Sepsis Caused by Food-borne Infection with *Escherichia coli*

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Abstract

We report a case of sepsis caused by *Escherichia coli* (*E. coli*) of serotype O-143. A 78-year-old man developed symptoms of gastroenteritis after ingesting raw meat on noodles. He rapidly developed respiratory failure. Blood culture grew *E. coli*. The sepsis seemed to have directly spread from a food-borne infection. The development of primary sepsis after ingesting *E. coli* is very rare. We suspect that bacterial translocation played a major role. Serotype O-143 is recognized in enteroinvasive *E. coli* (EIEC) as well as in *Shigella dysenteriae*. The pathogen in the present case is suspected of being EIEC although the isolated *E. coli* strain was negative for the *inv* and *ipa* genes.

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Key words: *Escherichia coli*, O-143, sepsis, bacterial translocation, food poisoning

Introduction

Escherichia coli (*E. coli*) is a pathogen that commonly causes bacteremia. *E. coli*, unlike other diarrheal pathogens such as *Vibrio vulnificus* or *Salmonella* sp. (1, 2), rarely causes primary sepsis. Only one case of sepsis caused by ingesting *E. coli* has been reported (3). About 60% of *E. coli* bacteremia cases originate from urinary tract infection and 15% originate from infection of the biliary tract (4). The present patient had not suffered from urinary tract infection nor infection of the biliary tract. This patient developed sepsis after eating raw meat on noodles. His blood culture grew *E. coli*. This patient may represent a highly unusual clinical example of primary sepsis caused by *E. coli*.

Case Report

On August 11, 1998, a 78-year-old man was admitted to our hospital with high fever and dyspnea. He had been on vacation for five days in Vietnam and reported being well until the night prior to the admission. The patient had visited Ho Chi Minh, Vietnam, and he reported that he carefully avoided potentially contaminated foods. His last meal in Vietnam was, however, raw meat on noodles. During the flight to Japan on August 11, he developed vomiting and diarrhea, followed by a fever of 40°C with chills and dyspnea. The episode started about 10 hours after ingestion of the meal with raw meat. Upon arrival in Japan, he sought medical care and was immediately referred to our hospital for treatment. He reported a history of a half gastrectomy in 1959 due to a gastric ulcer, chronic hepatitis C since 1979, and brain infarction in 1995. He had no history of either drug or food allergies. He had no habit of drinking alcoholic beverages. On presentation, the patient was conscious and alert. His body temperature was 40°C, the systolic blood pressure was 78 mmHg on palpation, heart rate was 108/min, and respiration rate was 26/min. Coarse crackles were audible in the left lower lung field. There was no heart murmur. There were no remarkable findings on examination of the abdomen. The laboratory profile on admission showed hemoglobin 10.0 g/dl, platelets 126,000/μl, WBC 1,200/μl with left shift, albumin 2.7 g/dl, blood urea nitrogen 30 mg/dl, creatinine 1.34 mg/dl, aspartate aminotransferase (AST) 67 IU/l, CRP 1.6 mg/dl, erythrocyte sedimentation rate 21 mm/h, and FDP 36.8 μg/dl. Arterial blood gas analysis revealed respiratory alkalosis due to hypoxia with pH 7.458, PaCO₂ 31.1 mmHg, and PaO₂ 54.4 mmHg (Table 1). The endotoxin level was 78.8 pg/ml. He was positive for hepatitis C antibody. *E. coli* was cultured from arterial and venous blood, bronchial lavage, and gastric fluids. MIC testing showed that the strain of *E. coli* was sensitive to several antibiotics including cefepime (Table 2). Chest X-rays taken on admission showed diffuse infiltration in both lungs (Fig. 1).

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Table 1. Clinical Data on Admission

Hematology		Immunology	
RBC	405×10 ⁴ /μl	CRP	1.6 mg/dl
Hb	10.0 g/dl	IgA	202 U/ml
Plt	12.6×10 ³ /μl	IgM	108 U/ml
WBC	1,200/μl	IgG	2,773 U/ml
Meta	17%	HCV Ab	(+) COI 99.3%
Band	37%	HIV I/II Ab	(-)
Seg	4%		
Ly	35%		
ESR	21 mm/h	Coagulation	
		PT	81%
		HPT	52%
Chemistry		FDP	36.8 μg/dl
AST	67 IU/l	Fibrinogen	244 g/dl
ALT	29 IU/l		
LDH	396 IU/l	Urinalysis	
TP	6.2 g/dl	pH	7
Alb	2.7 g/dl	Protein	-
BUN	30 mg/dl	Sugar	-
Cr	1.34 mg/dl	Occult blood	3+
Na	34 mEq/l	RBC	3-5/1F
K	3.5 mEq/l	WBC	3-5/1F
Cl	100 mEq/l		
BS	175 mg/dl		
Endotoxin	78.8 pg/ml		

RBC: red blood cell, Hb: hemoglobin, Plt: platelet, WBC: white blood cell, ESR: erythrocyte sedimentation rate, AST: aspartate aminotransferase, ALT: alanine aminotransferase, LDH: lactate Dehydrogenase, TP: total protein, Alb: albumin, BUN: blood urea nitrogen, Cr: creatinine, Na: sodium, K: potassium, Cl: chlorine, BS: blood sugar, CRP: C-reactive protein, HCV: hepatitis C virus, HIV: human immunodeficiency virus, PT: prothrombin time, HPT: heparin test, FDP: fibrinogen degradation products.

