Endogenous Endophthalmitis Due to Escherichia coli: Case Report and Review

Endogenous endophthalmitis is a rare complication of Escherichia coli septicemia in the antibiotic era. Emphysematous endophthalmitis due to E. coli is even more uncommon; only one case in a diabetic patient has been reported previously [1]. Herein, we describe a case of endogenous emphysematous endophthalmitis due to E. coli in a patient with urinary tract infection and endocarditis who did not have any medical history of diabetes mellitus.

A 50-year-old man was hospitalized on 2 April 1994 because of a 10-day history of fever, chills, and ocular pain followed by loss of vision in both eyes. He had no history of ocular trauma or surgery. In 1993, he had undergone aortic valvuloplasty because of aortic stenosis. Three years before hospitalization, he had also had a left kidney stone and had received extracorporeal shock wave lithotripsy. He had no medical history of diabetes mellitus.

Ten days before his admission, the patient had high fever, chills, and ocular pain. Erythematous change in both eyes was noted, and he complained of bilateral visual impairment during the following 2 days. He was treated with intravenous antibiotics (including amikacin) in a local medical clinic for the presumptive diagnosis of sepseis and endophthalmitis. A series of studies were done. Urinalysis showed pyuria, and cultures of urine and blood both yielded E. coli. He was later transferred to Taichung Veterans General Hospital (Taichung, Taiwan, ROC) because of deterioration of his clinical condition.

At the time of admission, he was acutely ill-looking and drowsy. The temperature was 37°C, the blood pressure was 110/60 mm Hg, and the pulse rate was 76. Physical examination disclosed a grade 3/6 systolic murmur; the murmur was heard best at the aortic area and left sternal border, and the sound transmitted to the neck. Ophthalmologic examination showed bilateral periorbital swelling and moderate injected conjunctivae. Vision in both eyes was only light perception. The cornea of both eyes had an epithelial defect with superficial stromal haziness of the lower portion. Exudate and fibrinous material were present in the bilateral anterior chambers, and there was mild opacity in the lenses of both eyes. The vitreous and fundi of both eyes could not be visualized. The other findings of the physical examination were unremarkable.

A CT of the orbits demonstrated a gas bubble in the left eye globe, a finding indicating emphysematous endophthalmitis. The chest roentgenogram obtained on admission did not show any abnormalities. Echocardiographic examination of the heart revealed a vegetation over the tricuspid septal leaflet, with aortic stenosis, aortic regurgitation, and mild pericardial effusion. An abdominal sonogram demonstrated left renal stones with cysts over both kidneys. Laboratory studies disclosed the following: WBC count, 12,980/mm³ (93% polymorphonuclear leukocytes and 7% lymphocytes); hemoglobin concentration, 10.3 g/L; and plasma glucose level, 115 mg/dL. Although many WBCs were found in the urine, no cultures of blood or urine yielded bacteria. An electrocardiogram showed atrial flutter with frequent premature ventricular beats.

An intravitreous injection of gentamicin (0.1 mg) was given bilaterally. Fortified gentamicin and cefazolin eye drops were then applied every 2 hours. Systemic therapy with intravenous oxacillin (2 g every 4 hours) and cefotaxime (2 g every 6 hours) was also given empirically. Culture of the vitreous aspirate later yielded E. coli that was susceptible to cefotaxime and gentamicin. Despite this aggressive therapy, his visual acuity deteriorated rapidly, and finally he lost his bilateral vision completely. During his hospitalization, atrial flutter or fibrillation with frequent premature ventricular beats persisted. Metabolic acidosis was found, and the patient died of severe sepsis 2 days after his admission.

Endogenous endophthalmitis due to E. coli is not a common disease. We reviewed the English-language literature and found 13 cases (including this case) with detailed data since 1930 [1–10]. Eleven patients had underlying diabetes mellitus or hyperglycemia. The primary source was urinary tract infection in 10 cases. Although aggressive therapy was given, the visual prognosis for all these patients was poor. E. coli emphysematous endophthalmitis is a very rare and severe variety of E. coli endophthalmitis; only one case in a diabetic patient has been reported previously [1]. Faraawi and Fong [1] postulated that a high concentration of glucose in the eye tissue may provide a substrate that bacteria can ferment to produce carbon dioxide and hydrogen. To our knowledge, the case described herein is the first report of emphysematous endophthalmitis in a patient without any medical history of diabetes mellitus.

The mechanism of emphysematous endophthalmitis in this case is unknown. However, E. coli is a gas-producing bacterium that ferments glucose, and a high inoculum of E. coli itself may produce an emphysematous change in infected tissue. Our patient had concurrent endophthalmitis and infective endocarditis. It is also rare that endophthalmitis and endocarditis occur simultaneously during the course of E. coli septicemia, and only three cases (including this case) have been reported previously [10].

