Influence of pathogenetic factors on prognosis in patients with native valve infective endocarditis

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Abstract

Introduction: Despite improvement in medical care the incidence of infective endocarditis (IE) has not decreased. Positive blood cultures are one of the most important diagnostic criteria of IE. There are no uniform data regarding the influence of pathogenetic factors on prognosis.

Aim: To analyse the results of blood and valve cultures in patients with IE of native valves and evaluate their influence on the risk of early and late deaths as well as recurrence of IE.

Methods: The study group consisted of 152 patients with IE of native valves. The IE diagnosis was based on the Duke criteria. Early and late mortality as well as recurrence of IE were analysed in patients hospitalised at the Institute of Cardiology in Warsaw from 1988 to 1998.

Results: Positive blood cultures were found in 103 (67.8%) of patients. In-hospital mortality was 5.9% (9 deaths). The incidence of early deaths was significantly lower in surgically treated patients (p=0.01). Late deaths occurred in 23 (16%) patients. Results of blood and valve tissue cultures were not related to mortality. Recurrent IE was observed in 7 (4.9%) patients. *Staphylococcus aureus* was an independent risk factor for recurrent IE (p=0.04). Six-year survival was 79%.

Conclusions: In patients with native valve infective endocarditis:

1. The risk of early and late death is not related to the results of blood and valve cultures.
2. *Staphylococcus aureus* aetiology increases the risk of disease recurrence.
3. The risk of early death is significantly lower in patients treated with surgery.

Key words: native valve endocarditis, pathogenetic factor, prognosis

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Introduction

Infective endocarditis (IE) is a relatively rare disease with an incidence of 1.5 to 7.0 per 100,000 patient-years [1-3]. Despite modern treatment methods, the mortality of patients with IE of the native valve (NVIE) according to different investigators ranges from 7.6% to 20% [4, 5].

All diagnostic criteria of IE available so far attribute the major importance to blood cultures [1, 6]. Frequently the type of cultured pathogen accelerates selection for surgery [7]. Despite widespread application of correct rules of blood sampling (including automatic) and the use of modern media, the frequency of negative cultures is still 5-31% [8, 9]. The main cause of negative cultures is the common use of antibiotics in hectic diseases prior to accurate diagnosis [1]. Recently, polymerase chain reaction, detecting bacterial and fungal DNA, was implemented in the microbiological diagnostics of IE [1, 10, 11].

Streptococcus viridans species are the most common cause of IE of native valves. They are detected in 30 to 60% of cases [12]. *Staphylococcus aureus* - in about 20% methicillin-resistant strains are isolated (methicillin-resistant *Staphylococcus aureus*, MRSA). *Staphylococcal endocarditis* has severe clinical manifestation, often with abscess formation [12-14]. *Staphylococcus epidermidis* belongs to the coagulase-negative strains that are

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natural microbial flora of the skin and mucose membranes. It causes from 8 to 33% of IE, particularly of the prosthetic heart valves [1, 12]. *Enterococcus* (*fecalis*, *durans*, *fecium*) strains are pathogenic when settled in the tissues outside the gastrointestinal tract. They may be identified in 5 to 15% of cases of IE [12]. Gram-negative bacteria (*Enterobacteriaceae*, *Pseudomonas sp.* and HACEK group) are present in about 10% of NVIE cases [1]. Infective endocorditis caused by *Pseudomonas aeruginosa* leads to fast destruction of the valvular tissue and vegetation formation [12]. Fungi such as *Candida albicans* and *Aspergillus sp.* are difficult to eliminate and fungal IE is most often seen in drug abusers, in patients with prolonged antibiotic therapy, corticosteroid therapy or cytostatic agent use [1, 12].

There is no doubt that positive blood cultures are important for the diagnosis and management of IE; however, there are no clear data regarding the risk of early and late death or recurrence of IE in relation to the identified pathogen or negative blood and/or valvular specimen cultures.

