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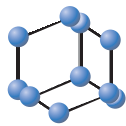


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The Role of the Coagulase-negative Staphylococci (CoNS) in Infective Endocarditis; A Narrative Review from 2000 to 2020



Mohammad A. Noshak^{1,2}, Mohammad A. Rezaee^{1,3,*}, Alka Hasani^{1,3} and Mehdi Mirzaii⁴

¹Immunology Research Center, Tabriz University of Medical Sciences, Tabriz, Iran; ²Student Research Committee, Tabriz University of Medical Sciences, Tabriz, Iran; ³Department of Medical Microbiology, Faculty of Medicine, Tabriz University of Medical Sciences, Tabriz, Iran; ⁴Department of Microbiology, School of Medicine, Shahrood University of Medical Sciences, Shahrood, Iran

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Abstract: Coagulase-negative staphylococci (CoNS) are part of the microbiota of human skin and rarely linked with soft tissue infections. In recent years, CoNS species considered as one of the major nosocomial pathogens and can cause several infections such as catheter-acquired sepsis, skin infection, urinary tract infection, endophthalmitis, central nervous system shunt infection, surgical site infections, and foreign body infection. These microorganisms have a significant impact on human life and health and, as typical opportunists, cause peritonitis in individuals undergoing peritoneal dialysis. Moreover, it is revealed that these potential pathogens are mainly related to the use of indwelling or implanted in a foreign body and cause infective endocarditis (both native valve endocarditis and prosthetic valve endocarditis) in patients. In general, approximately eight percent of all cases of native valve endocarditis is associated with CoNS species, and these organisms cause death in 25% of all native valve endocarditis cases. Moreover, it is revealed that methicillin-resistant CoNS species cause 60 % of all prosthetic valve endocarditis cases. In this review, we describe the role of the CoNS species in infective endocarditis, and we explicated the reported cases of CoNS infective endocarditis in the literature from 2000 to 2020 to determine the role of CoNS in the process of infective endocarditis.

Keywords: Coagulase-negative staphylococci, Staphylococcal species, infective endocarditis, pulmonic valve endocarditis, native valve endocarditis, peritonitis.

1. INTRODUCTION

Infective Endocarditis (IE), as an illness with high morbidity and mortality, is a severe condition that is often caused by several different bacterial pathogens [1]. An infection created by the bacterial pathogen in the cardiac endothelium or prosthetic material leads to IE and can affect native and prosthetic tissue [2]. Based on the published study, the global incidence of IE is 3-7 per 100,000 peoples. The mortality rate of IE is various and reported as follows: the overall mortality rate in hospitals is 15-30%, and this rate among elective patients and in emergency surgery was 10 % and >30%, respectively [1, 3, 4]. Moreover, it is reported that in 25 to 30 percent of IE cases to control infection, the combination of medical treatment with surgery is critical [5]. The severity of IE depends on several factors, including virulence of the pathogenic microorganism, characteristics of the patient (immunocompromised or non-immunocompromised patient), delays in assessment and treatment, delay in the timing of

surgery, surgical indications, and existence of the underlying illness [6, 7]. Several different pathogenic microorganisms can lead to IE; however, bacterial pathogens have an essential role and are the most common cause of IE [8, 9]. The most prevalent bacterial pathogens involved in IE are Gram-positive organisms, including *Staphylococcus aureus* (estimated incidence of 49.3%), *Streptococcus* species (estimated incidence of 24.7%), and enterococcal [10]. Among patients over the age of 60 years, these microorganisms together make >70- 90% of causative organisms [11]. CoNS species are part of the healthy flora of human skin and typically regarded as nonpathogenic contaminants, except in patients with prosthetic material [12-15]. In most cases, CoNS causes infection among patients with neurosurgical shunts, artificial joints, prosthetic heart valves, and central vascular catheters [16]. These microorganisms are related to various infections in immunocompromised individuals, including I) approximately 30% of all nosocomial bloodstream infections, II) urinary tract infection, III) endophthalmitis, IV) central nervous system shunt infection, V) endocarditis, VI) surgical site infections, and VII) foreign body infection [12, 17]. Coagulase-Negative Staphylococci (CoNS) can invade and destroy native tissue

*Address correspondence to this author at the Immunology Research Center, Tabriz University of Medical Sciences, Tabriz, Iran;
E-mail: rezaee@tbzmed.ac.ir

and are now recognized as essential pathogens in IE. The IE caused by CoNS, unlike IE created by *Staphylococcus aureus*, responds well to prescribed antibiotics and is infrequently complicated by congestive heart failure or valvular destruction [18-20]. The comprehensive list of reported cases of CoNS endocarditis from 2000 to 2020 is shown in Table 1 and Fig. (1). The CoNS cause a significant proportion of infections of both cardiac valve prostheses (Prosthetic Valve Endocarditis (PVE) and native cardiac valves (Native Valve Endocarditis (NVE)). Based on the International Collaboration of Endocarditis-Prospective Cohort Study reports, 7.8% of all cases of NVE are caused by CoNS (most cases caused by *Staphylococcus epidermidis*) and these organisms cause death in 25% of all NVE cases [18, 21]. In general, NVE can be caused by CoNS acquired from two ways; I) from the nosocomial setting, and II) from the community [22]. In community-acquired form, NVE is acquired from various ways, and it is related to several factors, including I) the presence of a long-term indwelling or implanted foreign bodies such as a pacemaker or central catheter, II) hemodialysis, and III) a new invasive procedure [23]. Moreover, it is revealed that 60% of all PVE cases are caused by methicillin-resistant CoNS (MR-CoNS) [1]. Various antibiotics such as gentamicin and rifampin are beneficial treatment in PVE cases; however, vancomycin is typically recommended for treatment of the MR-CoNS NVE cases [24]. In general, the collaboration of multidisciplinary teams, including infectious disease physicians, medical microbiologists, cardiologists, and cardiac surgeons, is critical in the treatment of IE [25-27]. The CoNS by two main features, including 1) attachment to matrix proteins formed on polymer surfaces of foreign bodies and 2) biofilm formation (protect bacteria from the action of antibiotics and the immune system) has a unique property and causes infection of native heart valves [28, 29]. Furthermore, these bacteria have low virulence and create a subacute or chronic infection [12]. Several epidemiological and clinical studies have shown that various CoNS isolates can create IE, especially *Staphylococcus lugdunensis*, *Staphylococcus epidermidis*, *Staphylococcus hominis*, *Staphylococcus capitis*, *Staphylococcus simulans*, *Staphylococcus saprophyticus*, *Staphylococcus haemolyticus*, and *Staphylococcus warneri* (Table 1). Also, it is reported that CoNS can contribute to the development or progression of IE [1]. Finally, aligned with the correlation between CoNS in IE, we attempted to explain the role of each CoNS and summarize the epidemiological report of them, which isolates in IE in order to determine the role of CoNS in the process of IE.

