

Original Article

Right-sided infective endocarditis: recent epidemiologic changes

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Abstract: Background: Infective endocarditis (IE) has been increasingly reported, however, little is available regarding recent development of right-sided IE. Methods: Right-sided IE was comprehensively analyzed based on recent 5½-year literature. Results: Portal of entry, implanted foreign material, and repaired congenital heart defects were the main predisposing risk factors. Vegetation size on the right-sided valves was much smaller than those beyond the valves. Multiple logistic regression analysis revealed that predisposing risk factors, and vegetation size and locations were independent predictive risks of patients' survival. Conclusions: Changes of right-sided IE in the past 5½ years included younger patient age, and increased vegetation size, but still prominent *Staphylococcus aureus* infections. Complication spectrum has changed into more valve insufficiency, more embolic events, reduced abscess formation, and considerably decreased valve perforations. With effective antibiotic regimens, prognoses of the patients seemed to be better than before.

Keywords: Anti-bacterial agents, blood-borne pathogens, cardiac surgical procedures, complications, embolism

Introduction

Infective endocarditis (IE) involves the aortic valve the most common, the mitral valve more common, and tricuspid and pulmonary valve the least common. Multiple valve involvements were seen in 17-22% of the patients: aortic plus mitral valves the most common, mitral plus tricuspid valves more common, and aortic plus tricuspid and aortic plus pulmonary valves the least common [1, 2]. Right-sided IE occupied 5-10% of all IE [3]. In the patients with congenital heart defects, left-, right- and both-sided IE accounted for 46.4%, 32.7% and 2.3%, respectively [4]. The prevalence of isolated tricuspid and pulmonary valve IE was 2.5-3.1% [5] and 2% [6], respectively.

It is notable that continuous changes have taken place with regard to epidemiology and prophylactic strategies of IE in the past decades [7]. Indwelling catheter, foreign medical device implants, and intravenous drug abusers have become the increasing risk factors for bacterial colonization, thus being a source of bacteremia [7, 8]. *Staphylococcus aureus* has become the

most common microorganism of IE, while *Streptococcus viridans* infections reduced. The novel trends of IE resulted in significant increases in mortality and morbidity irrespective of advanced modern diagnostic and therapeutic strategies [9]. However, little information is available regarding for the recent development of right-sided IE. This study aims at presenting the changing trends of epidemiology, predisposing risk factors, microbiology, and prognosis of right-sided IE under current antimicrobial treatments based on recent 5½-year literature.

Materials and methods

Recent 5½-year literature retrieval from January 1, 2008 to April 30, 2013 was made in PubMed database and Google search engine. The search terms included "right heart endocarditis", "right-sided endocarditis", "pulmonary valve endocarditis", "tricuspid valve endocarditis", "pacemaker lead endocarditis", "atrial septal defect endocarditis", "ventricular septal defect endocarditis", "Chiari network endocarditis", "Eustachian valve endocarditis", "pulmonary artery endarteritis", and "multiple valve

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Table 1. Clinical manifestations of 166 patients

Clinical manifestation	n (%)
Fever	152 (91.6)
Lethargy/fatigue/malaise/weakness	53 (31.9)
Dyspnea	37 (22.3)
Cough	29 (17.5)
Weight loss	17 (10.2)
Chest pain	18 (10.8)
Night sweats	12 (7.2)
Back pain	6 (3.6)
Abdominal pain	4 (2.4)
Congestive heart failure	10 (6.0)
Pleural effusions	12 (7.2)
Pericardial effusions	9 (5.4)
Hepatomegaly	11 (6.6)
Splenomegaly	5 (3.0)
Ascites	2 (1.2)
Skin lesions	11 (6.6)
Rash	2 (1.2)
Erythema nodosum migrans	1 (0.6)
Erythematous painful nodules	1 (0.6)
Janeway lesion	1 (0.6)
Macules extremity	1 (0.6)
Osler nodes	1 (0.6)
Petechiae	1 (0.6)
Petechial rash	1 (0.6)
Purpura	1 (0.6)
Purpuric rash	1 (0.6)

endocarditis". Libman-Sacks nonbacterial endocarditis caused by antiphospholipid syndrome, Loeffler's endocarditis, and patent ductus arteriosus endarteritis was not included. Patients with IE described as a long-term morbidity of current admission, with a history of right-sided IE, but current admission was for a left-sided one, or patients with non-active (healed) IE, were excluded.

Quantitative data were presented in mean \pm standard deviation with range and median. Comparisons of frequencies were made by Fisher's exact test. One-way ANOVA was taken for the univariant analysis. Multiple logistic regressions were used for predictive evaluation of patient survival/mortality. $p < 0.05$ was considered statistically significant.

Results

Literature retrieval yielded 401 publications. Following the exclusion criteria, totally 168 pub-

lications (14 original articles, 8 case series, and 146 case reports) [5, 10-176] including 299 patients were obtained. Gender was not reported for 37 patients. The remaining 262 patients included 182 males and 80 females with a male-to-female ratio of 2.3:1. The patients were at the age of 40.2 ± 21.3 (range, 0.04-88; median 41) years ($n=195$).

On admission, 76 patients had their cardiac murmurs recorded: 68 (89.5%) patients had a cardiac murmur (60 were systolic, 5 were diastolic, 2 were continuous, and 1 was both diastolic and diastolic), and 8 (10.5%) did not have a cardiac murmur. Locations of the cardiac murmurs were described in 50 patients: 25 (50%) at the left parasternal boarder, 11 (22%) at the tricuspid area, 5 (10%) at the right parasternal boarder, 4 (8%) at the mitral area, 3 (6%) at the pulmonary area, 1 (2%) at the mitral and tricuspid areas and 1 (2%) at the pulmonary and tricuspid areas.

Fourteen (4.7%) patients were afebrile, while 285 (95.3%) patients were febrile with a body temperature of 38.8 ± 0.7 (range, 37.5-40.3; median, 38.9) °C ($n=56$). Clinical manifestations were depicted in **Table 1**. Their heart rate was 102.8 ± 23.5 (40-152; median, 105) /min ($n=50$), systolic blood pressure 116.9 ± 22.7 (60-167; 120) mmHg ($n=47$), diastolic blood pressure 70.2 ± 13.4 (38-97; 70) mmHg ($n=45$), and respiratory rate 25.1 ± 6.5 (18-38; 22.5) /min ($n=18$). Arterial oxyhemoglobin saturation with room air was 93.7 ± 6.3 (range, 78-100; median, 97) % ($n=18$), and arterial oxyhemoglobin saturation with low-flow oxygen mask was 95-98% and 91% in 2 patients, respectively. Pulmonary artery hypertension was present in 21 (7.0%) patients with a systolic pulmonary arterial pressure of 54.4 ± 14.9 (40-90; 48) mmHg ($n=15$). Pulmonary arterial hypertension was moderate in 17 (81.0%), severe in 3 (14.3%), and classification of pulmonary arterial hypertension was unknown in 1 (4.8%) patient, respectively.

