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A Case of *Cedecea davisae* Peritonitis in a Liver Cirrhosis Patient

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간경변증 환자에서 발생한 *Cedecea davisae* 복막염 1예

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Cedecea davisae is a motile, Gram-negative rod in the family Enterobacteriaceae which is positive for lipase, DNase and catalase, and negative for gelatinase and oxidase. This bacterium is rarely isolated in the clinical specimens. We isolated *C. davisae* from the ascitic fluid of a 49-year old male patient with liver cirrhosis who was diagnosed as acute bacterial peritonitis. Bacterial identification was performed by API 20E and VITEK. Antimicrobial susceptibility test showed that the isolate was susceptible to cefotaxime, piperacillin, and imipenem. Peritonitis of this patient was improved by imipenem therapy. This is the first reported case of peritonitis caused by this organism.

Key Words : *Cedecea davisae,* Peritonitis, Liver Cirrhosis

Introduction

The genus Cedecea was described in 1981 and now includes *Cedecea. davisae, C. lapagei, C. neteri,* and two unnamed species[1-4]. Strains of *Cedecea* resemble the species of Serratia in many of the common tests used for identification, but the species of *Serratia* producing gelatinase, DNase are distinct from strains of *Cedecea* which are rarely isolated in clinical specimens. Among the species of *Cedecea, Cedecea davisae* is the most common. Seventeen clinical isolates of the species designated as *C. davisae* have previously been reported[1)]. Perkins and co-workers reported a case of bacteremia caused by *C. daviasae* in 1986. However, a literature search review revealed no reports of a prior isolation of *C. davisae* from ascitic fluid. We report a case of peritonitis caused by *C. davisae*.

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Case Report

A 49-year-old male with alcoholic liver cirrhosis was admitted to Chosun University Hospital due to abdo minal distension developed 15 days ago.

On the admission, he appeared acutely distressed but was mental alert. The vital signs were as follows: blood pressure 100/60mmHg, pulse rate 96/min, respiratory rate 24/min, body temperature 36.5℃. On physical examination, jaundice on the skin and sclera, multiple spider on the upper anterior chest wall, abdominal distension, and fluid wave, and shifting dullness were observed. Pitting

edema on the extremities was also noted.

The findings obtained from CBC analysis were as follows : WBC count 11,600/mm^3(composed of 82% PMNL, 8.3% lymphocyte and 8.8% monocyte), platelet count 65,000/mm^3, ESR 10mm/hr, hemoglobin 10.5g/dL, hematocrit 29%, PT 22.5 sec, aPTT 68.5 sec.

Routine chemistry showed total protein 5.2g/dL, albumin 1.8g/dL, AST 67U/L, ALT 10U/L, alkaline phosphatase 122 U/L, ratio of BUN to Cr 40/1.9mg/dL, glucose 85mg/dL, ratio of Na^+ to K^+ 128/4.5mEq/L, HBsAg negative, anti-HBs positive, and anti-HCV negative. Urine analysis showed no significant abnormal findings.

Ascitic fluid analysis revealed the following findings: WBC 98/mm^3 with 68% PMNL and 32% monocyte, protein 1.39g/dL, sugar 97mg/dL, LDH 70U/L and normal adenosine deaminase.

The patient was given on spironolactone and furosemide, and was started on a low salt diet. The abdominal distension was not improved but was worsened to 5 days after admission. Body temperature was elevated to 39.2℃, and tenderness and rebound tenderness appeared. But X-ray examination did not show any specific findings. The patient was diagnosed clinically as having primary bacterial peritonitis, and paracentesis was performed to confirm and identify the causative bacteria. Ascitic fluid was turbid with yellowish and contained 5,200/mm^3 WBC with 95% PMNL and 5% monocyte. Total protein and albumin were 1.0 g/dL and 253mg/dL, respectively. Glucose was not detected and LDH was 350U/L. On CBC analysis, WBC was 15,000/mm^3 and platelet 86,000/mm^3. Hemoglobin and hematocrit were 8.7g/dL and 26.1%, respectively. Furthermore, in the ascitic fluid culture, Gram-negative rods were isolated, which showed biochemical profile number of 3304321 when tested by the API 20E system(BioMerieux, France). According to the API profile index, the causative bacterium was *C. davisae* (%id=99.9 and T=0.75) that had not been reported to be isolated from ascitic fluid until that time. The bacterium was also identified as *C. davisae* by VITEK(BioMerieux AC-2, France: 99% *C. davisae*, 1% *C. lapagei*). No other bacteria were isolated. This patient was confirmed as having primery bacterial peritonitis caused by *C. davisae*. As the antibiotic susceptibility test showed *C. davisae* to be susceptible to cefotaxime, piperacillin, and imipenem but resistant to aminoglycosides, amoxicillin, amikacin, cefamandole and cefazolin, the patient was started on cefotaxime.

On hospital day 8, body temperature was 38.9℃, and abdominal distension, tenderness and rebound tenderness still remained. As the patient did not respond well to cefotaxime, we decided to replace it with imipenem.

On hospital day 10, body temperature of the patient returned to 36.5℃, and tenderness and rebound tenderness was diminished obviously. Ascitic fluid analysis showed WBC 5,184/mm^3 with 85% PMNL and 15% monocyte, but the bacterium was not found any longer in sediments of 5ml ascitic fluid.