The aim of this study was to analyse the results of blood and valve cultures in patients with NVIE with an evaluation of their influence on the risk of early as well as late deaths as well as recurrence of IE.

Table I. Clinical characteristics of studied patients

Data	n (%)
Gender:	
females	36 (24)
males	116 (76)
Age [years]	10-76; mean 46
Previously diagnosed valvular defect:	
yes	49 (32.2)
no	103 (67.8)
IE location:	
Aortic valve	78 (51.3)
Mitral valve	29 (19)
Aortic + mitral valves	32 (21.1)
Tricuspid valve	11 (7.2)
Aortic + tricuspid valves	1 (0.7)
Mitral + tricuspid valves	1 (0.7)
Heart failure (NYHA class):	
I	0
II	29 (19.1)
III	53 (34.9)
IV	70 (46)
Positive blood/valvular cultures:	
yes	103 (67.8)
no	49 (32.2)

Methods

The analysis involved 152 patients with NVIE treated at the National Institute of Cardiology in 1988-1998.

Diagnosis of IE was based on the currently approved standards of the Polish Cardiac Society [15]. Infective endocarditis was diagnosed if confirmed intraoperatively or during autopsy or when clinical criteria were met including 2 major criteria, or 1 major and 3 minor criteria, or 5 minor criteria.

The following parameters were evaluated in this group: number of early and late deaths, and number of IE recurrences. Single factor analysis was used for the evaluation of the influence of pathogen type on the prognosis of IE patients. Early deaths were defined as in-hospital deaths. Remote mortality was referred to as deaths following patients' discharge from hospital. Infective endocarditis recurrence was defined as every next occurrence of the disease after the recovered primary IE and negative follow-up blood cultures following the end of antibiotic therapy. The follow-up period ranged between 9 months to 15 years (mean 6 years). 15 February 2006 was assumed the follow-up end. The endpoint was the date of early death, late death, recurrence or late follow-up examination.

Statistical analysis

The χ^2 test was used for the analysis of the influence of positive blood/valve tissue cultures and negative cultures on the incidence of early and late deaths as well as disease recurrences. Fisher's exact test was used for an expected population count below 5. Single-factor model of Cox proportional hazard was applied for the identification of risk factors of early and late deaths as well as recurrence of the disease. Analysis of variance was used for the evaluation of continuous variables. Survival time and time to recurrence were illustrated with Kaplan-Meier curves, tested with survival test and analysed with Cox proportional hazard. Statistical analyses were performed using the SAS v. 8.2 statistical software. A p value of <0.05 was considered significant.

Results

Clinical characteristics of the group and infection location are shown in Table I. In 152 patients with NVIE, positive blood or valve cultures were found in 103 (67.8%) subjects. Types of identified pathogens are illustrated in Figure 1. The most common one was *streptococcus viridans* (19.1%). Staphylococci were the most often cultured group of bacteria (29.6%, Figure 1).

In the study group, 9 (5.9%) early deaths were recorded. No correlation was found between NVIE aetiology and incidence of early deaths (Table II). Early deaths were most commonly observed in NVIE caused by staphylococcal infection; however, no statistical significance was reached. All patients with NVIE caused by *Streptococcus viridans* were effectively treated. Among all 152 patients, only 17 (11.2%) subjects were conservatively treated, whereas 135 (88.8%) underwent surgery. Of 135 patients treated with surgery, 5 (3.7%) individuals died. In the group of 17 patients treated pharmacologically, 4 (23.5%) patients died.

Incidence of death among patients treated with surgery was significantly lower than in those receiving only antibiotics (p=0.01, Table II). In all patients inflammatory lesions were located on the aortic valve, and in one patient the mitral valve was also affected. All these patients were in NYHA functional class IV while operated on. Causes of death were mainly related to thrombotic complications, sepsis and heart failure resistant to therapy.