Predisposing risk factors for the occurrence of IE could be summarized into: portal of entry (36.1%), implanted foreign material (27.0%), underlying heart disease (22.8%), invasive dental, medical or surgical procedure (12.4%), distant infections (6.6%), a history of IE (2.5%), miscellaneous risk factors (23.2%), and no risk factor at all (1.7%). Intravenous drug user, pacer-

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Table 2. Predisposing risk factors in 241 patients with infective endocarditis

Predisposing risk factor	n (%)	p value (Fisher's exact test)
Portal of entry	87 (36.1)	<0.0001
Intravenous drug user	71 (29.5)	
Hemodialysis	8 (3.3)	
Central venous catheter	7 (2.9)	
Maintenance of blood transfusion for anemia	1 (0.4)	
Implanted foreign material	65 (27.0)	<0.0001
Pacemaker implantation	38 (15.8)	
Congenital heart defect surgery with a conduit, shunt or patch	16 (6.6)	
Prosthetic valve prosthesis (1 was percutaneous pulmonary valve implantation)	9 (3.7)	
Superior vena cava filter deployment	1 (0.4)	
Peritoneovenous (LeVeen) shunt after hepatic lobectomy	1 (0.4)	
Underlying heart disease	55 (22.8)	<0.0001
Congenital heart disease, unrepaired	51 (21.2)*	
Coronary artery disease	3 (1.2)	
Valvular heart disease	1 (0.4)	
Invasive dental, medical or surgical procedure	30 (12.4)	0.7870 (by excluding the variables with n≤2)
Dental problem	7 (2.9)	
Previous surgical operation (other than heart operation)	6 (2.5)	
Previous heart valve repair (probably with no implanted foreign material)	4 (1.7)	
Coronary artery bypass grafting	4 (1.7)	
Induced abortion	4 (1.7)	
Invasive diagnostic means (catheterization, prostate biopsy)	2 (0.8)	
Percutaneous transluminal coronary angioplasty	1 (0.4)	
Balloon dilation of pulmonary stenosis	1 (0.4)	
Acupuncture	1 (0.4)	
Distant infections	16 (6.6)	<0.0001
Abscess formation	13 (5.4)	
Infectious disease	2 (0.8)	
Gangrene of the foot	1 (0.4)	
History of infective endocarditis	6 (2.5)	--
Miscellaneous risk factors	56 (23.2)	<0.0001 (by excluding the variables with n≤2)
System disease (diabetes mellitus 2, sclerosis and systemic lupus erythematosus)	20 (8.3)	
Pregnancy	7 (2.9)	
Alcoholic consumption and alcoholic disease	6 (2.5)	
Cancer (including leukemia)	5 (2.1)	
Trauma and traumatic complication	5 (2.1)	
Postpartum	3 (1.2)	
Sporadic marihuana	2 (0.8)	
Parasite infection	2 (0.8)	
Animal bite/scratch	2 (0.8)	
Skin disease	2 (0.8)	
Necrotizing enterocolitis	1 (0.8)	
Arthritis	1 (0.4)	
Nil	4 (1.7)	--

*p<0.0001 comparing with "congenital heart defect surgery with a conduit, shunt or patch".

maker implantation and unrepaired congenital heart disease represented the first three predominant risk factors, respectively (**Table 2**). Unrepaired congenital heart disease accounted for 92.7% (51/55) of the underlying heart disease, while congenital heart defect surgery

with a conduit, shunt or patch accounted for 24.6% (16/65) of implanted foreign material ($\chi^2=56.05$, $p<0.0001$). Right-sided IE was due to cardiac surgical operations in 33 (11.0%) patients, including congenital heart defect surgery with a conduit, shunt or patch in 16

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Table 3. Management of infective endocarditis patients without a vegetation

Patient No.	Age (year)/sex	Predisposing risk factor	Blood culture	Antibiotics	Surgical indication	Surgical operation	Outcome	Reference
1	7/F	s/p Ross-Konno procedure plus mitral valvuloplasty for Shone syndrome	<i>Granulicatella adiacens</i>	vancomycin plus meropenem × 4 weeks	Subtotal obstruction of the conduit	Conduit replacement with a pulmonary valved homograft	Negative blood culture	[36]
2	5/M	Cardiac catheterization for infundibular pulmonary stenosis	<i>Granulicatella adiacens</i>	Ciprofloxacin and meropenem × 4 weeks	No	No	The blood cultures were sterile after one week antibiotic treatment	[36]
3	19/M	Cor triatriatum	<i>Staphylococcus aureus</i>	Intravenous vancomycin, gentamycin and meronem × 4 weeks	Stenosis of the right ventricular outflow tract and thick and narrow pulmonary valve	Stenosis of the right ventricular outflow tract and thick and narrow pulmonary valve resection; The membrane in the right atrium without causing any stenosis was left unresected	After antibiotherapy the body temperature returned to normal	[133]
4	61/M	Bicuspid aortic valve, non-coronary cusp perforation, ventricular septal defect, tricuspid pouch	<i>Streptococcus sanguis</i>	Penicillin and gentamicin	Bicuspid aortic valve, non-coronary cusp perforation, ventricular septal defect, tricuspid pouch	Ventricular septal defect closure, tricuspid repair (edge-to-edge), aortic valve replacement (23-mm Carpentier-Edwards Perimount)	Uneventful	[175]

Table 7. Positive results of 59 further microorganism investigations in 50 patients

Detection	Microorganism (n)
Culture	
Bronchioalveolar lavage fluid	<i>Aspergillus fumigatus</i> (1), <i>Staphylococcus aureus</i> (1)
Abscess	<i>Staphylococcus aureus</i> (4), coagulase-negative <i>Staphylococcus</i> (1)
Indwelling catheter	<i>Staphylococcus aureus</i> (2)
Pacemaker lead	<i>Propionibacterium</i> (1)
Valve tissue	<i>Enterococcus faecalis</i> and <i>Gemella morbillorum</i> (1), <i>Staphylococcus aureus</i> (1), <i>Staphylococcus lugdunensis</i> (2), <i>Propionibacterium</i> (1), <i>Enterococcus</i> (2), <i>Corynebacterium Diphtheriae</i> (1), <i>Candida albicans</i> (1), <i>Pseudomonas aeruginosa</i> (1)
Vegetation	<i>Aspergillus fumigatus</i> (1), <i>Candida tropicalis</i> (1), <i>Candida albicans</i> (1), α-hemolytic <i>streptococcus</i> (1), Methicillin-susceptible <i>Staphylococcus aureus</i> (1), <i>Streptococcus viridans</i> (1)
Knee joint aspirate	<i>Staphylococcus aureus</i> (1)
Pacemaker pocket discharge	<i>Staphylococcus epidermidis</i> (1)
Sputum	Methicillin-resistant <i>Staphylococcus aureus</i> (1)
Urine	<i>Escherichia coli</i> (2)
Pericardial effusion	<i>Staphylococcus aureus</i> (2)
Pulmonary artery emboli	<i>Candida albicans</i> (1)
Rectal swabs	<i>Escherichia coli</i> extended-spectrum β-lactamase (1)
Histology	
Vegetation	Gram-positive cocci (1), multiple bacterial colonies (cocci) (1), <i>Mycobacterium tuberculosis</i> (1)
Valve tissue	Gram-positive cocci (1), numerous budding organism (1)
Transbronchial lung biopsy	<i>Aspergillus</i> spp. (1)
Erythematous painful nodules in the foot	<i>Fusarium solani</i> (1)
Microbiologic study	
Valve tissue	Yeasts (1), <i>Aspergillus hyphae</i> (1), methicillin-resistant coagulase-negative <i>Staphylococcus</i> (1)
Vegetations	<i>Aspergillus hyphae</i> (1)
Polymerase chain reaction	
Valve tissue	<i>Hemophilus aphrophilus</i> (1), <i>Bartonella</i> species (1), <i>Bartonella Quintana</i> (1), <i>Bartonella hensellae</i> (1)
Pleural fluid	<i>Tropheryma whipplei</i> (1)
Blood	<i>Tropheryma whipplei</i> (1), <i>Neisseria</i> (1)
Vegetation	<i>Streptococcus agalactiae</i> (1)
Serology	<i>Bartonella hensellae</i> + <i>Bartonella quintana</i> (1), <i>Coxiella Burnetti</i> (1), <i>Bartonella quintana</i> (1), <i>Bartonella hensellae</i> (1); <i>Bartonella</i> (1)

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(48.5%), heart valve replacement in 9 (27.3%), heart valve repair in 4 (12.1%), and coronary artery bypass grafting in 4 (12.1%), respectively ($\chi^2=16.84$, $p=0.000762$).

