Res Cardiovasc Med. 2014 August; 3(3): e19432. DOI: 10.5812/cardiovascmed.19432

Published online 2014 August 15. **Research Article**

Bacteriologic Profile of Pericardial Infections After Cardiac Surgery: Study in an Iranian Cardiovascular Tertiary Care Center

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Received: April 12, 2014; **Revised:** June 16, 2014; **Accepted:** August 25, 2014

Background: Bacterial pericarditis is an important cause of post-surgery mortality and morbidity. This can be a preventable complication and the involved pathogens vary according to the time and location.

Objectives: The aim of this study was to investigate the bacteriologic profile in patients with pericardial infections after cardiac surgery in the largest tertiary care center for cardiovascular diseases in Iran. The results can be applied for prevention, diagnosis, and treatment of similar patients in Iran.

Patients and Methods: This prospective study was performed in Rajaie Cardiovascular Medical and Research Center (RCMRC), the largest tertiary care center for cardiovascular disease in Iran from March 2011 to March 2012. Patients who had undergone cardiac surgery with cardiopulmonary bypass and showed suggestive sign and symptoms of pericardial infections were registered and samples from their pericardial fluids were obtained to perform standard bacteriologic and antibiogram tests.

Results: A total of 158 patients were registered. Bacteriologic findings were positive in 30 patients (19%). *Staphylococcus epidermidis* was the most frequent isolated organism, which was found in 22 patients (73.3%) with eight of them being methicillin-resistant strains.

Conclusions: The bacteriologic profile in our patient is specific to our own community. Knowledge about this profile can help us to improve prevention, diagnosis, and treatment of the affected patients.

Keywords: Microbial Sensitivity Test; Methicillin-Resistant Staphylococcus Aureus; Drug Resistance, Microbial; Pericardial Effusion; Cardiac Surgical Procedures; Adverse Effects

1. Background

Bacterial pericarditis is defined as the swelling and irritation of pericardium, which is caused by infection with various kinds of bacteria. As a result, the inflamed pericardium rubs against the heart and the patient may feel pain with fluid accumulating in the pericardial sac. Since the introduction of antibiotics, bacterial pericarditis has become rare. Pericarditis occurs most often in men between 20 and 50 years of age, usually following respiratory infections. It is also seen after heart surgery as well as skin or oral infections that produce bacteremia. Diagnosis is made when the bacteria is detected in pericardial fluid. Despite the recent improvements in the intraoperative management and postoperative care, late-onset pericardial effusions are an important cause of morbidity following heart surgery. Pericardial effusions may adversely affect the recovery phase and become life-threatening once tamponade leads to hemodynamic compromise (1, 2). Because of the recent and widespread use of anticoagulant medications as well as the increasing complexity of operations, the incidence of such effusions might be higher (3, 4). Therefore, it is important to have enough information on the manifestations, risk factors, and natural history of pericardial effusion in order to develop better protocols for its prevention, diagnosis, and treatment.

2. Objectives

The aim of this study was to determine the bacteriologic profile in patients with pericardial infections after cardiac surgery and in the course of their treatment. The results could be applied for prevention, diagnosis, and treatment in similar patients in Iran.

3. Patients and Methods

3.1. Study Participants

This prospective study was performed on the patients who underwent cardiac surgery with cardiopulmonary bypass at Rajaie Cardiovascular Medical and Research Center (RCMRC), the largest tertiary care center for cardiovascular disease in Tehran, IR Iran, from March 2011 to March 2012. A total of 158 patients with clinical, laboratory, and echocardiographic suggestive findings of pericar-

ditis (1) were registered and undergone the bacteriologic investigations. The mean age of patients was 70 ± 14 years (range, 5 months to 100 years) and majority of them were females (116 [73.3%]). The study protocol was approved by Research Board and Ethics Committee of RCMRC.