In conclusion, we stress that E. coli endogenous endophthalmitis, especially the emphysematous type, is rare, but the progression of the disease is rapid. It is frequently caused by urinary tract infection, but infective endocarditis and other foci of infection are other possible sources. Most of the cases are associated with underlying diabetes mellitus, but there are exceptions. The visual prognosis of patients with E. coli endophthalmitis is grim. Death is a possible outcome if endophthalmitis is associated with severe systemic infection. Physicians should be alert that endophthalmitis is a sign of systemic infection, investigate the primary source, and administer treatment as early as possible.

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Cholera Among Americans Living in Peru

Cholera rarely occurred in North Americans before the recent Latin American epidemic. Only 10 cases were identified from 1960 to 1989, for an estimated rate of 1.7 per million travelers from the United States [1]. A European study identified 40 cases from 1975 to 1981, for an estimated rate of 2 cases per million travelers [2]. After a 100-year-long absence of cholera in the Americas, *Vibrio cholerae* biotype El Tor appeared in Peru in early 1991 [3]. In 1991 and 1992, >700,000 cases of cholera were reported in the Americas, ranging from Peru to Mexico [4]. A major increase in the number of case reports was also noted in the United States, where 26 imported cases were reported in 1991, and 102 were reported in 1992 [4,5].

The incidence of cholera, especially disease due to *V. cholerae* biotype El Tor, may be underestimated among Westerners because the illness can be mild compared to that in patients who are infected with the classical biotype and may be mistaken for traveler’s diarrhea, a common illness in developing countries [6]. Culture techniques are usually not available for diagnosis, and they are often not useful because patients with suspected traveler’s diarrhea have taken antibiotics for self-medication. We determined the rate of cholera among the U.S. Embassy employees in Peru; patients were seen at a clinic where fecal specimens could be cultured at the time of illness.

From 1991 to 1993, U.S. Embassy employees were asked to submit a stool specimen when they presented to the Embassy Health Clinic with diarrhea. Specimens were plated on thiosulfate citrate bile salts sucrose (TCBS) agar before and after enrichment in alkaline peptone water. Typical colonies were identified biochemically and confirmed as *V. cholerae* O1 by use of O group antisera. An employee census was obtained from the Personnel Office of the U.S. Embassy in Lima. Cholera case rates for Peru were obtained from the Office of General Epidemiology, Ministry of Health, Lima. The number of foreign tourists was obtained from the National Institute of Statistics and Information in Lima [7].

From 1991 to 1993, *V. cholerae* O1 was isolated from 8 (1.3%) of 620 patients submitting stool specimens, with a peak isolation rate of 2.2% in 1992. All cholera cases were in adults (five men and three women) who were infected in the summer months (November to April). Five cases occurred in U.S. citizens, and three occurred in Peruvian employees. The illness was characterized as moderate to severe diarrhea that responded to oral rehydration and treatment with doxycycline. None of the patients required hospitalization or intravenous rehydration. Three of the five Americans attributed their illness to ingestion of poorly prepared food while at the beach near Lima. An average of 317 U.S. citizens and 427 Peruvians were employed at the U.S. Embassy during the study. The incidence of cholera was 2.5 cases per 1,000 employees in 1991, 6.6 cases per 1,000 in 1992, and 1.4 per 1,000 in 1993 (average annual incidence, 3.6 per 1,000).

The incidence was higher among U.S. citizens (5.3 per 1,000) than among Peruvian employees (2.3 per 1,000). An incidence of cholera of 5.3 cases per 1,000 per year or 44 cases per 100,000 per month among U.S. citizens living in Lima is unexpectedly high in comparison to that described in reports on travelers returning to the United States or Europe (table 1). *V. cholerae* is likely transmitted to travelers and expatriates through contaminated food [8]. The expatriates in Lima reported that eating poorly prepared food was the most likely source of their infection. Diarrhea rates are generally lower among expatriates than among tourists because the expatriates eat fewer meals in restaurants. However, because expatriates are exposed to disease for longer periods, we estimate that the risk of cholera for travelers and expatriates is comparable. In the cases involving U.S. Embassy personnel, the patients presented with a rather severe form of traveler’s diarrhea, without distinguishing features such as severe dehydration requiring intravenous rehydration or hospitalization. Cholera was not distinguished as a separate disease from traveler’s diarrhea. If the cholera rates observed for expatriates were similar to those for tourists, there could have been 308 cholera cases (including 71 North Americans and 89 Europeans) among the 700,000 foreign tourists who visited Peru from 1991 to 1993. Few of these cases were actually detected in the tourists’ home countries because most of them recovered before returning home, or cultures were not performed [8,9].

Stool samples from returning travelers with diarrhea are rarely cultured, and it is even more rare that media such as TCBS are used to isolate *V. cholerae*. When specific methods for isolating *V. cholerae* are used, as was in the case for Japanese travelers with diarrhea who were returning from Thailand or Indonesia, cholera rates ranged from 5 per 100,000 for those returning from Thailand to 13 per 100,000 for those returning from Indonesia, principally Bali [9]. These rates suggest that cholera may be underreported in Westerners traveling to Asia as well as to Latin America.