Hemoglobin was 9.6 ± 2.5 (range, 4.2-17; median, 9.8) g/dl ($n=50$). Anemia was noted in 40 (80%) patients: 18 (45%) were mild, 20 (40%) were moderate, and 2 (4%) were severe anemia, respectively. Leukocyte count was reported in 97 (32.4%) patients with a value of 19.2 ± 19.4 (range, 2.9-16.7; median, 16.5) $\times 10^9/L$ ($n=73$). Fourteen (14.4%) patients had a normal white count, 82 (84.5%) patients had leukocytosis, and 1 (1.0%) patient had leukopenia. The platelet count was reported in 19 patients, which was 114.1 ± 120.5 (range, 7-404; median, 67) $\times 10^9/L$ ($n=19$). There were 12 (63.2%) cases of thrombocytopenia: 5 (41.7%) were mild, 3 (25%) were moderate, 1 was (8.3%) severe, and 3 (25%) were extremely severe thrombocytopenia. Erythrocyte sedimentation rate was reported in 46 (15.4%) patients: 44 (95.7%) patients were positive, and 2 (4.3%) were normal. The quantitative value of the abnormal erythrocyte sedimentation rate was 80.2 ± 38.0 (range, 8-140; median, 77) mm/h ($n=26$). Of the 90 (30.1%) patients with a reported C-reactive protein, 89 (98.9%) were positive, and 1 (1.1%) was normal. The quantitative C-reactive protein value was 21.3 ± 42.3 (0.15-297; median, 10.9) mg/dl ($n=66$). Serum creatinine was reported in 17 (5.7%) patients: 6 (35.3%) patients had a normal value 0.9 ± 0.2 (range, 0.6-1.2; median, 0.98) mg/dl ($n=6$), and 11 (64.7%) patients had an elevated value 2.0 ± 0.6 (range, 1.45-3.3; median, 1.9) mg/dl ($n=11$). The overall creatinine was 1.6 ± 0.7 (range, 0.6-3.3; median, 1.6) mg/dl ($n=17$), aspartate aminotransferase 126 ± 171.9 (33-573; 48) IU/L (normal reference, 7-56 IU/L) ($n=9$), and alanine transaminase 76.7 ± 67.2 (range 27-268; median, 59) IU/L (normal reference, 5-40 IU/L) ($n=11$). Aspartate aminotransferase and alanine transaminase were elevated in 6 patients each.

Both transthoracic and transesophageal echocardiographic studies were carried out for the diagnosis of intracardiac vegetations in 33 patients: identical results were obtained in 18 (54.5%) patients (however, the origin of the vegetation was not clearly visualized by transthoracic, but clearly visualized by transesophageal echocardiography); at least one vegetation was missed by transthoracic but supplemented

information was obtained by transesophageal echocardiography in 15 (45.5%) patients. In 51 (79.7%) of the surgical patients, echocardiographic vegetations conformed to the surgical exploration, however, transthoracic or transesophageal echocardiographic misdiagnoses of vegetations were disclosed by open heart surgery in 13 (20.3%) patients.

The locations of the vegetations were not given in 63 patients. Four (1.7%) patients did not have a vegetation. The managements of the four patients with no vegetation varied according to patient's age, predisposing risk factor (previous surgical maneuvers), and cardiac situations, etc. (Table 3). A single or multiple vegetations were found involving the right heart in 232 (98.3%) patients. Most of the vegetations were single on a single valve/site of the right heart. Multiple vegetations on a single right heart valve, multiple vegetations on multiple sites of the right heart and multiple vegetations on both sides of the heart totally amounted to one-third of the whole presentation (Table 4). The size of the right-sided vegetations was 1.96 ± 1.16 (range, 0.14-7; median, 1.75) mm ($n=114$). For the single vegetations of right-sided IE, no significant difference was found in the vegetation sizes between tricuspid and pulmonary valve IE (1.84 ± 0.76 mm vs. 1.77 ± 1.33 , $p=0.8420$). Vegetations developed on the right-sided valves (both tricuspid and pulmonary valves) were much smaller than those occurred beyond the valves on the right atrial or right ventricular free walls, superior vena cava, superior vena cava-right atrium junction, ventricular septal defect patch, or the pacemaker lead (1.82 ± 0.88 mm vs. 3.33 ± 1.45 mm, $p<0.0001$). Multiple vegetations on one valve (site) of the right heart were smaller than those of the single vegetation on one valve (site), but did not reaching a significant difference (1.63 ± 0.78 mm vs. 2.08 ± 1.14 mm, $p=0.0755$). Multiple vegetations on multiple sites of the right heart measured 2.00 ± 1.85 mm, and multiple vegetations on multiple sites of both the left and right heart measured 1.75 ± 1.29 mm.

Complications amounted to 251, which developed in 161 patients with a mean of 1.6/patient. Valvular insufficiency, embolic events and abscess formation were the most common complications of right-sided IE, representing 49.1% (79/161), 52.8% (85/161) and 15.5% (25/161), respectively. Leaflet perforation only

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Table 4. Locations of vegetation in 232 patients

Location of vegetation	n (%)
Single vegetation on a single right heart valve/site	147 (63.4)
TV	83 (35.8)
Pacemaker lead	19 (8.2)
PV	18 (7.8)
RV-PA conduit	7 (3.0)
RA free wall	5 (2.2)
RV free wall	5 (2.2)
VSD	3 (1.3)
Prosthetic PV	2 (0.9)
SVC	2 (0.9)
SVC-RA junction	1 (0.4)
Waterston shunt	1 (0.4)
Prosthetic TV	1 (0.4)
Multiple vegetations on a single right heart valve	32 (13.8)
TV	21 (9.1)
PV	10 (4.3)
Prosthetic PV	1 (0.4)
Multiple vegetations on multiple sites of the right heart	26 (11.2)
PV, RVOT, PA	3 (1.3)
PV, PA	3 (1.3)
TV, pacemaker lead	2 (0.9)
TV, RA	2 (0.9)
TV, PV	2 (0.9)
RV, VSD	2 (0.9)
PV, RVOT	2 (0.9)
RA, RV	2 (0.9)
TV, TV apparatus, VSD, adjacent endocardium	1 (0.4)
TV, TV papillary muscles, pacemaker lead	1 (0.4)
TV, Chiari network	1 (0.4)
TV, SVC, RA, RV	1 (0.4)
SVC, RA	1 (0.4)
TV, RV	1 (0.4)
PV, VSD patch	1 (0.4)
RA, pacemaker lead	1 (0.4)
Multiple vegetations on both sides of the heart	27 (11.6)
AV, TV	6 (2.6)
MV, TV	5 (2.2)
AV, MV, TV	3 (1.3)
AV, MV, PV	2 (0.9)
All 4 valves	1 (0.4)
AV, PV	1 (0.4)
AV, TV, pacemaker lead	1 (0.4)
AV, VSD near TV	1 (0.4)
Both sides of VSD patch	1 (0.4)
MV, aorta, RV	1 (0.4)
MV, PA	1 (0.4)
MV, VSD	1 (0.4)
Prosthetic MV, PV	1 (0.4)
TV, LA free wall	1 (0.4)
TV, RVOT, LA free wall	1 (0.4)

AV: aortic valve; LA: left atrium; MV: mitral valve; PA: pulmonary artery; PV: pulmonary valve; RA: right atrium; RV: right ventricle; RVOT: right ventricular outflow tract; SVC: superior vena cava; TV: tricuspid valve; VSD: ventricular septal defect.

cellaneous, complications of valvular insufficiency, embolic events, and abscess formation were the most common in portals of entry (most of which was intravenous drug abuse), which were 23.6% (17/72), 22.5% (16/71), and 34.8 (8/23), respectively. For vegetation location groups, single vegetation on single right-sided valve/site developed the three complications the most accounting for 54.9% (28/51), 61.4% (43/70) and 54.5% (12/22), respectively. Significant inter-group differences were noted (**Table 5**).