2. SEARCH STRATEGY

In this narrative review, for papers from January 1, 2000, to January 1, 2020, English databases, including Science Direct, Web of Science, PubMed, Embase, and the Cochrane Library were searched. We performed the searching process using the following keywords alone or in combination with “OR” and/or “AND” based on the medical subject heading (MeSH) terms, including: “endocarditis,” “Cardiovascular disease,” “heart disease,” “native valve endocarditis,” “prosthetic valve endocarditis,” “CoNS,” and “Coagulase-negative staphylococci. Moreover, we searched references for related

studies. In the current study, all case report studies in the English language that reported the treatment and outcome of patients with IE due to CoNS were included. The systematic reviews, editorials, letters, and articles that were irrelevant to the topic of interest or were not available in the English language were excluded from the study.

2.1. *Staphylococcus warneri*

Staphylococcus warneri (*S. warneri*) is a member of CoNS isolates, and, in most cases, it is considered as a typical inhabitant of the skin of healthy individuals and commonly is not considered pathogenic [23]. In normal conditions, *S. warneri* does not commonly cause severe infections in patients. However, it is revealed that this bacterium infrequently causes serious infections such as septicemia, urinary tract infections, subdural empyema, discitis, osteomyelitis, and endocarditis (both NVE and PVE) in humans, especially in immunocompromised individuals with medical device implants or invasive treatments [30, 31]. In the past years (from 2000 to 2020), six IE cases (four cases with NVE and two cases with PVE) were traced to *S. warneri* as the etiological agent (Table 1) [32-88]. The result of a published study revealed that *S. warneri* is one of the *staphylococci* recognized in the saliva of healthy individuals and proposed that the oral cavity is a route of entry for IE [31]. *S. warneri*, unlike *S. epidermidis*, produces acid from trehalose and, similar to other CoNS isolates, can cause IE following vasectomy in patients with standard heart valves [89]. Moreover, endocarditis cases involving community-acquired prosthetic valve or native valve replacements have been revealed [90]. *S. warneri* is capable of causing severe aortic ring abscesses and widespread valvular destruction and is associated with higher mortality compared with other CoNS isolates such as *Streptococcus viridans* [24]. In general, *S. warneri* contaminates prosthetic heart valves and central venous catheters, and it is a severe nosocomial pathogen in prosthetic vascular grafts, orthopedic surgery, and in ventriculoatrial shunts [32]. Like other CoNS, *S. warneri* have several mechanisms and virulence factors, including metabolic changes in different conditions, biofilm formation, and attachment to polymer surfaces that allow this bacterium to persist on foreign bodies and become resistant to used antibiotics [31, 91]. This bacterium with biofilm production is related to chronic polymer associated syndrome [92]. Generally, the susceptibility to antibiotics among the *S. warneri* recovered from the pediatric patients varied from those isolated from adults. The most *S. warneri* recovered from hospitalized pediatric patients with catheter-related infections are susceptible to oxacillin [93]. *S. warneri* has slow growth and prolonged delay among onset of clinical sign and establishing the diagnosis; therefore, antibiotic therapy, often, could not prevent aortic regurgitation [32]. In most cases of IE, due to this bacterium, surgical management is required [90]. In conclusion, it is suggested that when a patient with identified valvular dysfunction presents with decompensated heart failure, even in the absence of leukocytosis, fever, and other typical clinical manifestation, the clinicians should consider IE with a CoNS isolates such as *S. warneri*, a low virulence but invasive pathogen. Moreover, it is suggested that in patients with *S. warneri* bacteremia, echocardiography should be carried out.

Table 1. Coagulase-negative staphylococci endocarditis: reported cases in the English literature from 2000 to 2020.