Deaths in the group of pharmacological treatment occurred in patients with sepsis, cardiogenic shock and with prior antibiotic therapy continued for many weeks (Table II). They died on the first or second day of hospitalisation. These patients were selected for cardiac surgery too late. In the majority of subjects the infection involved both aortic and mitral valves.

The remaining 13 of 17 patients treated conventionally were not selected for surgery due to the lack of haemodynamic significance of valvular defects and small sizes of vegetations which regressed after antibiotic therapy. Two patients underwent surgery on during long-term follow-up: one due to the recurrence of IE and the other due to the progression of aortic regurgitation. One hundred and forty-three patients (94%) with NVIE were discharged home. Hospitalisation duration ranged between 12 and 67 days (mean 52 days).

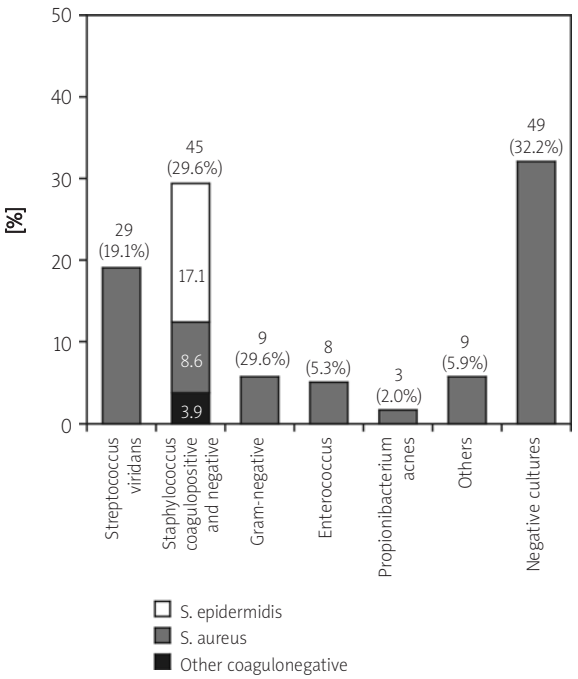

Figure 1. Aetiology of IE

Recurrence of IE was diagnosed in 7 (4.9%) patients. Time to IE recurrence ranged from 3 weeks to 67.2 months (mean 17.6 months) from the cured IE episode. The disease recurrences were significantly more

Table II. Early mortality rate with respect to aetiology and treatment

Aetiology	n=152	Early death		Surgical treatment	Conservative treatment
	n	n (%)	p	n	n
Streptococcus viridans	29	0 (0)	NS	(–)	(–)
Coagulase (+) and (–) *Staphylococcus* including:	45	5 (11.1)	NS	3	2
Coagulase (–) *Staphylococcus*	32	3 (9.4)	NS	2	1
Staphylococcus aureus	13	2 (15.4)	NS	1	1
Gram-negative	9	0 (0)	NS	(–)	(–)
Enterococcus	8	1 (12.5)	NS	1	(–)
Propionibacterium acnes	3	0 (0)	NS	(–)	(–)
Others	9	0 (0)	NS	(–)	(–)
Negative cultures (of blood and valves)	49	3 (6.1)	NS	1	2

Coagulase (+) and (-) Staphylococcus - Staphylococcus epidermidis + Staphylococcus aureus + other staphylococci; other pathogens include: 2 Bacillus sp. - gram-positive aerobic bacillus, 1 Gemella morbilorum - Streptococcus species, 1 Streptococcus pneumoniae - β-haemolytic streptococcus group A, 1 Streptococcus pyogenes - β-haemolytic streptococcus group A, 1 Streptococcus bovis – group D streptococcus, 2 Micrococcus sp - gram-positive aerobic cocci, 1 Acinetobacter radioresistens - small aerobic gram-negative bacillus