Microorganisms were identified either by blood cultures and/or by further investigations like serology, polymerase chain reaction, microbiology, or histology in the whole patient setting except for one patient who was still diagnosed as IE in the absence of microorganism evidence [157]. Blood culture results were unavailable in 101 patients. In the remaining 198 patients, blood cultures were sterile in 31 (15.7%) patients, and were positive in 167 (84.3%) patients. Staphylococci represented half (50.9%) of the causative primary microorganisms, followed by Streptococci (10.8%), Enterococci (9.0%) and fungi (4.2%) ($\chi^2=142.18$, $p<0.0001$) (**Table 6**). Miscellaneous Gram-negative bacilli accounted for a considerable proportion, much more than Gram positive bacilli ($\chi^2=32.05$, $p<0.0001$). Seven (4.2%) patients had polymicrobial infections: *Escherichia coli* and coagulase-negative *Staphylococcus* ($n=1$), *Enterococcus faecalis* and *Gemella morbillorum* ($n=1$), *Enterococcus faecalis* and *Candida albicans* ($n=1$), *Peptostreptococcus*, *micrococcus* and coagulase-negative *Staphylococcus* ($n=1$), *Pseudomonas aeruginosa*, Methicillin-resistant *Staphylococcus aureus* and *Klebsiella pneumoniae* ($n=1$), and *Enterococcus* and *Pseudomonas* ($n=1$), and *Escherichia coli* extended-spectrum β -lactamase (ESBL) and *Enterococcus faecalis* ($n=1$).

Further 79 microorganism investigations were performed in 63 patients by way of serology, polymerase chain reaction, microbiology, or histology of resected tissues, catheter or other body fluids. Negative results were noted in 17 (25.3%) tests in 17 patients. The remaining 59 (74.7%) positive tests in 50 patients were summarized in **Table 7**. Two (4%) patients had polymicrobial infections: *Bartonella henselae* and *Bartonella Quintana* ($n=1$) (serologies) and *Pseudomonas aeruginosa* and *Aspergillus fumigatus* ($n=1$) (culture of surgically resected vegetation).

accounted for 6.2% (10/161). For diverse predisposing risk factors by putting aside the mis-

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Table 5. 251 complications of infective endocarditis in 161 patients (Fisher's exact test)

Complications	Total (n)	Predisposing risk factor (n)						p value	Location of vegetation (n)				p value
		PoE	IFM	Inv	UHD	DI	MRF		I	II	III	IV	
Valvular insufficiency	79*	17	10	6	17	4	18	0.001	36	13	6	10	<0.0001
TR	65	12	9	5	15	4	18		28	12	4	7	
PR	10	4	1	1					4	1	2	2	
TR, PR	3	1			2				4				
RV-PA conduit regurgitation	1											1	
Valvular stenosis	13	5	2	1	1	0	2	0.138	7	2	1	0	0.002
PV	8	3	1	1	1		1		5	1	1		
TV	4	2					1		1	1			
PV conduit	1		1						1				
Embolus event	85	16	6	7	10	6	26	<0.0001	43	12	10	5	<0.0001
Lung	75	12	6	6	9	6	25		38	12	8	2	
Lung, spleen	1	1							1				
Lung, kidney	1	1							1				
Spleen, kidney	1	1							1				
Brain	5	1		1			1		2			3	
Spleen	1				1						1		
Small intestine, spleen, brain	1										1		
Valve destruction	13	4	1	0	3	0	3	0.063	4	4	4	1	0.418
TV	4	1			1		2		2	1	1		
PV	9	3	1		2		1		2	3	3	1	
Valve leaflet perforation	10	2	2	2	3	0	0	0.310	5	1	0	2	0.038
TV	4	1		1	2				2	1		1	
PV	3	1	1	1					2				
MV	2				1				1			1	
AV	1		1										
Abscess formation	25	8	0	5	4	1	5	0.011	12	4	3	3	0.008
Lung	9	3		1	3	1	1		5		2		
Aortic root + RV free wall	1				1					1			
AV-TV annulus	1			1									
Para-AV annulus	2	1					1		1			1	
Paraspinal and epidural	1	1							1				
Pulmonary outflow tract	1	1							1				
Purulent material of VSD extending to the AV	1	1							1				
PV annulus	2	1					1		1	1			
RA	1										1		
Root	1			1								1	
RV	1									1			
Skin cold abscess + purulent pericarditis	1			1					1				
TV ring	1			1								1	
Ventricular septum	1						1			1			
Brain	1						1		1				
Infarct of parenchymatous organ	10	3	0	1	2	0	4	0.108	4	1	5	1	0.171
Lung				1	1		2			1	3		
Spleen		1			1				2		2		
Kidney		1							1				
Brain		1					2		1			1	
Embolus stroke	2								1	0	0	1	1.000
AVB	3	2	0	0	1	0	0	0.509	3	0	0	0	0.018
Renal failure	5	1	1	1	2	0	0	0.868	4	0	1	0	0.020
Septic shock	4	0	0	0	3	0	1	0.046	2	0	0	2	0.086
Multiple organ failure	2	1	0	0	1	0	0	1.000	1	0	0	4	0.020
Total	251	59	22	23	47	11	59	—	122	37	30	29	—

*p=0.0002 comparing with valve stenosis; PoE: Portal of entry; IFM: Implanted foreign material; Inv: Invasive dental, medical or surgical procedure; UHD: Underlying heart disease; DI: Distant infections; MRF: Miscellaneous risk factors; I: Single vegetation on a single right heart valve/site; II: Multiple vegetations on a single right heart valve; III: Multiple vegetations on multiple sites of the right heart; IV: Multiple vegetations on both sides of the heart; TR: tricuspid regurgitation; PR: pulmonary regurgitation; RV: right ventricle; PA: pulmonary artery; PV: pulmonary valve; TV: tricuspid valve; MV: mitral valve; AV: aortic valve; RA: right atrium; AVB: atrioventricular heart block.

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Table 6. Microorganisms identified by blood culture in 167 patients