<i>Staphylococcus warneri</i>				
Case	Country (Gender/Age)	Cardiac Abnormality	Treatment (Outcome)	Refs.
1	Romania (Male/79)	Native valve endocarditis (degenerative valvular heart disease)	IV vancomycin, gentamicin, oxacillin, and heart surgery (Survived).	[23]
2	USA (Male/67)	Early prosthetic valve endocarditis	Oxacillin and vancomycin, oral rifampin, and valve replacement surgery (Survived).	[24]
3	USA (Male/59)	Native valve endocarditis	IV nafcillin- cefazolin, and surgical treatment (Survived).	[30]
4	USA (Female/78)	Native valve endocarditis	IV nafcillin with gentamicin (Survived).	[31]
5	Austria (Male/48)	Endocarditis of the native aortic valve	IV vancomycin, fucidic acid and rifampicin orally (Patient clinical condition improved).	[32]
6	France (Male/71)	Early prosthetic valve endocarditis	IV vancomycin, gentamicin, oral pefloxacin, cefuroxime was used as prophylactic surgical antibiotherapy. (The patient remained well and afebrile when he was discharged).	[33]
<i>Staphylococcus haemolyticus</i>				
Case	Gender (Gender/Age)	Cardiac Abnormality	Treatment (Outcome)	Refs.
1	Belgium (Male/79)	Early prosthetic valve endocarditis	IV vancomycin, oral rifampin, coronary artery bypass graft surgery (Survived)	[34]
2	Italy (Female/70)	Congestive heart failure	Vancomycin plus rifampin, aortic valve replacement (No relapse was observed during a 12-month follow-up).	[35]
3	Italy (Male/72)	Embolus in the distal radial artery	Vancomycin and rifampin, combined with gentamicin during the initial 2 weeks of therapy, aortic valve replacement. (Seven months after antibiotic discontinuation, the patient developed a new episode of IE caused by <i>Candida albicans</i>).	[35]
4	Italy (Male/77)	Infective endocarditis	Vancomycin and rifampin. (The patient progressively improved with clearance of bacteremia, lost after a 3-month follow-up).	[35]
5	Italy (Male/65)	Cerebral, probably mycotic, aneurysm at the right communicans posterior artery	Vancomycin plus rifampin, aortic valve replacement (Patient died after 9 days in a deep coma).	[35]
6	Spain (Male/76)	Pulmonary valve infective endocarditis	(Patient remained asymptomatic and without further recurrences).	[36]
<i>Staphylococcus saprophyticus</i>				
Case	Country (Gender/Age)	Cardiac Abnormality	Treatment (Outcome)	Refs.
1	Japan (Male/61)	Native valve endocarditis	Vancomycin and clindamycin, cardiac surgery for aortic valve replacement. (The patient's clinical course was uneventful, and was discharged home).	[37]
2	Spain (Male/50)	Native valve endocarditis	Intravenous ampicillin, gentamicin and vancomycin, surgical treatment (The postoperative course was uneventful and the patient was discharged for follow-up as an outpatient).	[38]
<i>Staphylococcus lugdunensis</i>				
Case	Country (Gender/Age)	Cardiac Abnormality	Treatment (Outcome)	Refs.
1	USA (Male/20)	Pulmonic valve infective endocarditis	Vancomycin, cardiothoracic surgery (Improved).	[39]
2	UAE (Male/44)	Native valve endocarditis	Vancomycin and flucloxacillin, surgical intervention (Survived).	[40]
3	USA (Male/14)	Right-sided endocarditis	Vancomycin, gentamicin, IV cefazolin and rifampin, nafcillin, surgical intervention and central line placement (Survived).	[41]
4	Japan (Male/2)	Right-sided endocarditis	Vancomycin, cardiovascular surgery (There was no sign of recurrent infection 8 months after the surgery).	[42]
5	Saudi Arabia (Male/43)	Destructive native triple valve endocarditis	Gentamicin and penicillin, vancomycin, cardiac surgery (Discharged in a stable condition).	[43]
6	Spain (Male/66)	Infective endocarditis	Surgical intervention (Survived).	[44]

(Table 1) Contd....

<i>Staphylococcus lugdunensis</i>				
Case	Country (Gender/Age)	Cardiac Abnormality	Treatment (Outcome)	Refs.
7	USA (Infant/<1)	Infective endocarditis	Vancomycin and IV nafcillin (The patient was discharged home after completing 6 weeks of antibiotics).	[8]
8	USA (Male/63)	Infective endocarditis	Gentamicin, nafcillin, rifampin, IV daptomycin, mitral valve replacement surgery (He was feeling well).	[9]
9	Japan (Male/74)	Mitral valve endocarditis	Vancomycin, gentamicin, cefazolin, rifampicin, surgical intervention. (The patient was discharged from the hospital with no complaints and was negative for C-reactive protein after 6 weeks of antibiotic therapy).	[45]
10	USA (Male/53)	Destruction of the ventricular septum and multiple native valves.	Intravenous vancomycin and ceftriaxone, daptomycin, open heart surgery (The patient was discharged home with daptomycin to complete 6 weeks of treatment).	[46]
11	USA (female (33) and male (41)) (Age: 21-86)	11 of 74 patients with Infective endocarditis	Vancomycin and linezolid (4 patients were died).	[47]
12	France (Male/2)	Infective endocarditis	Vancomycin with gentamicin and ceftriaxone, oxacillin, surgical intervention (Survived).	[48]
13	Spain (Male/68)	Infective endocarditis	Right hip surgery (Survived).	[49]
14	Austria (Female/35)	Infective endocarditis	Flucloxacillin, penicillin, cardiac surgery (Ambulant follow-up visits 3, 5, 10, and 16 months after surgery showed that the patient is in good health, yet with persistent moderate perivalvular regurgitation of the artificial mitral valve).	[50]
15	USA (Female/66)	Prosthetic valve endocarditis	Open heart surgery (Survived).	[51]
16	USA (Male/36)	Native valve endocarditis	IV nafcillin and vancomycin, valvular Surgery (Survived).	[52]
17	USA (Male/48)	Native valve endocarditis	Cefazolin and rifampin, cardiothoracic surgery (The patient's cardiac status gradually worsened over the following days, and he ultimately died).	[53]
18	Norway (Male/56)	Infective endocarditis	Vancomycin with gentamicin, cardiac surgery (The conclusion in the autopsy report was that the patient died of <i>S. lugdunensis</i> endocarditis with complications).	[54]
19	Spain (Female/4)	Recurrent infective endocarditis	IV penicillin G, rifampicin, gentamicin, contegra valved conduit replacement (The patient was discharged in excellent clinical state without showing infectious complications over the last 2 years).	[55]
20	USA (female (5) and male (7), Age (29-69)	12 patients with destructive cause of coagulase negative staphylococcus infective endocarditis.	Valve surgery (One patient died).	[56]
21	USA (Male/66)	Mitral valve endocarditis after prostate biopsy.	Intravenous vancomycin and oral rifampicin, cardiothoracic surgery (The patient had an uneventful postoperative course and was discharged on a 4-week regimen of antibiotics).	[57]
22	Japan (Male/55)	Mitral valve endocarditis	IV penicillin and gentamicin, surgery (Postoperative echocardiography 1 year after the procedure confirmed a good mitral valve function without either mitral regurgitation or any signs of recurrent infection).	[58]
23	Italy (Female/18)	Endocarditis complicated by embolis with mitral valve prolapse.	Oxacillin and gentamicin, heart surgery (At one-year follow-up patient is well).	[59]
24	USA (Female/74)	Pacemaker related infective endocarditis	IV nafcillin and rifampin, heart surgery (Patient was placed in hospice care and expired two days later).	[60]
25	Taiwan (Female/66)	Pacemaker related infective endocarditis	Vancomycin and gentamicin (The patient died from sudden cardiac arrest 2 days later).	[28]
26	USA (Male/22)	Native tricuspid valve infective endocarditis	Vancomycin, gentamicin, daptomycin and cefazolin, cardiac surgery (Survived).	[61]
27	USA (Male/49)	Infective endocarditis	Nafcillin, gentamicin, daptomycin (The patient made a complete clinical recovery with no residual effects of the disease).	[62]
28	USA (Male/58)	Infective endocarditis	Vancomycin, Daptomycin (To date, the patient is doing well and has made a full recovery).	[62]
29	New Zealand (Female/47)	Bicuspid aortic valve and an aneurysm of the ascending aorta.	IV flucloxacillin, heart surgery (Survived).	[63]