Table III. Recurrence rate with respect to aetiology

Aetiology		Recurrence	
	n	n (%)	p
Streptococcus viridans	29	0	NS
Coagulase (+) and (–) *Staphylococcus* including:	40	4 (10.0)	NS
Coagulase (–) *Staphylococcus*	29	2 (6.9)	NS
Staphylococcus aureus	11	2 (18.2)	0.03
Gram-negative	9	1 (11.1)	NS
Enterococcus	7	0	NS
Propionibacterium acnes	3	0	NS
Others	9	0	NS
Negative cultures (of blood and valves)	46	2 (4.3)	NS

Table IV. Long-term survival with respect to aetiology

Aetiology	n=143	Recurrence	
	n	n (%)	p
Streptococcus viridans	29	3 (10.3)	NS
Coagulase (+) and (–) *Staphylococcus* including:	40	6 (15.0)	NS
Coagulase (–) *Staphylococcus*	29	4 (13.8)	NS
Staphylococcus aureus	11	2 (18.2)	NS
Gram-negative	9	1 (11.1)	NS
Enterococcus	7	1 (14.3)	NS
Propionibacterium acnes	3	1 (33.3)	NS
Others	9	1 (11.1)	NS
Negative cultures (of blood and valves)	46	10 (21.7)	NS

common in patients infected with *Staphylococcus aureus* (p=0.03) (Table III). Other pathogens or negative cultures had no significant influence on the incidence of IE recurrences.

There were 5 recurrences observed in 130 subjects treated with surgery and antibiotics due to IE. In 3 patients the aetiology of recurrence was different than that of the primary infection. In the remaining two, the recurrence was caused by the same pathogen. Four patients were selected for further surgery; 1 patient with small vegetations found on echocardiography was conservatively treated. All patients were successfully cured. A second recurrence occurred in 2 patients and both underwent surgery. One patient was cured and the other one died during surgery. The autopsy confirmed myocardial fibroelastosis. In both cases the aetiology of the second recurrence (*Pseudomonadaceae* species bacteria) was the same as of the first recurrence. Abscesses were associated with the recurrences and were found intraoperatively in the majority of patients. Formation of an abscess was secondary to the second episode of endocarditis. The presence of an abscess in the primary IE did not determine the incidence of recurrence.

Two recurrences were seen in the group of 13 IE patients cured with antibiotics only. The recurrence was caused by a different pathogen than the primary IE. One case of recurrence was treated with drugs and the other with surgery. Both patients were cured and died during long-term follow-up.

During long-term follow-up, 23 (16%) of 143 cured patients died. The difference in the incidence of late deaths in the group of positive and negative blood or valve cultures was statistically insignificant: in 97 patients with positive cultures 13 (13.4%) died, and in 46 patients with negative blood cultures 10 (21.7%) deaths were observed. No significant differences were noted in the incidence of late deaths with respect to the pathogens of IE (Table IV). Nineteen of 23 late deaths occurred in the group treated surgically and 4 in the conservative treatment group. These included mainly sudden deaths. They were observed primarily in patients with negative blood cultures and all occurred in patients after surgically treated IE. Heart failure unresponsive to the treatment was observed more often within a 3-year or longer period following recovery of IE. Ten patients died within the first day after the surgical treatment of IE. In 3 subjects death was caused by the recurrence of IE. The other causes of death in this period were: embolic stroke, sudden death and cancer.

Single-factor analysis using Cox proportional hazard method showed that aetiology of IE of the native valves did not influence the risk of early and late death. *Staphylococcus aureus* was a risk factor for recurrence of IE. Six-year survival of patients with IE was 79%. Long-term follow-up covered 98% of the study patients.