Microorganism	n (%)
<i>Staphylococcus</i>	85 (50.9)
<i>aureus</i>	74 (44.3)
Methicillin-sensitive <i>Staphylococcus aureus</i>	21 (12.6)
Methicillin-resistant <i>Staphylococcus aureus</i>	14 (8.4)
<i>epidermidis</i>	3 (1.8)
<i>lugdunensis</i>	3 (1.8)
coagulase-negative	3 (1.8)
<i>capitis</i>	1 (0.6)
<i>haemolyticus</i>	1 (0.6)
<i>Streptococcus</i>	18 (10.8)
<i>viridan</i>	6 (3.6)
<i>agalactiae</i>	5 (3.0)
<i>sanguis</i>	3 (1.8)
<i>intermedius</i>	1 (0.6)
<i>milleri</i>	1 (0.6)
<i>pyrogenes</i>	1 (0.6)
<i>sobrinus</i>	1 (0.6)
<i>Enterococcus</i>	15 (9.0)
<i>faecalis</i>	11 (6.6)
<i>gallinarum</i>	1 (0.6)
Fungus	8 (4.2)
<i>Candida Albican</i>	4 (2.4)
<i>Absidia corymbifera</i>	1 (0.6)
<i>Candida parapsylosis</i>	1 (0.6)
<i>Candida tropicalis</i>	1 (0.6)
<i>Kodamaea (Pichia) ohmeri</i>	1 (0.6)
Gram-negative bacilli	32 (19.2)
<i>Pseudomonas</i>	7 (4.2)
<i>Neisseria</i>	2 (1.2)
<i>Suttonella indologenes</i>	1 (0.6)
<i>Stenotrophomonas maltophilia</i>	1 (0.6)
<i>Salmonella enteritidis</i>	1 (0.6)
<i>Haemophilus parainfluenzae</i>	1 (0.6)
<i>Mycobacterium fortuitum</i>	1 (0.6)
<i>Pasteurella multocida</i>	1 (0.6)
<i>Pasteurella pneumotropica</i>	1 (0.6)
<i>Achromobacter xylosoxidans</i>	1 (0.6)
<i>Aggregatibacter actinomycetemcomitans-HACEK</i>	1 (0.6)
<i>Escherichia coli</i>	6 (3.6)
<i>Brucella</i>	5 (3.0)
<i>Capnocytophaga canimorsus</i>	2 (1.2)
<i>Burkholderia cepacia</i> complex	1 (0.6)
Gram-positive bacilli	7 (4.2)
<i>Granulicatella adiacens</i>	2 (1.2)
<i>Gemella</i>	1 (0.6)
<i>Peptostreptococcus, micrococcus</i>	1 (0.6)
<i>Pneumococcus</i>	1 (0.6)
<i>Propionibacterium</i>	1 (0.6)
<i>Erysipelothrix rhusiopathiae</i>	1 (0.6)
Gram-negative coccus	1 (0.6)
Gram-positive coccus	1 (0.6)

Management was not indicated in 71 patients. Of the remaining 228 patients, conservative treatments with antibiotic therapy without a cardiac operation were undertaken in 75 (32.9%) patients: pacemaker lead removal, pacemaker reimplantation, indwelling catheter removal and local abscess drainage were performed in 26, 3, 3 and 2 patients, respectively. Patients with *Staphylococcus aureus* infection and with non-*Staphylococcus aureus* infections showed an approximate statistical significance in terms of mortality [8.8% (5/57) vs. 17.7% (23/130), $\chi^2=1.89$, $p=0.0535$]. For the patients with *Staphylococcus aureus* infection, mono-antibiotic therapy and 2 or more antibiotic therapy regimens were listed in **Table 8**. They caused no difference in patients' mortality [14.3% (2/14) vs. 4.2% (1/24), $\chi^2=0.33$, $p=0.2757$]. Following blood cultures, antibiotic regimens were modified in 12 patients with *Staphylococcus aureus* infection and 20 patients in patients with non-*Staphylococcus aureus* infections ($\chi^2=0.90$, $p=0.3997$). The antibiotic adjustment following blood culture were unchanged, less and more than before in 7, 4 and 1 patients in *Staphylococcus aureus* infection, and in 6, 7 and 7 patients in non-*Staphylococcus aureus* infection patients, respectively ($\chi^2=0.02$, $p=0.1553$). During antibiotic treatment following blood culture, further antibiotic adjustments were necessary in 9 patients (once adjustment in 7 and twice adjustments in 2 patients) due to bacterial changes, being complicated by cough, sputum, and persistent fever, associated fungal infection, unknown etiology of the Gram-negative bacilli and the severity of the disease, blood cultures obtained eight days after starting imipenem/cilastatin therapy growing *Alcaligenes xylosoxidans* subspecies *denitrificans*, discharge home, critical infection, increased sized vegetation and initial antibiotic allergy. Deference, sterile blood culture, vegetation dwindling, and vegetation disappearance were noted at 27.4 ± 68.7 (range, 2-365; median, 10) days ($n=27$), 30.1 ± 32.8 (range, 1-168; median, 23) days ($n=27$), 33.4 ± 15.5 (range, 10-60; median, 36) days ($n=17$) and 34.0 ± 14.6 (range, 10-56; median, 36) days ($n=8$) following antibiotic use after admission.

Surgical operations were performed in 153 (67.1%) patients, of which the right heart valve replacement was the most common procedure

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Table 8. Antibiotic regimens for *Staphylococcus aureus* and non-*Staphylococcus aureus* infections

Antibiotics	<i>Staphylococcus aureus</i>	Non- <i>Staphylococcus aureus</i>
Mono-antibiotics	cefazolin	ceftriaxone (2 g twice daily, 4-6 weeks)
	ceftriaxone	cloxacillin (8 weeks)
	cloxacillin	doxycycline, rifampin
	cloxacillin, gentamicin	ertapenem (10 weeks)
	flucloxacillin	flucloxacillin (6 weeks)
	nafcillin	imipenem/cilastatin
	penicillin G	penicillin G (3 weeks)
2 or more antibiotics	vancomycin	piperacillin/tazobactam (8 weeks)
		vancomycin (1 g/day, 5 weeks)
		amphotericin B (0.8-10 mg/ kg/day, 8 weeks)
	ampicillin/sulbactam, linezolid	amphotericin B, fluconazole (30 days)
	ceftriaxone, gentamicin	ampicillin, gentamicin
	cephradine, ciprofloxacin	ampicillin, nafcillin, gentamicin
	cephradine, gentamicin	ceftriaxone (50 mg/kg every 12 hours), gentamicin (0.7 mg/kg every 12 hours)
	cloxacillin, gentamicin	ceftriaxone, doxycycline, gentamicin
	flucloxacillin, gentamicin	flucloxacillin, rifampicin (6 weeks)
	flucloxacillin, gentamicin, rifampicin	gentamicin, doxycyclin
	flucloxacillin/tobramycin, furosemide, lisinopril	liposomal amphotericin B (2 weeks)
	imipenem, cilastatin, ciprofloxacin	meropenem, ciprofloxacin
	levofloxacin, piperacillin/tazobactam, vancomycin	doxycycline (oral) and hydrochloroquine (12 months)
	linezolid, meropenem	penicillin G, gentamicin (8 weeks)
	nafcillin, gentamicin	rifampin, gentamicin
	oxacillin, gentamicin, ceftriaxone	teicoplanin, ciprofloxacin (2 months)
	penicillin G, gentamicin	vancomycin (2 g/day), metronidazole (2 g/day) (2 weeks)
	piperacillin/tazobactam, erythromycin, fluconazole	vancomycin, cefotaxime, gentamicin
	teicoplanin, rifampicin	
	vancomycin, amikacin	
	vancomycin, arbekacin	
	vancomycin, ceftriaxone	
	vancomycin, gentamicin	
vancomycin, gentamycin, meronem		
vancomycin, rifampicin, daptomycin		

(Table 9). The indications for heart operations were enlarged vegetation ($n=8$), not regressed vegetation ($n=4$), progressed hemodynamic deterioration ($n=2$), enlarged Gerbode defect ($n=1$), persisted fever ($n=6$), heart failure ($n=4$), pulmonary edema ($n=1$), sepsis ($n=2$), emboli ($n=3$), valve dysfunction ($n=4$), and bacteremia ($n=4$) despite antibiotic use for 26.4 ± 18.1 (range, 4-77; median, 24.5) days ($n=42$). The indications for urgent heart operations were septic shock and disseminated intravascular coagulation ($n=1$), temperature 40°C with hemodynamic deterioration ($n=1$), and worsening heart failure ($n=1$). Two pregnant patients were operated on immediate after cesarean sections.