(Table 1) Contd....

<i>Staphylococcus lugdunensis</i>					
Case	Country (Gender/Age)	Cardiac Abnormality	Treatment (Outcome)		Refs.
30	Taiwan (Male/67)	Tricuspid endocarditis.	Oxacillin, gentamicin, and ceftriaxone; gentamicin and ceftriaxone, heart surgery (A nosocomial infection complicated the patient's recovery and he died of severe sepsis).		[64]
31	USA (Male/46)	Native tricuspid valve endocarditis	Vancomycin, nafcillin, surgical intervention (Survived).		[65]
<i>Staphylococcus simulans</i>					
Case	Country (Gender/Age)	Cardiac Abnormality and Complication	Treatment (Outcome)		Refs.
1	USA (Female/64)	Mycotic inferior mesenteric artery aneurysms (Native valve endocarditis).	Daptomycin and surgical treatment (Patient was monitored in follow up at 2 months and was asymptomatic with resolution of her abdominal pain).		[66]
2	Greece (Male/46)	Native valve endocarditis	IV vancomycin and teicoplanin, clindamycin hydrochloride orally (Survived).		[67]
<i>Staphylococcus epidermidis</i>					
Case	Country (Gender/Age)	Cardiac Abnormality	Treatment	Outcome	Refs.
1	-	Lead-dependent infective endocarditis (43 patients)	Broad-spectrum antibiotics (Survived)		[3]
2	Switzerland (Male/39)	Pacemaker-associated endocarditis	Intravenous vancomycin and rifampicin switched to flucloxacillin and rifampicin treatment, open-heart surgery (Survived).		[68]
3	China (Female/40)	Left-sided endocarditis and secondary hemophagocytic lymphohistiocytosis	Intravenous vancomycin and daptomycin, cardiothoracic surgery (Survived).		[69]
4	France (Male/46)	Infective endocarditis after percutaneous pulmonary valve implantation	Intravenous vancomycin (Survived).		[70]
5	Germany (Male/55)	Severe aortic valve stenosis, ectasia of the aorta ascendens and reduced left ventricular function, progressive prosthetic valve endocarditis.	Vancomycin plus rifampicin, daptomycin plus fosfomycin, cardiac surgery (Survived).		[71]
6	Spain, Age (60-62)	17/620 Patients with native valve endocarditis, 18/620 prosthetic valve endocarditis, 25/620 intracardiac device	Vancomycin, cloxacillin, cardiovascular surgery (Survived).		[72]
7	Spain (Male (61), Female (39), Age (70))	Infective endocarditis on pacemaker devices	Open heart surgery (Early mortality was 24% (33% of medically, 21% of surgically treated patients).		[17]
8	France (Male/82)	Infective endocarditis	Vancomycin, gentamicin and rifampicin (Patient expired).		[73]
9	USA (Male/80)	Aortic valve abscess	Intravenous vancomycin and meropenem (Survived).		[74]
10	Brazil (Male (14) and female (12))	Early onset prosthetic valve endocarditis (Embolism, acute renal failure, and heart failure)	Cephazolin, cefuroxime, vancomycin, heart valve surgery (Twelve patients (46.2%) were surgically treated and 14 patients (53.8%) only had clinical treatment. Among the surgical group, six died (50%) whereas among the clinical group 10 died).		[10]
11	USA (Male/75)	Hypertension and congestive heart failure	Nafcillin, cefazolin, intravenous vancomycin (He has since had restoration of health without further sequella).		[75]
12	USA (Male/76)	Native-valve endocarditis	IV vancomycin change to daptomycin, cardiac surgery (The patient has remained stable and continues to do well at 18 months after discontinuing daptomycin).		[18]
13	Israel (Male/23)	Infective endocarditis	IV vancomycin and rifampin change to daptomycin and rifampin orally (On follow-up visits, the patient remained afebrile).		[19]
14	Italy (Male/75)	Infective endocarditis	Vancomycin, teicoplanin, intravenous linezolid, aortic and mitral valve replacement (At 6-months follow-up after the end of the linezolid treatment, no complications were noted and infection appeared to be controlled).		[76]
15	UK (Female/66)	Brachial artery mycotic aneurysm in a patient with infective endocarditis	Teicoplanin, valve replacement surgery (Survived)		[77]
16	Netherlands (Male/49)	Prosthetic valve endocarditis	Oxacillin, gentamicin, rifampicin, vancomycin, fusidic acid, and surgical treatment (Survived).		[78]

(Table 1) Contd....

