy's sign. Laboratory values included WBC 13.6, Na 131, CO_2 13, and bilirubin 1.4. A contrast CT confirmed acute cholecystitis, showing pericholecystic fluid, and a distended gallbladder with large sludge (Figure 1a). The patient was placed on nil per os (NPO), intravenous (IV) fluids, and piperacillin tazobactam.

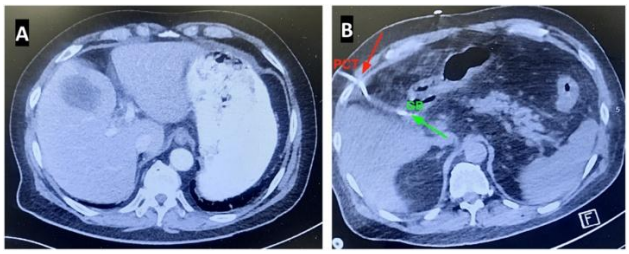

Figure 1: (A) Contrast computed tomography abdomen and pelvis revealing acute cholecystitis accompanied by centralized dilation of the intrahepatic biliary duct. There is pericholecystic fluid and a gallbladder containing a significant volume of sludge; and (B) status-post PCT (red arrow) procedure shows successful placement into gallbladder (green arrow).

Twenty-two hours after presenting to the ED, the patient became febrile (102 °F) and developed septic shock with BP 60/40 mmHg, HR in the 110-120s, tachypnea with a drop of platelets to 75,000, and lactate at 7.69. Fluid resuscitation and vasopressor therapy were initiated, and he underwent emergent ultrasound-guided percutaneous cholecystostomy tube (PCT) placement (Figure 1b). On hospital day 3, bile culture revealed moderate growth of HPI (Figure 2). The patient had elevated BNP (2,250) and worsening thrombocytopenia (58,000), but heparin-induced thrombocytopenia was ruled out.

The patient was weaned off vasopressors by the morning of day 4. Later that day, he developed new-onset 2 of 4 atrial fibrillation and a left lower lobe pneumonia was confirmed, with procalcitonin at 14.4 (Table 1). Anticoagulation therapy, ceftriaxone and metronidazole were initiated. The patient improved and was downgraded from the ICU on day 7. He was noted to have a bloody discharge through his PCT, but this was resolved by day 10. He was discharged to a short-term rehabilitation facility by day 15 on a 14-day course of piperacillin-tazobactam.

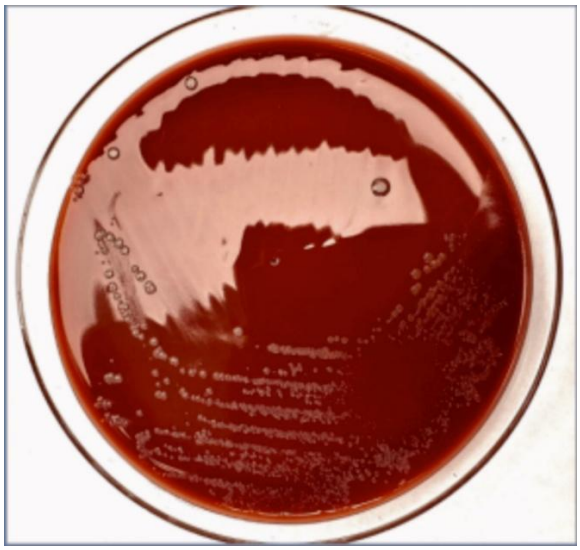

Figure 2: PCT bile fluid culture deposited onto chocolate agar revealing moderate growth of HPI. Circular cleaning shows culture is beta-lactamase negative.

Table 1: Trends of pertinent lab values from admission to hospital day 8.