There were 268 survivors and 31 deaths with a survival rate of 89.6% and a mortality of 10.4%. Sixty-eight (90.7%) patients survived and 7 (9.3%) patients died in the conservative patients, and 141 (92.2%) patients survived and 12 (7.8%) patients died in the surgical patients. No statistical significance was present in the survival and mortality between conservative and surgical patients ($\chi^2=0.15$,

$p=0.7994$). Univariate analysis did not show any significant correlation between patients' survival and predisposing risk factors ($F=0.608$, $p=0.7700$), size of the vegetations ($F=0.725$, $p=0.8480$) or location of the vegetations ($F=2.330$, $p=0.0760$). Multiple logistic regression analysis revealed that the predisposing risk factors of IE, size of the vegetations, and location of the vegetations were independent predictive risks relative to patients' survival (Overall Model Fit: $\chi^2=7.9976$; $df=3$; $p=0.0461$). Of them, the location of the vegetations was of special statistical significance ($p=0.0082$). The odds ratios and 95% confidence intervals were listed in Table 10.

Discussion

Murdoch *et al.* [177] made a prospective cohort study revealed that fever, temperature $>38^\circ\text{C}$ (96%), elevated C-reactive protein level (62%) and elevated erythrocyte sedimentation rate (61%) were the most common clinical manifestations of IE. New murmur was present in 48%. Persistent fever associated with pulmonary events, anemia, and microscopic hematuria,

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Table 9. Cardiac operations performed in 153 patients with infective endocarditis

Cardiac operations	n (%)
Valve replacement	47 (30.7)
Tricuspid/pulmonary valve repair	12 (7.8)
Valve debridement of vegetation	12 (7.8)
Valve repair, VSD closure	11 (7.2)
Valve replacement, valve repair	10 (6.5)
Valve replacement, VSD/ASD closure	9 (5.9)
Right ventricle-pulmonary artery conduit explantation	8 (5.2)
Leaflet replacement	7 (4.6)
Hypertrophic infundibular stenosis resection	4 (2.6)
Bentall operation, tricuspid/pulmonary valve repair	3 (2.0)
VSD closure	3 (2.0)
Right heart fistula repair	3 (2.0)
Percutaneous pulmonary valve implantation	2 (1.3)
VSD re-patch	2 (1.3)

ASD: atrial septal defect; VSD: ventricular septal defect.

Table 10. Odds ratios and 95% confidence intervals

Variable	Odds Ratio	95% confidence intervals	
		Low	High
Predisposing risk factor	1.1846	0.8040	1.7455
Size of the vegetation	1.0082	0.5799	1.7526
Location of the vegetation	0.4304	0.2304	0.8041

the so-called “tricuspid syndrome”, are the signs of clinical alert for tricuspid valve IE [178]. Fever, multiple pulmonary emboli and sustained bacteremia by *Staphylococcus aureus* are signs of clinical alert for right-sided IE [179].

Symptoms related to pulmonary emboli usually forced patients to seek medical attention and dominated the clinical picture. Pulmonary events occurred in 80% of these cases, and varied from minor atelectases to large infiltrates, pleural exudates and cavitation, generally involving the lower lobes [5]. The simultaneous occurrence of multiple right and left circulation septic emboli led to the suspicion of an undiagnosed intracardiac shunt, and a repeated transthoracic echocardiogram with an agitated saline bubble contrast study was performed. No intracardiac shunt between right and left chambers was found; however, delayed appearance of bubbles in the left heart chambers was suggestive of an intrapulmonary shunt [103]. Multiple organ involvements were reported in 49% IE patients: brain (49), spleen (34), lung (13), bone and joints (13), kidney (10), and liver (3) [1]. In IE (including both left- and

right-sided), the prevalence of leaflet perforation (52.9%) and abscess formation (47.1%) was high. Valve repair techniques varied with sliding plasty, patch repair or commissural reconstruction according the locations of valve destruction and surgeons' preferences [180]. Apparent embolic events occurred in 46% (23/50) pediatric IE patients: brains 34.8 (8/23), lungs 47.8% (11/23), limbs 8.7% (2/23), intestine 8.7% (2/23), kidney 4.3% (1/23) and spleen 4.3% (1/23), respectively. In adults, embolic events were more common with right-sided vegetations than with left-sided vegetations [181]. Abscess formation developed in 36% of IE patients. Majority (81%) of them had a history of surgery, either for valve replacement and debridement of a cardiac abscess, or removal of an infected device [149]. Renal insufficiency was thought to be secondary to IE, because the abdominal ultrasonography and urinalysis showed no pathologic findings [55]. In the patients with congenital heart defect, right-sided IE involved the tricuspid valve (15.2%), pulmonary valve (8.8%), ventricular septal defect (5.2%), right ventricle (2.9%), pulmonary artery (2.3%), right ventricle-pulmonary artery conduit (2.1%), pulmonary and tricuspid valves (1.2%) and atrial septal defect (0.4%) [4].

Staphylococcus aureus IE was the most common organism and more likely to affect the tricuspid valve in intravenous drug users, and tricuspid valvular IE occurred more frequently in heroin users [182]. There was a significant association between *Staphylococcus aureus* etiology and tricuspid valve involvement with a mortality rate of 16% [183]. Pacemaker or implantable cardioverter defibrillators accounted for 4.6% of IE, while electrode lead endocarditis occurred in less than 1%. Conservative treatment without explantation of the devices resulted in 100% failure of treatment, but combined surgical and antibiotic therapy was associated with a mortality rate of 12.5% in spite of an effective infection control [184]. Prosthetic material increases the risk of associated infections, and IE remains one of the most common complications of congenital heart defects. Knirsch and Nadal [4] made a comparison

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between unpaired and paired congenital heart defects, and found the calculated ratio of post-interventional IE including all locations of either left-, right- or both-sided, multiple or extracardiac, in repaired congenital heart defects (41%) compared to IE in unrepaired congenital heart defects (59%) was 2:3. Nevertheless, the present study showed that patients with repaired congenital heart defects especially those with foreign material implants were of higher risks for right-sided IE than the patients with unrepaired congenital heart defects. Between 1970-1980, gram-negative microorganism (*Pseudomonas aeruginosa*) prevailed; whereas between 1981-1990, gram-positive (*Staphylococcus aureus*) predominated, and most (21/32) were antibiotic-resistant [185]. In the last several years, *Staphylococcus aureus* was 18% of the pathogen of right-sided IE, while *Staphylococcus aureus* was not the pathogen for multiple valve IE [186]. Responsible microorganisms for IE relating to central venous catheter infections included *Staphylococcus aureus* in 54.6%, coagulase-negative staphylococcus in 37.5%, *Candida* species (*spp.*) in 16.6%, and *Enterococcus* in 12.5%, and 5 cases were polymicrobial [187]. In intravenous drug use-related IE, *Staphylococcus aureus* was present in 50-75% [179]. In addition, right-sided IE should be suspected in any pneumonic illness that complicates post-abortion infection or other inadequately treated sepsis [188].

Acute IE is usually caused by infections of methicillin-susceptible or methicillin-resistant *Staphylococcus aureus*, streptococci, or enterococci. Empiric therapy for such infections would be vancomycin until microorganism evidence is available. Vancomycin-resistant *Enterococcus faecalis* may be susceptible to ampicillin and IE affected by this pathogen may be curable to synergistic ampicillin and gentamicin. Tigecycline, in combinations with other agents, has been reported to be effective for the treatment of vancomycin-resistant *Enterococcus faecalis* infections [189]. Daptomycin therapy was successful in 63% in patients with left-, right- or both-sided IE [190]. For refractory Methicillin-resistant *Staphylococcus aureus* IE the combination of vancomycin, rifampin and trimethoprim-sulfamethoxazole (SMZ/TMP) should be considered [46]. Linezolid may be useful in right-sided endocarditis

but further investigations are required [191]. Patients with right-sided IE more often required emergency operations [192]. Time from diagnosis to surgery in the acute phase of IE was 22.9 days [1]. Indications for cardiac operations were persisted fever despite effective antibiotic treatment (100/173), enlarged vegetation (50/173), recurrent embolism (22/173), annular abscess (21/173) and atrioventricular block (5/173) [1].