Table 2. Susceptibility of the *E. coli* Isolated from the Present Patient to Various Chemotherapeutic Agents

Agent	Minimum inhibitory concentration (μg/ml)
PIPC	16
IPM/CS	≤1
CFPM	1.56
CPR	≤1
CZOP	≤1
MINO	≤1

PIPC: piperacillin, IPM/CS: imipenem, CFPM: cefepime, CPR: ceftazidime, CZOP: ceftazidime, MINO: minocycline.

The cardio-thoracic ratio was 51%. A computed tomographic scan of the chest, taken on the day of admission, showed a diffuse ground glass appearance and air space consolidation (Fig. 2). The degree of the infiltration worsened progressively on the second day of admission. Pulmonary artery wedge pressure was within the normal range. We made the

diagnosis of acute respiratory distress syndrome accompanied by decreased WBC with left shift. The HIV antibody was negative. The CD4/CD8 was normal. Erythromycin, cefepime and gamma globulin were administered intravenously. At the same time, oxygen was given by mask. However, his hypoxia worsened by the evening and he was intubated and started on mechanical ventilation. *E. coli* were identified using the VITEK 2 system (BioMerieux Corporation, Tokyo, Japan) and the subgroup was determined using an *E. coli* antisera kit (Denka Seiken Co., Ltd., Tokyo, Japan). The *E. coli* was positive for O-143 antigen, and was suspected of being enteroinvasive *E. coli* (EIEC). We also performed polymerase chain reaction. However, the isolated *E. coli* was negative for the *inv* and *ipa* genes, which encode proteins involved in its invasiveness. The *E. coli* was not tested for the *sen* gene. He had loose stools almost every day. The stools were sent for routine culture. The results showed normal flora. The stools were not tested for O-antigens of *E. coli*. As soon as we made the diagnosis of bacteremia caused by *E. coli*, adsorption therapy of endotoxin was started. Continuous hemodiafiltration was

Figure 1. Chest X-ray taken on admission shows diffuse infiltration in both lungs.

Figure 2. High resolution computed tomographic scan of the chest, taken on the day of admission shows a diffuse ground glass appearance and air space consolidation.

performed for four days. As a result of this intensive treatment, blood cultures and other culture specimens became negative for *E. coli* by August 13, indicating that the antibiotics we had chosen were effective. Despite treatment, the hypoxia persisted and the patient died on August 23 (Fig. 3).

Discussion

The present case of sepsis is unusual because it was caused by *E. coli* of serotype O-143 and it appears to have caused bacteremia via an intestinal infection. *E. coli* is one of the most frequent isolates from patients with sepsis (1). However, most cases of sepsis that are caused by *E. coli*, occur from urinary tract infection and cholangitis. Some cases of sepsis occur as a result of manipulation such as urinary catheterization. Simple intestinal *E. coli* infection rarely causes sepsis (6).

We suspect the pathogen in the present case to be EIEC, since among all *E. coli* strains, serotype O-143 is known only as enteroinvasive *E. coli* (1). In order to find genes involved in its invasiveness, we performed polymerase chain reaction for the two most common genes involved in the invasiveness of *E. coli*. However, the *E. coli* strain isolated from our patient was negative for these two genes, and further studies should be conducted.

In addition to transmission to the blood by the previously described routes, *E. coli* are known to translocate easily (7). Translocation is defined by Berg and Garlington (8) as the passage of viable bacteria from the gastrointestinal tract to

the mesenteric lymph nodes and possibly other organs. Translocated *E. coli* does not always result in sepsis. Instead, it tends to result in portal septicemia, which is subsequently cleared by Kupffer cells (8). Since our patient had neither urinary tract infection nor cholangitis, septicemia seemed to have developed after an intestinal infection. The cause of the intestinal infection in our patient is assumed to be the raw beef since the first symptoms appeared 10 hours after ingestion (9, 10). Septic symptoms such as high fever and chills, appeared after the initial intestinal symptoms. After the patient was admitted, *E. coli* was isolated from the arterial blood, venous blood, gastric aspirate and pulmonary lavage. These findings are consistent with systemic infection of *E. coli* which occurs immediately after intestinal infection.

The development of sepsis following intestinal infection with *Klebsiella pneumoniae* and *E. coli* has been reported (3). In that report, a 28-year-old, previously healthy male ate a hamburger and developed symptoms of gastroenteritis, which rapidly deteriorated to multi-organ failure. Blood and hamburger cultures grew *K. pneumoniae* and *E. coli*. This previous case indicates that sepsis can indeed follow an intestinal infection in a healthy person. The pH range in which *E. coli* can grow is between 6.0 and 8.0, and some growth is possible at pH values of approximately 1.0 above and below this range (11). The physiological condition in our patient as a result of the half gastrectomy, such as high pH, could have been favorable for the survival of *E. coli* of serotype O-143 (11) and the subsequent severe intestinal infection. The number of *E. coli* organisms ingested by our patient as well as his

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Figure 3. Clinical course of the patient after hospitalization.

age and fatigue from traveling may have facilitated the activity of this organism. Our observations indicate the importance of monitoring for sepsis in cases of severe intestinal *E. coli* infection.

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