<i>Staphylococcus epidermidis</i>					
Case	Country (Gender/Age)	Cardiac Abnormality	Treatment	Outcome	Refs.
17	Switzerland (Male/29)	Endocarditis after mitral valve reconstruction	Quinupristin/dalfopristin, levofloxacin, and vancomycin, elective cardiac surgery (Survived).		[5]
18	Spain (Male/67.1)	8 patients with infection after cardiac surgery	Cefazolin, vancomycin, and cardiac surgery (2 patients were died after antibiotic therapy).		[79]
19	USA (Male/66)	Native valve endocarditis	Vancomycin, open-heart surgery for valve replacement (Cured).		[22]
20	USA (Male/62)	Native valve endocarditis	Vancomycin, open-heart surgery for valve replacement (Cured).		[22]
21	USA (Male/76)	Native valve endocarditis	Nafcillin/ rifampin, open-heart surgery for valve replacement (Cured).		[22]
22	USA (Female/33)	Native valve endocarditis	Vancomycin/gentamicin, open-heart surgery for valve replacement (Cured).		[22]
23	USA (Female/55)	Native valve endocarditis	Nafcillin, open-heart surgery for valve replacement (Died after cardiopulmonary arrest postoperatively).		[22]
<i>Staphylococcus hominis</i>					
Case	Country (Gender/Age)	Cardiac Abnormality	Treatment (Outcome)		Refs.
1	USA (Female/86)	Eustachian valve endocarditis	Vancomycin, open heart surgery with removal of eustachian valve (Survived).		[80]
2	Italy (Male/53)	Cardiovascular implantable electronic device endocarditis	Daptomycin (Survived).		[81]
3	Spain (Female/47)	Mitral valve endocarditis	Cloxacillin (Patient expired).		[82]
4	Turkey (Male/65)	Endocarditis created by pacemaker	IV vancomycin, gentamicin, and open-heart surgery (Survived).		[83]
5	USA (Male/53)	native valve infective endocarditis	IV vancomycin (Survived).		[84]
<i>Staphylococcus capitis</i>					
Case	Country (Gender/Age)	Cardiac Abnormality	Treatment (Outcome)		Refs.
1	USA (Male/79)	Mild aortic stenosis, paroxysmal atrial fibrillation	IV vancomycin - oral rifampin, cardiothoracic surgery (Gangrene of left foot, echo after 6 weeks, no vegetation).		[85]
2	USA (Male/80)	CABG, aortic sclerosis, mitral regurgitation	IV vancomycin switched to nafcillin, surgical treatment (Patient died of multiorgan failure).		[2]
3	USA (Male/72)	Aortic stenosis S/P aortic valve replacement, CABG, atrial fibrillation	IV vancomycin- gentamicin- oral rifampin, aortic valve replacement (Patient expired).		[86]
4	USA (Female/48)	Mitral valve repair and aortic valve replacement	IV vancomycin -gentamicin – oral rifampin, aortic valve replacement (Patient expired).		[86]
5	Saudi Arabia (Male/35)	Native valve endocarditis. Aortic valve affected.	Vancomycin plus rifampin (Clinical follow-up at 6 months showed no recurrence of endocarditis).		[1]
6	USA (Male/10)	Endocarditis with resultant osteomyelitis	Daptomycin/rifampin (Survived).		[87]
7	Japan (Female/79)	Prosthetic valve endocarditis	Vancomycin and oral, aortic valve replacement minomycin (No signs of infection were seen during 630 days of follow up).		[85]
8	Japan (Female/79)	Prosthetic valve endocarditis	Vancomycin + rifampin, aortic valve replacement (Recovered fully within a month, no signs of infection were observed during 332 days of follow-up).		[85]
9	Japan (Male/76)	Prosthetic valve endocarditis	IV teicoplanin, vancomycin, and linezolid, aortic valve replacement (No signs of infection were seen during 224 days after the surgery).		[85]
10	Japan (Female/68)	Prosthetic valve endocarditis	IV vancomycin and gentamicin, mitral valve re-implantation (No signs of infection were seen during 163 days of follow-up).		[85]
11	Italy (Male/46)	Acute endocarditis	Oxacillin 4 g/die for six weeks and gentamicin 240 mg/die for two weeks, cardiac surgery (The patient did not reveal worsening of the clinical condition).		[88]

2.2. *Staphylococcus haemolyticus*

In general, among clinical isolates of methicillin-resistant staphylococci, *Staphylococcus haemolyticus* (*S. haemolyticus*) is the 3rd most common microorganism and causes sev-

eral hospital-acquired infections, especially in intensive care unit patients or hematologic patients [35]. Besides, according to the published results, *S. haemolyticus* is frequently isolated from 22% to 24% of the central nervous system, and after *Staphylococcus epidermidis* is considered as the 2nd most