The present study illustrated the patients aged 40 years old at the time of right-sided IE onset, several years older than previously reported 27-35 years decades ago [188, 193], but similar to the 43 years old reported years ago [192]. Comparison to the literature, more patients in this cohort had fever $>38^{\circ}\text{C}$ and elevated erythrocyte sedimentation rate and C-reactive protein, and prevalence of *Staphylococcus aureus* IE and patients receiving a heart surgery increased. Portal of entry and implanted foreign material were the two main predisposing risk factors for right-sided IE, and the underlying heart disease previously reported as the principle cause have declined to the third place. Vegetations developed on the right-sided valves (both tricuspid and pulmonary valves) were much smaller than those occurred beyond the valves on the right atrial or right ventricular free walls, superior vena cava, superior vena cava-right atrium junction, ventricular septal defect patch, or the pacemaker lead (1.82 ± 0.88 mm vs. 3.33 ± 1.45 mm, $p < 0.0001$). Multiple vegetations in one valve (site) of the right heart were smaller than those of the single vegetations in one valve (site). Compared with what have been reported that the mean vegetation size was 1.3-1.5 cm in patients with left-sided infective endocarditis [194-196], and the mean diameter of the vegetations was 17 ± 6 mm of right-sided IE two decades ago [197], the present study revealed a larger vegetation size. Valvular insufficiency, embolic events and abscess formation were the most common complications of right-sided IE. The portal of entry and the single vegetation on single right-sided heart valve (site) were at increased risks for being complicated valve insufficiency, embolic events and abscess formation. Increased embolic events especially in the lungs were an outstanding epidemiological feature of right-sided IE, which might be associated with increased hematological changes such as leukocytosis and thrombophilia in the current setting.

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David *et al.* [198] reported that operative mortality for patients with active infective endocarditis was 12%, and late deaths were 23%. Nakagawa *et al.* [199] presented their comparative results of IE between 1990s and 2000s, and noted that the in-hospital mortality of IE was 5.4% in the 1990s and 13.3% in the 2000s. In the past, the mortality of right-sided IE was 17% [200]. Univariate analysis by other authors revealed that late mortality linked to preoperative renal insufficiency (serum creatinine >2 mg/dl), peripheral vascular disease, postoperative persistent bacteremia, and postoperative renal failure requiring dialysis. Cox regression analysis identified that postoperative renal failure was an independent predictor of late mortality [201]. Moreover, further studies showed vegetation size >2 cm and fungal etiology were associated with in-hospital mortality in right-sided cases by univariate analysis; while size of vegetation >2 cm achieved statistical significance for mortality in multivariate analysis [202]. The present study revealed an overall mortality of 10.2% and a surgical mortality of 7.8%, indicating an improved prognosis of right-sided IE of recent years. More severe valve dysfunctions might be resulted from larger vegetation sizes growing beyond valve leaflets. Reduced abscess formation, considerably decreased valve perforation and relatively better survival might be attributable to the current effective antibiotic regimens. Multiple logistic regression analysis revealed that the predisposing risk factors of IE, vegetation size and location of the vegetations were independent predictive risks relative to patients' survival.

In conclusion, epidemiologic changes have taken place in right-sided IE in the past 5½ years in younger patients, prevailed predisposing risk factor of portal of entry (in particular intravenous drug use), larger vegetations and more *Staphylococcus aureus* infections. Complication spectrum has changed into more valve insufficiency, more embolic events, reduced abscess formation, and considerably decreased valve perforation, indicating a more valve-functional than structural damage. With effective antibiotic regimens, prognoses of the patients seemed to be better than before.

Disclosure of conflict of interest

None.

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References

- [1] Fayad G, Leroy G, Devos P, Hervieux E, Senneville E, Koussa M and Leroy O. Characteristics and prognosis of patients requiring valve surgery during active infective endocarditis. *J Heart Valve Dis* 2011; 20: 223-228.
- [2] López J, Revilla A, Vilacosta I, Sevilla T, García H, Gómez I, Pozo E, Sarriá C and San Román JA. Multiple-valve infective endocarditis: clinical, microbiologic, echocardiographic, and prognostic profile. *Medicine (Baltimore)* 2011; 90: 231-236.
- [3] Chan P, Ogilby JD and Segal B. Tricuspid valve endocarditis. *Am Heart J* 1989; 117: 1140-1146.
- [4] Knirsch W and Nadal D. Infective endocarditis in congenital heart disease. *Eur J Pediatr* 2011; 170: 1111-1127.
- [5] Heydari AA, Safari H and Sarvghad MR. Isolated tricuspid valve endocarditis. *Int J Infect Dis* 2009; 13: e109-e111.
- [6] Ramadan FB, Beanlands DS and Burwash IG. Isolated pulmonic valve endocarditis in healthy hearts: a case report and review of the literature. *Can J Cardiol* 2000; 16: 1282-1288.
- [7] D'Agostino D, Bottalico L and Santacroce L. Infective endocarditis: what is changed in epidemiology and prophylaxis. *Acta Medica Mediter* 2012; 28: 311-319.
- [8] Naber CK. *Staphylococcus aureus* bacteremia: epidemiology, pathophysiology, and management strategies. *Clin Infect Dis* 2009; 48 Suppl 4: S231-S237.
- [9] Hill EE, Herijgers P, Claus P, Vanderschueren S, Herregods MC and Peetermans WE. Infective endocarditis: changing epidemiology and predictors of 6-month mortality: a prospective cohort study. *Eur Heart J* 2007; 28: 196-203.
- [10] Abandeh FI, Bazan JA, Davis JA, Zaidi AN, Daniels CJ and Firstenberg MS. *Bartonella henselae* prosthetic valve endocarditis in an adult patient with congenital heart disease: favorable outcome after combined medical and surgical management. *J Card Surg* 2012; 27: 449-452.
- [11] Akiyama T, Chikuda H, Yasunaga H, Horiguchi H, Fushimi K and Saita K. Incidence and risk factors for mortality of vertebral osteomyelitis: a retrospective analysis using the Japanese

