

Chryseomonas luteola from Bile Culture in an Adult Male with Severe Jaundice

Anuradha S De, Parul P Salunke, Harshal R Parikh, Sujata M Baveja

Department of Microbiology, L.T.M. Medical College, Mumbai, India

Address for correspondence: Dr. Anuradha De, E-mail: dr_anuradhade@yahoo.com

ABSTRACT

A 60-year-old male was admitted in this hospital with severe jaundice, who had open cholecystectomy done 2 months ago. ERCP was performed and bile was sent for culture. It grew *Chryseomonas luteola* in pure culture. He underwent hepaticojejunostomy after 1 month. Total bilirubin improved gradually. His condition was stable on discharge. Prompt diagnosis of non-fermenters is required, as some of them are resistant to multiple antibiotics. Clinicians have to be made aware of the pathogenic role of *C. luteola* and its resistance to ampicillin and cephalosporins.

Keywords: Bile culture, jaundice, *Chryseomonas luteola*

DOI: 10.4103/0974-2727.66708

www.jlponline.org

INTRODUCTION

Chryseomonas luteola though rarely reported as a human bacterial pathogen, has been implicated in producing septicemia,^[1] meningitis,^[2] prosthetic valve endocarditis,^[3] peritonitis usually in association with indwelling catheters or prostheses,^[4] osteomyelitis,^[5] as well as in invasive cutaneous infection.^[6] We report here a case of *C. luteola* in biliary infection.

CASE REPORT

A 60-year-old male hailing from Akola, Maharashtra, was admitted on 15th April 2009 in our hospital with complaints of jaundice and itching for 2 months, along with loss of appetite and weight loss. He had undergone open cholecystectomy in a private hospital in February 2009.

On examination, he was afebrile with pallor and icterus. His pulse was 86/minute and blood pressure was 110/70 mmHg. No abnormality was detected in cardiovascular, central nervous system, and respiratory system. Abdomen was soft. Total leukocyte count on admission was 15,000/cu mm, with neutrophils 85%, lymphocytes 13%, eosinophil 1%, and monocyte 1%. Hemoglobin was 10 gm/dl. His total bilirubin was 10.6 mg/dl (direct 5.9 and indirect 4.7 mg/dl). Fasting

blood sugar level was 101 mg/dl. HIV antibody and HBsAg were negative.

Endoscopic retrograde cholangiopancreatography (ERCP) revealed total tear of the bile duct at supraduodenal level. A percutaneous transhepatic drainage was done under local anesthesia and a bile sample was sent for culture. The specimen was directly plated on MacConkey agar (MA) and blood agar (BA), incubated at 37°C overnight. Simultaneously enrichment was also done in 0.5% bile broth. Next day 2-3 mm circular, smooth, convex, non-lactose fermenting colonies appeared on MA, which were catalase positive and oxidase negative. On BA, colonial morphology was same with a dull yellow pigment. Hanging-drop preparation revealed that the organism was motile. It was identified as *C. luteola* by standard biochemical tests.^[7] It utilized glucose, mannitol, and maltose oxidatively, hydrolyzed esculin, reduced nitrates to nitrites, did not decarboxylate lysine, did not hydrolyze urea and did not produce indole. It was sensitive to Polymyxin B, but resistant to Penicillin G.

Subculture from bile broth onto MA also grew the same bacteria. Antibiotic susceptibility was tested by the Kirby-Bauer disk diffusion method onto Mueller-Hinton agar according to CLSI guidelines.^[8] It was susceptible to amikacin, ciprofloxacin and imipenem, but resistant to ampicillin, amoxicillin-clavulanic

acid, cefotaxime, ceftriaxone, ceftazidime, piperacillin, and piperacillin-tazobactam. Daily fluid output from the drain was 500-600 ml. He was given ciprofloxacin 500 mg twice daily and omeprazole once a day on an empty stomach. Fluid output gradually reduced and total bilirubin became 3.94 mg/dl. But total leukocyte count was 11,100/cu mm, with N 80%, L 17%, E 1%, M 2%.

