Role of *Chryseobacterium indologenes* Infection in a Female with Mediastinitis

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Abstract

Background: *Chryseobacterium indologenes* is an uncommon human pathogen that has been rarely reported in the literature as a cause of infection, but despite low virulence, this opportunistic bacteria is resistant to many antimicrobial agents.

Case Presentation: We present the first case in which *C. indologenes* was isolated from a 67-year-old female who was admitted urgently to the cardiosurgical area of the hospital for mediastinitis after rehabilitation at another hospital. Despite an unclear history, *Chryseobacterium* spp. was identified after *ab ingestis* pneumonia. The strain identified was resistant to several antibiotic classes. Trimethoprim-sulfamethoxazole was the only active antibiotic. Despite the pharmacologic support, septic shock was unpreventable. The patient died after 57 days of hospitalization.

Conclusion: The management of *C. indologenes* as a pathogen causing infection in a cardiosurgical area requires better screening and monitoring because immunocompromised patients often have lengthy periods of hospitalization.

CHRYSEOBACTERIUM INDOLOGENES causes severe nosocomial infections, especially in immunocompromised, critically ill patients [1]. Prolonged catheterization and invasive interventions also have been identified as risk factors [1]. Infections due to *Chryseobacterium* spp. occurs among the elderly (>65 years old) with nosocomial pneumonia and bacteraemia, sometimes associated with indwelling devices (feeding tubes, intravascular catheter, surgical drainage) [2], urinary tract infections [3], biliary tract infection, peritonitis, surgical site infection, and cellulitis [4]. In the hospital environment, this bacteria is found frequently on wet surfaces and in water systems by virtue of its ability to contaminate and persist in fluid-containing apparatuses [5,6].

Case Presentation

We report a case of a 67-year-old female admitted to the cardiosurgical department for an aortic valve surgical re-

placement because of a severe aortic valve stenosis at the expense of internal carotid artery. A 21 Carpentier-Edwards perimount bioprosthesis (Edwards Lifesciences, Irvine, CA) was implanted. Following a regular post-operative period, the patient was transferred for a period of five days to another hospital for rehabilitation.

After this period, the patient was admitted again to the cardiosurgical department with a deterioration in her clinical condition; specifically, she had difficulty breathing and a wound infection was reported. The presence of a pericardial effusion, with minimal presence of mitral insufficiency was reported by a trans-thoracic ultrasound. Despite empiric antibiotic therapy (piperacillin-tazobactam and vancomycin), the biochemical parameters showed no signs of improvement; C reactive protein (CRP) was elevated at 309 mg/L and white blood cells (WBC) count was 18,000/mm³. The tracheal aspirates and mediastinal drainage samples were positive for methicillin-resistant *Staphylococcus aureus* (MRSA).

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FIG. 1. Surgical revision of the mediastinum (right): Complete opening of the sternum injury highlighted a purulent mediastinitis (left) with diaphragmatic and pericardial material production.

Mediastinitis was diagnosed clinically and a negative pressure wound therapy (NPWT) was introduced. The rapid evolution of the patient's clinical conditions led to a severe septic shock because of MRSA several days later. Antibiotic therapy was changed to daptomycin and meropenem.

The patient's clinical conditions did not improve (CRP: 256 mg/L, WBC 17,000/mm³), thus a surgical revision of the mediastinum was performed (Fig. 1). A complete opening of the sternum injury highlighted a purulent mediastinitis with diaphragmatic and pericardial material collection. The patient was supported medically with coping with septic shock. Slow respiratory weaning was attempted, but on the 44th day of hospitalization during a phase of respiratory recovery, the patient had an accidental inhalation that caused aspiration pneumonia in the right lower lobe. By radiographic survey a minimal amount of basal deposit with lower lobe consolidation was detected (Fig. 2). Forty-six days after her admission, the respiratory sample and blood cultures showed Gram negative bacilli identified as *C. indologenes* by the automatic analyser Vitek 2 System (BioMérieux, Marcy l'Etoile, France), and sequencing analysis confirmed bacterial identification.

The close communication between surgeons, microbiologists, and infectivologists led to a further modification of the antibiotic regimen consisting of levofloxacin, cotrimoxazole, and metronidazole. *Chryseobacterium indologenes* is resistant intrinsically to aminoglycosides, most β -lactams, chloramphenicol, linezolid, and glycopeptides, and susceptible to levofloxacin and piperacillin-tazobactam [2]. In our case, trimethoprim-sulfamethoxazole (MIC <20 μ g/ml) was the only active agent [7]. A further microbiological investigation showed a mechanism of resistance due to the AmpC activity and a porine loss production. On the 53rd hospitalization day a new episode of septic shock occurred, thus, the patient underwent a continuous venovenous hemodiafiltration (CVVHDF) to reduce the bacterial load, which was in vain.

Since 2014, in the cardiosurgical department there has been an active hospital acquired infection surveillance system to determine and elucidate putative transmission events between patients and operators. Molecular investigation of the MRSA strain, sequenced and compared with the other collected specimens in the unit, did not document any correlation between our case and other patients and operators, suggesting that MRSA isolated has not been contracted in our hospital.

The patient died after 57 days of hospitalization. Post-mortem examination revealed chronic fibrinous pericarditis and signs of multi-organ failure associated with foci of necrosis compatible with septic shock (Fig. 3).

Discussion and Literature Review

Even if *C. indologenes* is an environmental organism and opportunistic pathogen usually associated with nosocomial or device-related infections [1], we can only surmise that it had a role in the clinical status of the patient. As reported by Chou [1] most of the intensive care unit (ICU) patients had received more than 14 days of a broad-spectrum antibiotics, which might have led to selective pressure of this pathogen.

Chryseobacterium indologenes had not been reported as a cause of bacteremia in human beings until 1996 [1,3], but since that report, there have been relatively few studies [6] and most of them occurred in Taiwan [1,3]. Only two cases reported in the literature were in Italy [2]. We do not know if septic shock was because of the presence of *C. indologenes* because we are aware that MRSA was eradicated with appropriate antibiotic therapy; however, the

FIG. 2. Radiographic survey performed at the 44th day of hospitalization revealed a minimal amount of basal deposit with lower lobe consolidation (right) not present in previous radiographic images (left).

FIG. 3. Post-mortem examination revealed a chronic fibrinous pericarditis and signs of multi-organ failure associated with foci of necrosis compatible with septic shock.

clinical condition of the patient did not improve despite the antibiotic treatment.

Conclusion

More clinical evidence is required to elucidate the acquisition of *C. indologenes* and to develop efficacious preventive measures, especially because this pathogen occurred simultaneously with polymicrobial bacteremia [8]. The management of *C. indologenes* as a pathogen causing infection in a cardiosurgical area needs better screening and monitoring, especially because empiric treatment of infections because of *C. indologenes* is difficult because of limited published data.

Author Disclosure Statement

All of the authors declare that they have no competing interests.

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Abbreviations Used

CRP = C reactive protein
CVVHDF = continuous venovenous hemodiafiltration
ICU = intensive care unit
MRSA = methicillin-resistant *Staphylococcus aureus*
NPWT = negative pressure wound therapy
WBC = white blood cells