3.2. Smear Preparation and Culture of the Samples

A single drop of the fluid was placed on a glass microscopic slide to form a smear. Gram's staining method was then performed to impart appropriate staining qualities to different microorganisms that might be found in the smear of the fluid. A laboratory specialist examined the stained slide under the microscope for the presence of bacteria. The organisms were identified based on their color, size, and shape (4). By definition, infectious pericarditis is manifested by positive gram staining and fluid culture results with the use of conventional microbiologic methods. We have benefitted from the same policy. Fluid culture studies were done according to standard procedures. Two blood agar media were inoculated; one for anaerobic and another one for aerobic microorganisms. One chocolate agar medium (incubated in Candle jar), one MacConkey agar, and thioglycollate broth (as enrichment medium) were also used. All the inoculated plates were assessed after incubation for 24 hours. The plates that were negative for any microbial growth were further incubated for another 24 hours and subculture from an enrichment medium was performed to detect fastidious and slow-growing bacteria in the samples.

3.3. Identification of Positive Cases

Bacteria that belong to the *Staphylococcus* and *Streptococcus* genera and Enterobacteriaceae family were identified according to the standard methods. *Staphylococcus* species were tested for coagulase positivity using rabbit plasma. DNase activity was also checked by means of DNase agar. Coagulase-negative and DNase-negative isolates were identified further by sucrose fermentation, ornithine decarboxylation, and urea hydrolysis. DNase-positive and coagulase-positive isolates were identified as *Staphylococcus aureus*.

3.4. Antibiotic Susceptibility Testing

Antibiotic susceptibility testing of all the isolates was performed according to the Kirby-Bauer disk diffusion method, using Mueller-Hinton agar (MHA). The following antibiotic disks were used (provided by Hi-media, Padtan teb, ABTEK, ROSCO): penicillin (10 U), ciprofloxacin (5 μg), amikacin (30 μg), gentamycin (10 μg), tobramycin (10 μg), ceftriaxone (30 μg), cefepime (30 μg), imipenem (10 μg), ampicillin (10 μg), amoxicillin/clavulanate (30 μg), cefoxitin (30 μg), cefazolin (30 μg), and meropenem (10 μg). Cefoxitin was used to determine the methicillin-resistant *S. aureus* (MRSA) strains.

4. Results

4.1. Bacterial isolates

A total of 158 samples were assessed in which only 30 cases (19%) had positive results for bacteria. Only two major species of microorganisms were isolated, namely *Staphylococcus* and *Streptococcus*. Of the 30 positive cases, *Staphylococcus* species were present in 27 specimens (90%). In this group, 26 isolates were coagulase negative and only one *S. aureus* isolate was found. The remaining three positive cases (10%) were from the *Streptococcus* species all of which were found to be *Streptococci viridans* (Table 1).

Table 1. Isolates of Different Species in Pericardial Fluid of Patients

Isolated Organisms	No. (%)
Staphylococcus epidermidis	22 (73.3)
Staphylococcusschleiferi	2 (6.6)
Staphylococcus haemolyticus	1 (3.3)
Staphylococcus aureus	1 (3.3)
Staphylococcuswarneri	1 (3.3)
Streptococcus viridans	3 (3.3)

Table 2. Antibiotic Sensitivity Pattern of Isolated Organisms [a,b]

	Staphylococcus aureus (n = 1)	*Staphylococcusepidermidis* (n = 22)	*Staphylococcuswarneri* (n = 1)	*Staphylococcushaemolyticus* (n = 1)	*Staphylococcusschleiferi* (n = 1)	*Streptococcus viridans* (n = 2)
Amikacin	1	17	0	1	1	0
Cefoxitin	1	0	0	0	1	0
Ampicillin	0	0	0	0	0	2
Tobramycin	1	17	0	1	1	0
Ceftriaxone	1	15	0	1	1	2
Ciprofloxacin	1	18	0	1	1	2
Meropenem	0	19	0	1	1	2
Penicillin	0	0	0	0	0	2
Gentamycin	1	17	0	1	1	0
Cefepime	1	18	0	1	1	2
TMP/SMX	1	11	0	1	1	0
Imipenem	1	19	0	1	1	2

[a] Abbreviation: TMP/SMX, Trimethoprim/Sulfamethoxazole (co-trimoxazole).
[b] Data are presented as No.