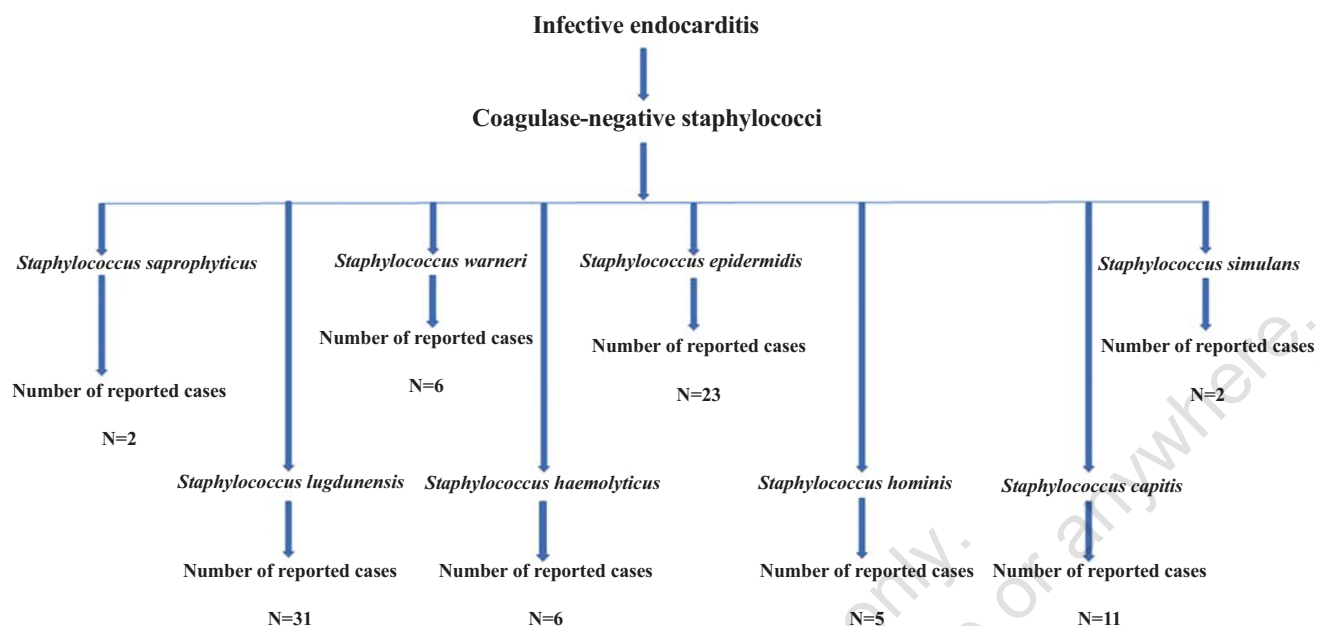


Fig. (1). A graphic of number of cases reported on infective endocarditis attributed to coagulase-negative staphylococci from 2000-2020. (A higher resolution/colour version of this figure is available in the electronic copy of the article).

common cause of CoNS bacteremia [94] (Fig. 2). The bacteremia due to *S. haemolyticus* is correlated with rare morbidity or mortality rates in patients with hematologic malignancies [95]. This organism can cause different infections, including skin or soft tissue infections, bacteremia, sepsis due to foreign bodies, meningitis, and prosthetic joint infections [34, 96]. *S. haemolyticus* have been considered to be low-virulent pathogens and is commonly considered a contaminant when isolated from sterile patients' specimens [36], but during the last decades, six cases (Table 1) of NVE and PVE due to this microorganism reported. IE caused by methicillin-resistant *S. haemolyticus* leads to various severe abnormalities such as nosocomial and non-nosocomial health-care-associated IE, embolic complications, left-sided valvular disruption, and finally death [35]. Moreover, *S. haemolyticus* is commonly resistant to the various classes of antibiotics, and, recently, this organism showed resistance to glycopeptides, particularly to teicoplanin [97, 98]. It is suggested that the patients with *S. haemolyticus* IE require more hospitalization and share increasing rates of predisposing comorbid conditions.

2.3. *Staphylococcus saprophyticus*

Staphylococcus saprophyticus (*S. saprophyticus*) as part of the healthy skin flora is clinically significant CoNS that infrequently reported as a human pathogen [37]. This organism commonly colonizes the genitourinary tract and causes Urinary Tract Infections (UTI) in most of the cases [99]. *S. saprophyticus*, after *Escherichia coli* considered the second most prevalent cause of uncomplicated cystitis between young women [37, 99, 100]. Also, several rare abnormalities such as endophthalmitis, pyelonephritis, and septicemia reported due to this microorganism [38]. It is revealed that *S. saprophyticus* can cause both NVE and PVE in patients with foreign bodies or patients with neurogenic bladder [37].

Moreover, *S. saprophyticus* develop NVE with a frequency of five to eight percent [101]. In general, from 2000 to 2019, only 2 cases of IE due to *S. saprophyticus* have been reported [37, 38]. The results of these studies revealed that *S. saprophyticus* causes a severe life-threatening IE; however, with intravenous ampicillin, gentamicin and vancomycin treatment, the patients' clinical courses were uneventful and were discharged for follow-up as an outpatient. Besides, *S. saprophyticus* among intravenous drug abusers is an important pathogen and can cause and develop IE [102]. It is revealed that *S. saprophyticus* is transmitted to these patients through contaminated injected drugs [103].

2.4. *Staphylococcus lugdunensis*

For the first time, *Staphylococcus lugdunensis* (*S. lugdunensis*) has emerged since the 1990s as a virulent coagulase-negative staph organism that causes an extensive range of severe infections [40, 104]. *S. lugdunensis*, along with other CoNS, isolates and comprise the microbiota of the skin of healthy humans and colonizes 30% to 50% of patients [49]. However, it behaves similarly to *S. aureus* and causes several infections such as osteomyelitis, urinary tract infections, bloodstream infections, cardiovascular-related infections (for example, IE in specific patients), aggressive valve destruction with abscess formation and lead to the progression of congestive heart failure [56, 57]. The global incidence of *S. lugdunensis* infective endocarditis is varied from 3 to 7 per 100,000 person-years annually. Moreover, it is indicated that in 18% to 46% of bacteremia cases, *S. lugdunensis* cause IE and this organism is recovered from 1.1% of all cases of IE in an adult inhabitant [40, 105]. The IE caused by *S. lugdunensis* is considered as catastrophic and requires widespread antibiotic therapy and surgical intervention [43]. According to the published report, approximately 36% of patients with *S. lugdunensis* IE die each year, and in 51% of patients with *S. lugdunensis* IE, valve replacement is