Discussion

Determination of risk factors of early death, late death and IE recurrence may influence management and therefore improve prognosis of patients. The aetiology of IE varies across years and between countries. In the 1970s and 1980s, the aetiology of IE was mainly *Streptococcus viridans*, as described in the study of Delahay et al. from France [9]. In subsequent years along with the increased frequency of prosthetic valve implant surgery, *Streptococcus viridans* became a major pathogen only in NVIE [4], and coagulase-negative Staphylococcus in patients with IE of the prosthetic valves [1, 12]. Current observations of many authors from England [8], Spain [4] and Italy [5] showed a marked

increase of *Staphylococcus* infections in IE. Also in Poland according to the data of Wos et al. *Staphylococcus* was an aetiological factor of IE in 56% of cases compared to only 13% for *Streptococcus viridans* [16].

On the basis of the analysis of blood and valve cultures from the presented material in the Institute of Cardiology in 1988-1998 we found that staphylococci are cultured more often than viridans streptococci in patients with NVIE. These results differ from those of the previous years: then, similarly to other countries, the most common aetiology of IE was *streptococcus viridans*. When interpreting these findings one should recognise that as many as 32% of cases were culture negative. Such a high percentage of negative blood cultures most likely associated with out-patient antibiotic administration prior to blood sample collection may lead to false evaluation of aetiological factors. The results obtained suggest that the aetiology of NVIE has changed in Poland: the number of NVIE cases caused by staphylococci has increased. Karchmer [12] also paid attention to the presence of coagulase-negative staphylococci in the aetiology of NVIE with different sensitivity to antibiotics than those on the prosthetic valves.

A significant decrease in early mortality due to IE has been noted since the 1990s. In France, early mortality in NVIE decreased from 21.6% to 16.6% [9] and in Spain - from 19% to 12% [4]. In our group early mortality in NVIE was 5.9%. Such effective treatment of NVIE was achieved due to modern antibiotic therapy, early selection for surgery, the experience of cardiac surgeons and professional perioperative care. As early as the 1980s first generation cephalosporins in combination with netilmicin were introduced. The use of netilmicin was associated with its potential lower ototoxic and nephrotoxic effects compared to gentamicin. In complicated streptococcal and staphylococcal infections with MRSE strains vancomycin has been used. Endocarditis caused by gram-negative flora or anaerobic bacteria was treated with imipen-cilastatin. Teicoplanin was introduced into the therapy of IE of enterococcal and staphylococcal aetiology (with MRSE strains) as early as 1996. Such antibiotic therapy preceded current European standards by several years [1].

The analysis carried out revealed that in our study group pathogenetic factors did not influence the risk of early death in patients with IE of the native valve. Many authors state that *Staphylococcus aureus* increases the risk of death. Delahay et al. published data derived from the follow-up of patients with IE of native and prosthetic valves in the 1980s [9]. Following the analysis of 123 patients with IE treated with surgery in 1988-1996 Bauernschmitt et al. concluded that the risk of death in patients in whom *Staphylococcus aureus* was cultured was higher compared to patients with different aetiology [17]. Renzulli et al., Wallace et al. and Castillo et al. did not validate the reports showing increased early mortality in patients with IE caused by *Staphylococcus aureus* [4, 5, 8]. In our study the risk of early death in NVIE was also not increased with *S. aureus* aetiology.

The incidence of late deaths after recovery from NVIE was not related to the aetiology of infection either. Other investigators have also found that type of infectious agent did not influence long-term survival. Mansur et al. presented results of IE treatment in 420 patients in 1978-1994 and provided evidence that microbe type did not affect long-term prognosis. Risk factors included, however, elderly and recurrent IE [18].

It is widely known that patients with past endocarditis are at risk of IE recurrence [18-20]. The views on definition of recurrent IE have changed over the years. Differences concern both whether the recurrent infection may be caused by the same pathogen as well as the time period between the recovery from primary IE and next IE, which is needed to diagnose recurrent IE and not persistent infection. Some authors suggest that recurrent IE may be caused by the same pathogen [4], whereas others claim that infection with another microorganism is obligatory [1, 9, 18]. Time from recovery of the primary IE to the diagnosis of recurrent IE varies between investigators from 17 to 180 days [4, 9, 18, 19]. Recent guidelines of ESC announced a new definition of recurrent IE where recurrent IE is another episode of endocarditis occurring within one year after surgery [1].