5. Discussion

Bacterial infections of pericardium are relatively uncommon; however, they are much more likely to form purulent effusions and to proceed to cardiac tamponade or pericardial constriction (5, 6). Purulent pericarditis is almost exclusively seen as a secondary infection in patients with serious underlying diseases such as AIDS and those undergoing hemodialysis, thoracic surgery, and chemotherapy (7, 8). It is not typically a primary infection but rather almost exclusively a complication of an underlying infection. In the pre-antibiotic era, patients most often showed bacterial pericarditis after pneumonia with empyema, and the most common germ was *Streptococcus pneumoniae* (9). In the antibiotic era, the most common cause is *S. aureus* (10). Recent studies have revealed a trend toward diverse bacteria; in addition, anaerobes have been reported as common causes of such infections (11, 12). On blood testing, patients with pericarditis show evidence of systemic inflammation such as leukocytosis and increased erythrocyte sedimentation rate (ESR). It can be technically difficult to obtain a sample from fluid or tissue aseptically, especially during an ongoing surgical procedure in an infected tissue. When dealing with organisms that belong to the normal skin flora, the question of contamination always arises (13, 14). In most cases, there are sufficient numbers of bacteria in the infected tissue that can be detected in primary cultures. We detected only one case of *S. aureus* infection from the primary cultures. This made it possible to report culturing results (i.e. bacteria and antibiogram) to the clinician faster than with the traditional method, that is, subculturing only from enrichment broth. In rare cases, subculturing from enrichment broth on day four was necessary to identify infection with coagulase-negative *Staphylococcus* (CNS) species microbiologically. In our study group that consisted of 30 infected patients with positive culture results, 20 (66.6%) were clinically considered to have an infection and 10 (33.3%) were considered otherwise. These paraclinical findings included leukocytosis (white blood cell > 10000/mm^2) and an elevated ESR (> 40 mm/h). Adding a microbiologic standard for infection could help identify infected patients among those lacking obvious visible signs of infection. The probability for agreement between diagnosis of infection based on clinical signs and symptoms and diagnosis based on our microbiologic criterion was significant for all infected cases, which were mostly infected with CNS species. Our findings were different from what the other studies showed (15). In other words, our study revealed that, infections with CNS species could be identified by general signs and symptoms of infection as well. Infection with *S. viridans* is rarely found in purulent pericarditis. In addition, it is usually combined with other micro-organisms (11). The clinical course of infection with *S. viridans* is usually subacute or chronic, with low or absence of toxic signs (16). The common sources of purulent pericarditis with *S. viridans* include mediastinitis from esophageal rupture, dental caries, retropharyngeal abscess, infective endocarditis, chest surgery, trauma, and pneumonia (9, 10). The possible source in our case might be related to dental procedures according to the history. The majority of the CNS species that cause pericarditis have been reported as methicillin resistant during the last decade (13). The results demonstrated that eight of the CNS isolates were methicillin resistant. We did not have as many such cases as was reported Mossad et al. and Tegnell et al. who found methicillin-resistant CNS isolates in 73% and 92% of studied cases, respectively (13, 17). Another retrospective study from 1984 to 1995 in the same setting, found that 69% of CNS isolates were methicillin resistant (18). As this resistance limits the therapeutic options, it is important not to overestimate the proportion of methicillin-resistant strains or the number of infected patients. To diagnose a postoperative pericarditis caused by staphylococci correctly, the susceptibility pattern to methicillin in the infective strain is essential, as the methicillin-sensitive strains can be treated with isoxazolyl penicillins and cephalosporins. We also found that most of the isolated CNS strains were sensitive to cephalosporins. An overestimation of methicillin resistance might lead to unnecessary use of vancomycin (15), which should be avoided due to the risk for selection of genes coding for vancomycin resistance (19). Moreover, it would consider the interest of the patient who might otherwise undergo a long-term course of intravenously administered antibiotic treatment. Purulent pericarditis is a potential lethal disease and should be considered as an indolent underlying disease of cardiac tamponade. A stat Gram staining as well as other pericardial effusion analysis should be performed in every patient with cardiac tamponade. Prompt percutaneous catheter drainage of pericardial fluid along with appropriate antibiotic therapy according to bedside Gram staining can rapidly terminate the life-threatening condition associated with purulent pericarditis and cardiac tamponade.