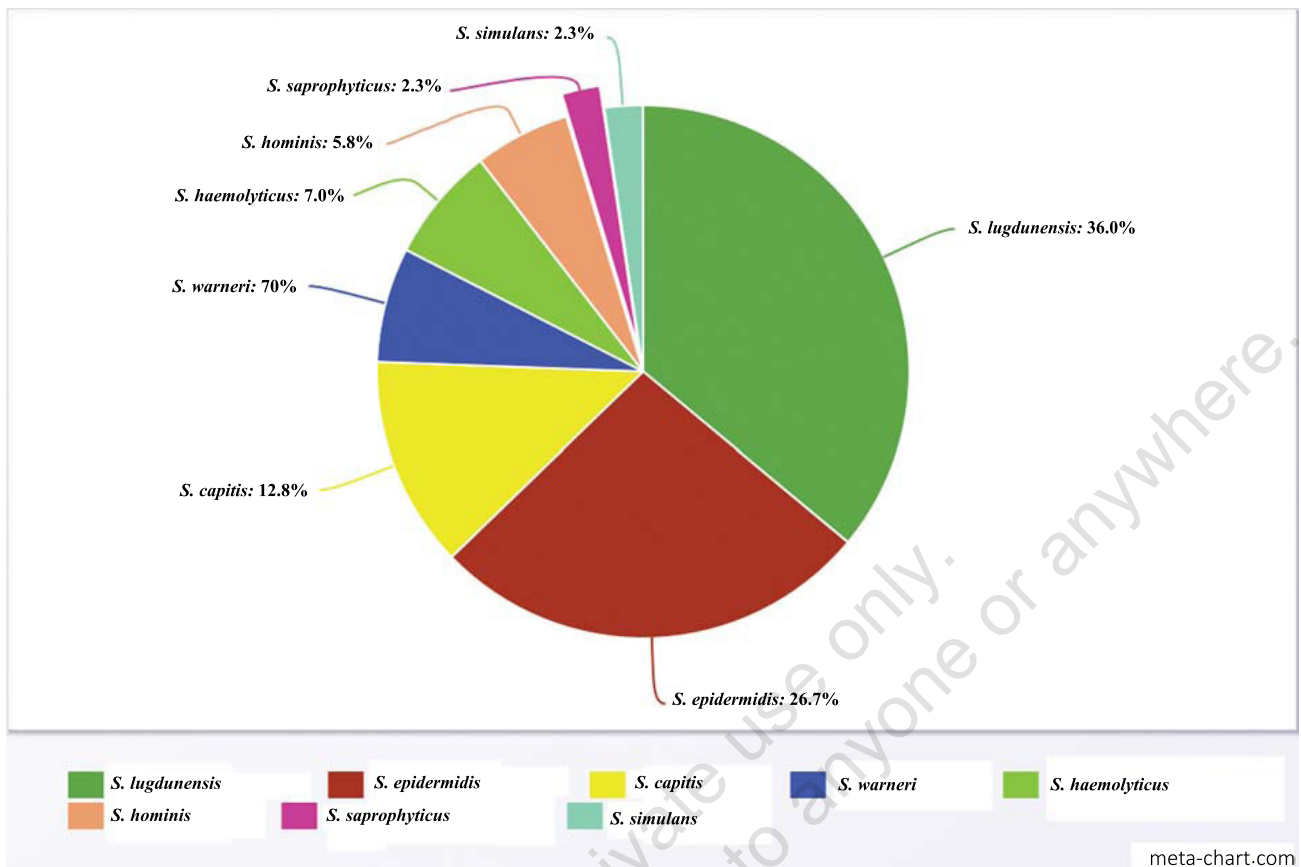


Fig. (2). The proportion of each coagulase-negative staphylococci species caused infective endocarditis. (A higher resolution/colour version of this figure is available in the electronic copy of the article).

required [47]. *S. lugdunensis* bind to the von Willebrand factor by specific proteins such as Fbl, a fibrinogen-binding protein that facilitates the attachment of the organism to vessel walls and cardiac valves. The binding of bacterium to the von Willebrand factor is considered as one of the critical steps in the initiation and development of IE [46, 106]. This organism is unable to bind and activate platelets [106]. On the other hand, like other CoNS species, *S. lugdunensis* produces biofilm and is adhere to bioprosthetic materials and native tissues; therefore, it can cause endocarditis in patients with prosthetic valves and cardiac implantable devices [58]. This bacterium has proteolytic activity and produces delta-like hemolysin that is one of the main factors in pathogenesis. Therefore, compared with other CoNS, *S. lugdunensis* causes more-aggressive infections [9, 104]. In most cases, *S. lugdunensis* is associated with native valve endocarditis and leads to different severe abnormalities, including septic emboli, valve perforation, and myocardial abscess [46, 105, 107]. Middle-aged men and women with *S. lugdunensis* endocarditis are susceptible to embolic stroke secondary [108]. In conclusion, it is suggested that in severe cardiac diseases such as destructive valve infective endocarditis, *S. lugdunensis* should be considered as an organism that has high levels of pathogenicity that can cause increased morbidity and mortality among patients.

2.5. *Staphylococcus simulans*

Staphylococcus simulans (*S. simulans*) as coagulase-negative staphylococci, in most cases, is considered as com-

mon animal (cattle, sheep, and other domestic animals) pathogens, infrequently found on human skin [109]. In general, *S. simulans* have several virulence factors and mechanisms involved in pathogenesis. These various mechanisms and factors are as follows; I) extracellular polysaccharide or biofilm formation, II) toxic or destructive substances secretion such as proteases, hemolysin by bacteria affected host tissues, and III) resistant to prescribed antibiotics, especially methicillin [11]. Like to *S. aureus* and in contrast to other CoNS species, *S. simulans* causes different severe infections in humans including I) vertebral osteomyelitis, chondronecrosis with osteomyelitis, and mandibular osteomyelitis II) omphalitis, III) bacteremia, IV) unilateral periorbital cellulitis, V) urinary tract infection, VI) gangrenous dermatitis, VII) UTI, and VIII) arthritis [66, 67, 110]. Moreover, *S. simulans* can cause IE (NVE) in patients [110]. *S. simulans* may enter the intestinal villous epithelial cells and penetrate the bloodstream and causes endocarditis [111]. In conclusion, endocarditis associated with this organism may be related to increased mortality without specific signs.