Some believe that the highest chance of recurrent IE is within the first 6 months following recovery from the primary infection [19]. In our study we adopted the definition of Gillinov and Netzer: another IE episode after prior clinically, biochemically and microbiologically proven full recovery of IE. Factors that might contribute to the recurrence of IE were analysed in detail. Identification of risk factors is more difficult due to the low incidence of recurrences. McGiffin et al. identified one risk factor, which was duration of extracorporeal circulation [19]. Risk factors listed by Mansur et al., who described the long-term results of 420 patients successfully recovered from IE, were: male gender and prosthetic valve IE within the first year postoperatively [18]. Frequency of recurrences ranges from 1.1% to 13% [4, 20]. Instantly recurrent IE was observed in 4.9% of 143 patients after recovery from NVIE. Staphylococcal aetiology was the risk factor of recurrent IE. *Staphylococcus aureus* was the risk factor in patients with NVIE.

Long-term results of combined treatment of IE are improving. In our group six-year survival of patients with IE is 79%. Survival from NVIE after a mean of 5 years according to other investigators ranges from 75% to 88% [4, 20].

Conclusions

In patients with native valve infective endocarditis:

1. the risk of early and late death is not related to the results of blood and valve cultures;
2. *Staphylococcus aureus* aetiology increases the risk of disease recurrence;
3. the risk of early death is significantly lower in patients treated with surgery.

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Wpływ czynnika patogenetycznego na rokowanie u chorych z infekcyjnym zapaleniem wsierdzia na naturalnych zastawkach

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Streszczenie

Wstęp: Pomimo poprawy opieki zdrowotnej częstość występowania incydentów infekcyjnego zapalenia wsierdzia (IZW) nie zmniejszyła się. Dodatnie wyniki posiewów krwi są jednym z ważniejszych kryteriów diagnostycznych IZW. Nie ma jednoznacznych danych mówiących o wpływie czynnika patogenetycznego na rokowanie.

Cel: Analiza wyników posiewów krwi i z materiału zastawkowego u chorych z infekcyjnym zapaleniem wsierdzia na naturalnych zastawkach, z oceną ich wpływu na ryzyko zgonu wczesnego, odległego i nawrotu IZW.

Metodyka: Grupę badaną stanowiło 152 chorych z IZW na naturalnej zastawce. Rozpoznanie IZW weryfikowano w oparciu o kryteria Duke. Śmiertelność wczesną, odległą i nawroty IZW analizowano u chorych hospitalizowanych w Instytucie Kardiologii w Warszawie w latach 1988–1998.

Wyniki: Dodatnie posiewy stwierdzono u 67,8% (103) chorych. Śmiertelność szpitalna wynosiła 5,9% (9 zgonów). Zgony odległe wystąpiły u 23 (16%) chorych. Wyniki posiewów krwi i zastawek nie miały wpływu na śmiertelność. U 7 (4,9%) chorych wystąpił nawrót IZW. *Staphylococcus aureus* był niezależnym czynnikiem ryzyka nawrotu IZW (p=0,04). Przeżycie 6 letnie wynosiło 79%.

Wnioski: W infekcyjnym zapaleniu wsierdzia na naturalnej zastawce:

1. ryzyko zgonu wczesnego i odległego nie zależy od wyników posiewów krwi i materiału zastawkowego;
2. etiologia *Staphylococcus aureus* zwiększa ryzyko nawrotu;
3. ryzyko wczesnego zgonu jest istotnie niższe w grupie chorych leczonych operacyjnie.

Słowa kluczowe: infekcyjne zapalenie wsierdzia na zastawkach naturalnych, czynnik patogenetyczny, rokowanie

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