Variables	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Reference ranges
WBC (×10³/µl)	13.6	11.2	9.7	10.8	8.4	4.3	6	5.6	3.8-11
Lactate (mmol/l)	-	7.69	3.76	1.37	1.97	-	-	-	0.7-2
BNP (pg/ml)	-	-	2250	2330	2920	453	74	-	0-300
Platelets (×10³ K/µl)	134	75	58	57	67	65	102	144	130-400
Procalcitonin (ng/ml)	-	-	-	-	14.4	-	-	2.88	<0.1

DISCUSSION

HPI is an emerging opportunistic pathogen, most often seen in children. It primarily resides in the oropharynx, comprising approximately 10% of the upper respiratory tract microbiome. Though commonly associated with respiratory infections, bacteremia, endocarditis, and meningitis, HPI is rarely implicated in biliary infections. A literature review revealed only 32 intra-abdominal infections due to HPI, with just 12 biliary infections reported.[1] In a study of 273 patients, HPI isolates were predominantly found in the respiratory tract (64%), followed by ear-nose-throat infections (17.9%), surgical infections (3%), genitourinary infections (13.2%), and bloodstream infections (1%), with no biliary tract isolates, revealing the rarity of this pathogen in biliary infections.[1]

Biliary tract infections are most frequently caused by *Klebsiella pneumoniae* and *Escherichia coli*. The mechanism of colonization from the oropharynx to the gastrointestinal tract may occur by swallowing sputum containing HPI; this view is supported by a study that

detected HPI in 20.7% of fecal samples.[3,4] HPI may enter the bile fluid via an ascending route or hematogenous seeding.

Septic shock as the initial presentation of acute cholecystitis is highly unusual, highlighting the need for clinical vigilance. In similar cases, septic shock has been a first sign of gangrenous cholecystitis, a severe complication associated with high mortality.[5] Although gallbladder perforation and complications typically occur in older patients with comorbidities, an initial presentation including septic shock is rare. This case emphasizes the importance of early recognition and aggressive management of this condition, particularly in patients with conditions such as coronary artery disease or immunosuppression. The rarity of HPI as the causative pathogen further complicates management, making heightened awareness essential when patients rapidly deteriorate.[4,5]

HPI has been identified as part of the gastrointestinal microbiome in certain conditions, adding an important dimension to its role in biliary infections. A study by Palmer demonstrated that HPI was isolated from 26% of fecal samples from patients with gastrointestinal symptoms, highlighting its potential to survive and proliferate in the intestines. Coupled with the presence of factor V in bile, this suggests that HPI may enter the biliary tract via an ascending route, supported by bile's ability to nurture its growth.[4] This explains how swallowed sputum containing HPI can lead to rare biliary infections, as seen in this case.

Furthermore, HPI's presence in fecal samples supports the idea that this bacterium may form a biofilm within the gastrointestinal environment, allowing its migration into the biliary system under conditions of disrupted mucosal barriers or immune dysregulation.[4,6] While bile provides factor-V, the lack of haemin (factor-X) in bile might limit the growth of haemin-dependent Haemophilus species, enabling haemin independent strains like HPI to thrive.[4] This adaptation may explain why HPI, though rarely involved in intra-abdominal infections, can become pathogenic in favorable conditions such as acute cholecystitis where inflammation predominates.

Within the bile ducts, HPI's adhesins allow it to attach to biliary epithelial cells, proliferate, and invade the mucosa, potentially leading to biliary stasis and infection. HPI is likely underestimated in clinical settings, emerging as an opportunistic pathogen, particularly during the COVID-19 pandemic, where cases of HPI related infections have been reported due to immune dysregulation.[6] The disk diffusion method on chocolate agar supplemented with hemolyzed erythrocytes (factor-X) and NAD+ (factor-V) aids in determining antibiotic susceptibilities, though HPI's growth requirements contribute to its underdiagnosis.[4] Beta-lactamase-positive HPI strains exhibit resistance to certain antibiotics, but successful outcomes have been reported with cefotaxime, cefpodoxime and new cephalosporin cefditoren.[7]

CONCLUSION

HPI, though a commensal organism in the oropharynx, can reach the gastrointestinal tract and is emerging as an opportunistic pathogen in patients with comorbid conditions or immune dysregulation, contributing to significant morbidity. Recognizing HPI's role in biliary infections and employing appropriate diagnostic techniques and tailored treatment approaches is essential for optimizing patient outcomes. With only a dozen reports of HPI found in the biliary tract, our patient represents the thirteenth case. Although rare, HPI should be considered as a potential culprit in complicated biliary infections, especially in patients with rapid clinical deterioration.

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