Acknowledgements

The authors would like to thank the staff members of surgery wards of Rajaie Cardiovascular Medical and Research Center for their kind collaboration in data acquisition.

Authors' Contributions

Kambiz Mozaffari performed the data collection and wrote the manuscript. Hooman Bakhshandeh performed the consultation, collaborated in writing the manuscript, and revised the manuscript. Hengameh Soudi cooperated in data collection and writing the manuscript.

Funding/ Support

This research project was financially supported by Rajaie Cardiovascular Medical and Research Center.

References

1. LeWinter M. *Pericardial diseases*. 8. Braunwald's Heart Disease editor. Philadelphia: Saunders Elsevier; 2007.
2. Price S, Prout J, Jaggar SI, Gibson DG, Pepper JR. 'Tamponade'

following cardiac surgery: terminology and echocardiography may both mislead. *Eur J Cardiothorac Surg.* 2004;**26**(6):1156–60.
3. Kulik A, Rubens FD, Wells PS, Kearon C, Mesana TG, van Berkom J, et al. Early postoperative anticoagulation after mechanical valve replacement: a systematic review. *Ann Thorac Surg.* 2006;**81**(2):770–81.
4. Little W, Oh J. *Pericardial diseases*.Philadelphia: Saunders Elsevier; 2011.
5. Little WC, Freeman GL. Pericardial disease. *Circulation.* 2006; **113**(12):1622–32.
6. LeWinter M, Kabbani S. *Pericardial disease.* Braunwald's heart disease editor. Philadelphia: Saunders; 2005.
7. Pankuweit S, Ristic AD, Seferovic PM, Maisch B. Bacterial pericarditis: diagnosis and management. *Am J Cardiovasc Drugs.* 2005;**5**(2):103–12.
8. Keersmaekers T, Elshot SR, Sergeant PT. Primary bacterial pericarditis. *Acta Cardiol.* 2002;**57**(5):387–9.
9. Klacsmann PG, Bulkley BH, Hutchins GM. The changed spectrum of purulent pericarditis. *Americ J Med.* 1977;**63**(5):666–73.
10. Rubin RH, Moellering RC. Clinical, microbiologic and therapeutic aspects of purulent pericarditis. *Americ J Med.* 1975;**59**(1):68–78.
11. Brook I. Microbiology of Acute Purulent Pericarditis. *Arch Int Med.* 1996;**156**(16):1857.
12. Brook I. Pericarditis due to anaerobic bacteria. *Cardiology.* 2002;**97**(2):55–8.
13. Mossad MD, Sherif B, Serkey RNJD, Janet M, Longworth MD, David L, et al. Coagulase-Negative Staphylococcal Sternal Wound Infections After Open Heart Operations. *Anna Thorac Surg.* 1997;**63**(2):395–401.
14. Ottino G, De Paulis R, Pansini S, Rocca G, Tallone MV, Comoglio C, et al. Major Sternal Wound Infection after Open-Heart Surgery: A Multivariate Analysis of Risk Factors in 2,579 Consecutive Operative Procedures. *Annal Thorac Surge.* 1987;**44**(2):173–9.
15. Tammelin A, Hambraeus A, Stahle E. Mediastinitis after cardiac surgery: improvement of bacteriological diagnosis by use of multiple tissue samples and strain typing. *J Clin Microbiol.* 2002;**40**(8):2936–41.
16. Mylonakis E, Calderwood SB. Infective endocarditis in adults. *N Engl J Med.* 2001;**345**(18):1318–30.
17. Tegnell A, Arén C, Öhman L. Coagulase-negative staphylococci and sternal infections after cardiac operation. *Anna Thorac Surg.* 2000;**69**(4):1104–9.
18. Stahle E. Sternal wound complications incidence, microbiology and risk factors. *Eur J CardioThorac Surge.* 1997;**11**(6):1146–53.
19. Preventing the Spread of Vancomycin Resistance–A Report from the Hospital Infection Control Practices Advisory Committee prepared by the Subcommittee on Prevention and Control of Antimicrobial-Resistant Microorganisms in Hospitals; comment period and public meeting–CDC. Notice. *Fed Regist.* 1994;**59**(94):25758–63.