Therefore, hepaticojejunostomy was done after 1 month of admission. Postoperatively he received cefotaxime, gentamycin, and metronidazole intravenously for 2 weeks. He recovered gradually and was discharged after removing the T-tube. His condition was stable on discharge.

DISCUSSION

C. luteola was initially assigned to CDC Group Ve-1 and was named as *Chromobacterium typhiflavum* and later as *Pseudomonas luteola*.^[1-3] It is a motile, aerobic, gram-negative, nonfermentative bacilli with dull yellow pigment and is oxidase negative. Though Chihab *et al*,^[2] have reported *C. luteola* growing after 48 h incubation on heart infusion agar supplemented with blood, we got excellent growth of the same bacteria on MA and BA after 24 h. This bacterium can be easily distinguished from other yellow pigmented nonfermenters by its negative oxidase reaction. From *Flavimonas oryzihabitans* it can be distinguished by its hydrolysis of esculin and from *Stenotrophomonas maltophilia* it is differentiated by its inability to decarboxylate lysine.^[7]

The habitat of *C. luteola* is unclear, but it is frequently found in water, soil, and moist environments.^[1,4] In PubMed search with '*Chryseomonas luteola* in biliary infections in India', no item was found. This may be the first case report of *C. luteola* in biliary infection from India. *C. luteola* is extremely resistant to cephalosporins and ampicillin, but susceptible to aminoglycosides and ciprofloxacin,^[2] as is also reported by us. Mortality due to *C. luteola* is also known,^[2] but in our case the patient recovered and was discharged. Severe immune deficiency due to HIV infection, diabetes, anemia,

and liver dysfunction can be responsible for *C. luteola* infection.^[6] The present case had severe jaundice. It is unlikely that it was a contaminant, because *C. luteola* grew in pure culture on direct plating and also on subculture from bile broth. Moreover, no other cultures prior to or immediately after this have grown this bacterium in our laboratory.

Thus, proper identification of the nonfermenters is the need of the day, by putting up a battery of biochemical tests based on oxidase test and motility.^[7] Clinicians and laboratory personnel also have to be made aware of the pathogenic role of *C. luteola* in certain clinical circumstances, which may become increasingly prevalent in the near future. Clinicians should be made aware of the characteristic resistance of *C. luteola* to ampicillin and cephalosporins and susceptibility to aminoglycosides and quinolones.

REFERENCES

1. Freney J, Hansen W, Etienne J, Vandevesch F, Fleurette J. Postoperative infant septicaemia caused by *Pseudomonas luteola* (CDC Group Ve-1) and *Pseudomonas oryzihabitans* (CDC Group Ve-2). J Clin Microbiol 1988;26:1241-3.
2. Chihab W, Alaoui AS, Amar M. *Chryseomonas luteola* identified as the source of serious infection in a Moroccan University hospital. J Clin Microbiol 2004;42:1837-9.
3. O'Leary T, Fong IW. Prosthetic valve endocarditis caused by Group Ve-1 bacteria (*Chromobacterium typhiflavum*). J Clin Microbiol 1984;20:995.
4. Silver MR, Felegie TP, Sorkin MI. Unusual bacterium, Group Ve-2, causing peritonitis in a patient on continuous ambulatory peritoneal dialysis. J Clin Microbiol 1985;21:838-9.
5. Rahav G, Simhon A, Mattan Y, Moses AE, Sachs T. Infections with *Chryseomonas luteola* (CDC Group Ve-1) and *Flavimonas oryzihabitans* (CDC Group Ve-2). Medicine 1995;74:83-8.
6. Ghosh SK. A rare infection caused by *Chryseomonas luteola*. J Infect 2000;41:109-10.
7. Koneman EW, Allen SD, Janda WM, Schreckenberger PC, Winn WC Jr. The nonfermentative gram-negative bacilli. In Color Atlas and Textbook of Diagnostic Microbiology. 5th ed. Philadelphia: Lippincott; 1997. p. 273-4, 291-3,295.
8. Performance Standards for Antimicrobial Susceptibility Testing; Seventeenth Informational Supplement. Clinical Laboratory Standards Institute. 2007; M100-S17: p 38-9.

Source of Support: Nil, Conflict of Interest: None declared.