2.6. *Staphylococcus epidermidis*

Staphylococcus epidermidis (*S. epidermidis*) is often considered a culture contaminant, and it is a typical healthy microbiota of the human skin [4]. It is indicated that *S. epidermidis* has several different genes that are critical in adaptation to the changing of environmental conditions [68]. However, in contrast to many CoNS species, genes for toxic shock syndrome toxin one and enterotoxins are not found in

S. epidermidis [74]. *S. epidermidis* has several virulence factors and mechanisms, including serine protease, cysteine protease, fatty acid modifying enzymes, lipase, biofilm formation in the presence of foreign bodies, metalloprotease with elastase activity, delta toxin, and resistance to antibiotics [71, 112]. This organism, similar to different CoNS species, has a capability to biofilm formation and cause biofilm-associated infection in patients [72]. In most cases, *S. epidermidis* causes several hospital-acquired infections including I) catheter-related bloodstream and surgical site infections (with frequency 50% to 70%), II) urinary tract infection, III) central nervous system shunt infections, IV) peritoneal dialysis-related infections, V) infections of indwelling prosthetic devices, VI) ophthalmologic infections and VII) pacemaker and cardiac defibrillator lead endocarditis, especially in immunocompromised patients [4, 78, 79]. There are various reports about the role of *S. epidermidis* in infective endocarditis among patients (Table 1). According to several reports, approximately 80% of NVE are cases caused by *S. epidermidis* [5, 11]. However, studies revealed that this organism is the predominant species isolated in PVE [70]. It is indicated that most *S. epidermidis* isolated from PVE cases have a nosocomial origin and are non-susceptible to methicillin; while, 59% of NVE isolates are susceptible to methicillin and acquired from the community [72]. Gentamicin and rifampin plus vancomycin are used for the treatment of PVE cases; however, antimicrobial treatment of methicillin-resistant *S. epidermidis* NVE performs just by a glycopeptide agent, such as vancomycin [69]. In conclusion, it is recommended that in the treatment of *S. epidermidis* IE, a combination of surgery and antibiotic therapy may be necessary. Moreover, it is suggested that in high-risk patients, blood cultures positive for *S. epidermidis* must be surveyed attentively, and suitable antibiotic susceptibility test is carried out.

2.7. *Staphylococcus hominis*

Staphylococcus hominis (*S. hominis*) are Gram-positive cocci and common commensals of human skin [81]. Similar to *S. epidermidis*, this bacterium is sensitive to desferrioxamine; however, unlike to *S. epidermidis*, *S. hominis* strains infrequently are resistant to novobiocin and produce acid from trehalose [84]. Results of a study revealed that *S. hominis* and *S. warneri* are the frequently isolated *Staphylococcus* species in the semen, prostatic secretions, and urethra, which comprise 14% of CoNS isolated from the body fluids [113]. Like other CoNS isolates, *S. hominis* may sporadically cause several infections in immunocompromised individuals and around prosthetic devices [80]. *S. hominis* as the potential pathogen, in lead endocarditis, is a rare agent; however, it is presumed that *S. hominis* via breaks in the host-defense barriers of the genitourinary tract, enter the body and cause IE after vasectomy [83, 113]. Furthermore, IE in the eustachian valve (Eustachian Valve Endocarditis (EVE)) due to *S. hominis* has been reported [80]. Besides, in a patient with hypertrophic obstructive cardiomyopathy, it is established that *S. hominis* lead to the native mitral valve bacterial endocarditis [84]. *S. hominis*, similar to *S. epidermidis*, produce the slime layer and can produce catheter induced IE. However, its pathogenicity ability is less than *S. epidermidis*, which isolates and rarely develops endocarditis [114].

2.8. *Staphylococcus capitis*

Staphylococcus capitis (*S. capitis*) as a member of CoNS isolates, constitutes 5% of all CoNS bacteria and is a resident commensal bacterium of the human scalp, face, neck, and ears [86]. Like to *S. aureus*, this organism can cause different severe infections including I) pneumonia, II) late-onset sepsis, III) urinary tract infection IV) catheter-associated bloodstream infections, V) device-associated bone and joint infections, VI) cellulitis VII) infective endocarditis, VIII) prosthetic valve endocarditis, and IX) native valve endocarditis [1, 2, 115]. These abnormalities mostly occur among immunocompromised patients or patients with an underlying cardiac abnormality [116]. In recent years, studies revealed that *S. capitis* is a significant pathogen among infants with very-low-birth-weight (<1,500 g) and can cause late-onset sepsis in this group of subjects [115]. An overview of the published studies from 2000 to 2020 revealed that only eleven cases of IE (PVE, NVE, and implanted transvenous pacemaker) caused by *S. capitis* had been reported (Table 1). NVE caused by *S. capitis* has a favorable prognosis and, in most cases, is treated with various antibiotics including vancomycin, rifampin, nafcillin, gentamicin, daptomycin, Minomycin, and teicoplanin alone even in the existence of a significant embolic phenomenon [1, 85]. This bacterium, like other CoNS, produces biofilm on indwelling medical devices and is resistant to various disinfectants used during surgery. However, unlike most CoNS, *S. capitis* has a low attachment ability to foreign body surfaces [87]. Moreover, it is demonstrated that *S. capitis* produces exoenzymes such as DNase with extended incubation that is a recognized virulence factor of *S. aureus*. This enzyme could play a central role in the pathogenesis of both PVE and NVE [85]. On the other hand, this bacterium expresses the *mecA* gene and encode penicillin-binding protein 2a (PBP2a), which leads to resistance to β -lactam antibiotics such as methicillin. However, *S. capitis* is commonly susceptible to different antibiotics, such as chloramphenicol, vancomycin, cephalosporins, clindamycin, and aminoglycosides [117]. In general, Native valve endocarditis due to *S. capitis* is treated with antibiotic therapy and is usually managed conservatively with excellent outcomes.

CONCLUSION

Generally, CoNS species are isolated from the skin in individuals as healthy microbiota and considered as an uncommon cause of severe infections in hospitalized individuals, especially in immunocompromised patients. Among CoNS species, *S. epidermidis* and *S. lugdunensis* frequently isolated from both PVE and NVE cases. *S. lugdunensis* should be considered as an organism that has high levels of pathogenicity. According to what was announced about frequency, treatment, and role of CoNS species in IE, it is concluded that species diagnosis of CoNS in clinical specimens is vital, and physicians should not overlook low virulence CoNS species. Moreover, it is suggested that in the treatment of CoNS endocarditis, a combination of surgery and antibiotic therapy may be necessary.

AUTHOR CONTRIBUTION

All authors made substantial contributions to the conception and design, acquisition of data, or analysis and interpre-

tation of data. They played an active role in drafting the article or revising it critically to achieve compelling intellectual content, gave the final approval of the version to be published, and agreed to be accountable for all aspects of the work.

CONSENT FOR PUBLICATION

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CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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