On hospital day 13, abdominal tenderness and rebound tenderness were not observed any longer. However, ascitic fluid analysis revealed WBC 2,592/mm^3 with 83% PMNL and 15% monocyte. CBC analysis showed WBC count was 8,700/mm^3 and platelet 75,000/mm^3. Hemoglobin was 10.7g/dL and hematocrit 31%.

After hospital day 15, symptoms and signs of peritonitis disappeared completely, and ascitic fluid analysis revealed 240/mm^3 with 67% PMNL and 33% monocyte.

On hospital day 16, sudden onset of large amount of hematemesis was developed. Variceal rupture on the gastric cardiac portion was observed on the emergency endoscopy. Hematemesis and melena continued until hospital day 18. The patient and his family wanted to go home. So, on hospital day 19, the patient was hopelessly discharged.

Bacterial Characteristics

Cedecea davisae isolated was a motile, gram-negative rod, positive for lipase, DNase and catalase, and negative for gelatinase and oxidase. Bacterial characteristics tested with API 20E system, VITEK and several conventional laboratory tests were summarized as shown in Table 1.

Discussion

Until now, seventeen clinical isolates of the species

Table 1. Biochemical Chrarcteristics of *Cedecea davisae* Isolate

Tests	Result
Ortho-nitrophenyl-galactose	+(−)*
Arginine dihydrilase	+(−)*
Lysine decarboxylase	−
Ornithine decarboxylase	+
Citrate utilization	+
H_2S prodution	−
Urease	−
Tryptophan desaminase	−
Indole production	−
Voges-Proskauer	−
Gelatinase	−
Glucose(Oxidative)	+
Mannitol	+
Inositol	+(−)*
Sorbitol	−
Rhamnose	−
Sucrose	+
Melibiose	−
Amygdalin	+
Arabinose	−
Oxidase	−
DP 300	−
Acetamide	−
Esculine	−
Plant indican	+
Malonate	+
Polumyxin B	+
Lactose	−
Maltose	+
Xylose	+
Raffinose	−
Adonitol	−
p-Coumaric	+
Glucose(Fermentative)	+
DNase at 25℃	−
Lipase	+
Motility	+
Pigment production	−
Growth in media without thiamone	−

*Positive in API 20E system but negative in VITEK system

designated as *C. davisae* have previously been reported[1]. More than 50% of the clinical isolates derived from respiratory tract, especially from sputum. Other rare sources included a gall bladder, hand wounds, and an eye swab from a 4-day-old infant. In 1986, Perkins and co-workers[5] reported a case of bacteremia caused by *C. davisae*.

This is considered to be a first case in that *C. davisae* was isolated from ascitic fluid. In this case, obvious symptoms and signs of peritonitis were observed. No other bacteria capable of causing peritonitis were isolated. Moreover, no other etiological agent was recognized.

The possible transmission routes of the peritonitis are intestinal perforation, bacterial seeding via the lymph, blood or direct invasion through the weak bowel wall. In this case, bowel perforation was ruled out because there was no evidence of bowel perforation. Bacterial seeding could be excluded because blood culture was negative. *C. davisae* in this case is thought to be a nosocomial infection, possibly from his own flora.

In this report the result of susceptibility test to cefotaxime and the clinical course are inconsistent. First of all, the contrast was thought to be attributed to the difference between *in vitro* and *in vivo*. Secondly, as Perkins et al.[5] emphasized, *Cedecea davisae* may only be moderately sensitive to cefotaxime. These findings regarding *C. davisae* infection may be clinically useful.

Summary

복막염을 동반한 간경변증을 앓고 있는 49세 남자 환자의 복수로부터 *Cedecea davisae*가 분리되었다. 환자는 내원하기 15일 전부터 시작된 간경변증에 의한 복부팽창을 주소로 내원한 환자로서 입원후 복부팽창을 치료하던 중 고열, 복부압통과 반동압통 등의 복막염 증세를 보였으며 복수로부터 *C. davisae*가 분리되었다. 분리된 이 세균은 항생제 감수성 검사에서 cefotaxime, piperacillin과 imipenem에 감수성을 보였고 imipenem 투여 후 환자의 복막염은 효과적으로 치료되었다. 장내세균과에 속하는 이 세균에 의한 감염증은 매우 드물다. 이 보고는 복수로부터 *C. davisae*를 분리한 최초의 보고이다.

References

1) Farmer JJ Ⅲ, Davis BR, Hickman-Brenner FW, McWhorter A, Huntley-Carter GP, Asbury MA *et al.*: *Biochemical identification of new species and biogroups of Enterobacteriaceae isolated from clinical specimens. J*

Clin Microbiol 21:46-76, 1985

2) Farmer JJ III and Kelly MT : *Enterobacteriaceae. In: Barlows A, Hausler WJ Jr, Herrmann KL, Isenberg HD, Shadomy HJ, eds. Manual of Clinical Microbiology. 5th ed. p360, American Society for Microbiology, Washington D.C., U.S.A., 1991*
3) Grimont PAD, Grimont F, Farmer III JJ, Asbury MA : *Cedecea devisae gen. nov., sp. nov. and Cedecea lapagei sp. nov., new Enterobacteriaceae from clinical specimens. Int J Syst Bacteriol 31:317-326, 1981*
4) Holt JG, Krieg NR, Sneath PHA, Staley JT, Williams ST : *Bergey's manual of determinative bacteriology. 9th ed., 1994 The Williams & Wilkins Co., Baltimore, U.S.A.*
5) Perkins SR, Beckett TA, and Bump CM : *Cedecea davisae Bacteremia. J Clin Microbiol 24:675-676, 1986*