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- diagnosis procedure combination database. *BMJ Open* 2013; 3. pii: e002412.
- [12] Akkinepally S, Douglass E and Moreno A. Tricuspid valve *gonococcal* endocarditis: fourth case report. *Int J Infect Dis* 2010; 14 Suppl 3: e196-e197.
- [13] Aliaga L, Santiago FM, Martí J, Sampedro A, Rodríguez-Granger J and Santalla JA. Right-sided endocarditis complicating an atrial septal defect. *Am J Med Sci* 2003; 325: 282-284.
- [14] Alozie A, Yerebakan C, Westphal B and Podbielski A. *Bartonella quintana* endocarditis of the tricuspid and mitral valves. *Thorac Cardiovasc Surg* 2012; 60: 363-365.
- [15] Anaya P, El-Chami MF, Kalogeropoulos AP, Martin RP and Lerakis S. Application of contrast echocardiography in the evaluation of a right-sided vegetative lesion. *Eur J Echocardiogr* 2007; 8: 501-503.
- [16] Aoki K, Kanazawa H, Okamoto T, Takahashi Y, Nakazawa S and Yamazaki Y. Awake partial sternotomy pacemaker implantation under thoracic epidural anesthesia. *Gen Thorac Cardiovasc Surg* 2009; 57: 418-420.
- [17] Aoki K, Watanabe M and Ohzeki H. Successful surgical treatment of tricuspid valve endocarditis associated with vertebral osteomyelitis. *Ann Thorac Cardiovasc Surg* 2010; 16: 207-209.
- [18] Araújo IR, Nunes Mdo C, Gelape CL, Carvalho VT, Lacerda BE, Oliveira GB, Brant LC and Ferrari TC. Challenge in the management of infective endocarditis with multiple valvular involvement. *Rev Soc Bras Med Trop* 2012; 45: 272-274.
- [19] Atamanyuk I, Raja SG and Kostolny M. *Bartonella henselae* endocarditis of percutaneously implanted pulmonary valve: a case report. *J Heart Valve Dis* 2011; 20: 94-97.
- [20] Aubert S, Levy Praschker B and Gandjbakhch I. Acute pulmonary valve endocarditis. *Arch Cardiovasc Dis* 2008; 101: 793-794.
- [21] Basak S, Solomonsz FA and Anumba DO. Infective endocarditis affecting the pulmonary valves in pregnant intravenous drug users. *J Obstet Gynaecol* 2011; 31: 78-80.
- [22] Bhat DP, Forbes TJ and Aggarwal S. A case of life-threatening *Staphylococcus aureus* endocarditis involving percutaneous transcatheter prosthetic pulmonary valve. *Congenit Heart Dis* 2013; 8: E161-E164.
- [23] Bilen E, Yasar AS, Bilge M, Kurt M, Karakas F and Aslantas U. Isolated pulmonic valve endocarditis in an adult patient with ventricular septal defect and infundibular pulmonary stenosis. *Echocardiography* 2008; 25: 904-907.
- [24] Boethig D, Westhoff-Bleck M, Hecker H, Ono M, Goerler A, Sarikouch S and Breymann T. Bovine jugular veins in the pulmonary position in adults – 5 years' experience with 64 implantations. *Thorac Cardiovasc Surg* 2009; 57: 196-201.
- [25] Bolton WD, Fox ER, Winscott JG and Aru GM. *Methicillin-resistant Staphylococcus aureus* pulmonary valve endocarditis. *Am Surg* 2009; 75: 1265-1266.
- [26] Brunetti ND, De Gennaro L, Basile DP, De Cillis E, Acquaviva T, Boscia F, Di Biase M and Bortone AS. A “strange cough”: 3D-echocardiography for diagnosis of late tricuspid valve endocarditis in a former drug addict with septic pulmonary emboli. *Int J Cardiol* 2011; 153: e15-e18.
- [27] Buckley DA. *Staphylococcus aureus* endocarditis as a complication of acupuncture for eczema. *Br J Dermatol* 2011; 164: 1405-1406.
- [28] Capoun R, Thomas M, Caputo M and Asimakopoulos G. Surgical treatment of tricuspid valve endocarditis: a single-centre experience. *Perfusion* 2010; 25: 169-173.
- [29] Carrillo-Córdova JR and Amezcua-Guerra LM. Autoimmunity as a possible predisposing factor for *Stenotrophomonas maltophilia* endocarditis. *Arch Cardiol Mex* 2012; 82: 204-207.
- [30] Castillo JG, Sanz J, Fischer GW, Bowman K and Filsoufi F. Management of anomalous left circumflex artery encircling the aortic annulus in a patient undergoing multivalvular surgery. *J Card Surg* 2009; 24: 667-669.
- [31] Chaurasia AS, Nawale JM and Yemul MA. Double-chambered right ventricle with pulmonary valve endocarditis. *Echocardiography* 2013; 30: E167-E170.
- [32] Cheung LL, Yue CS, Fung K, Chu CM and Keung Tso EY. Daptomycin as successful treatment for a refractory case of prosthetic valve endocarditis because of methicillin-sensitive *Staphylococcus aureus*. *Heart Lung* 2011; 40: 172-176.
- [33] Chung KP, Chang HT, Liao CH, Chu FY and Hsueh PR. *Staphylococcus lugdunensis* endocarditis with isolated tricuspid valve involvement. *J Microbiol Immunol Infect* 2012; 45: 248-250.
- [34] Cil BE, Canyigit M, Serter T, Peynircioglu B, Yorgancioglu C and Demircin M. Emergent transprosthetic valve coil embolization of ruptured mycotic pulmonary artery pseudoaneurysms secondary to right-sided endocarditis. *J Vasc Interv Radiol* 2008; 19: 783-784.
- [35] Dayan V, Gutierrez F, Cura L, Soca G and Lorenzo A. Two cases of pulmonary homograft replacement for isolated pulmonary valve endocarditis. *Ann Thorac Surg* 2009; 87: 1954-1956.
- [36] De Luca M, Amodio D, Chiurchiù S, Castelluzzo MA, Rinelli G, Bernaschi P, Calò Carducci FI and D'Argenio P. *Granulicatella* bacteraemia in

Right-sided infective endocarditis

- children: two cases and review of the literature. *BMC Pediatr* 2013; 13: 61.
- [37] Demarie D, De Vivo E, Cecchi E, Marletta G, Forsennati PG, Casabona R, Sansone F and Bignamini E. Acute endocarditis of the patch caused by *Staphylococcus capitis* in treated tetralogy of Fallot. An unusual location by an unusual bacterium. *Heart Lung Circ* 2012; 21: 189-192.
- [38] Deng H, Ma Y, Zhai H and Miao Q. Surgical valve repair of isolated pulmonary valve endocarditis. *Interact Cardiovasc Thorac Surg* 2013; 16: 384-386.
- [39] Derber C, Elam K, Forbes BA and Bearman G. *Achromobacter* species endocarditis: a case report and literature review. *Can J Infect Dis Med Microbiol* 2011; 22: e17-e20.
- [40] Dimassi A and Rushton T. Right-sided infective endocarditis due to methicillin-resistant *Staphylococcus aureus* in an injecting drug user: outbreak or slow epidemic? *W V Med J* 2009; 105: 18-19.
- [41] Dourakis S, Sambatakou H, Tsiachris D, Kittou N, Alexopoulou A and Archimandritis A. A 70-year-old stock-breeder with tricuspid valve and defibrillator lead *brucella* endocarditis. *Int J Cardiol* 2008; 126: e47-e49.
- [42] Dungu J, Juli C and Nihoyannopoulos P. Left coronary sinus to pulmonary artery fistula, complicated by pulmonary valve endocarditis. *Eur Heart J* 2011; 32: 1299.
- [43] Ehrenborg C, Hagberg S, Alden J, Makitalo S, Myrdal G, Larsson E, Hjelm E and Friman G. First known case of *Bartonella quintana* endocarditis in Sweden. *Scand J Infect Dis* 2009; 41: 73-75.
- [44] Ercan S, Sari E, Davutoglu V and Kis C. Post-partum tricuspid *staphylococcal* endocarditis complicated with multiple pulmonary pseudomonas abscesses. *BMJ Case Rep* 2013; 2013. pii: bcr2012008469.
- [45] Fordyce CB, Leather RA, Partlow E and Swiggum EA. Complete heart block associated with tricuspid valve endocarditis due to extended spectrum β -lactamase-producing *Escherichia coli*. *Can J Cardiol* 2011; 27: 263, e17-e20.
- [46] Fujino T, Amari Y, Mohri M, Noma M and Yamamoto H. MRSA tricuspid valve infective endocarditis with multiple embolic lung abscesses treated by combination therapy of vancomycin, rifampicin, and sulfamethoxazole/trime-thoprim. *J Cardiol* 2009; 53: 146-149.
- [47] Furui M, Ohashi T, Yoshida T, Oka F, Hirai Y, Ohyoshi N and Kojima A. Ventricular septal perforation caused by right-sided infective endocarditis associated with giant vegetation. *Ann Thorac Surg* 2010; 89: 959-961.
- [48] Gabus V, Grenak-Degoumois Z, Jeanneret S, Rakotoarimanana R, Greub G and Genné D. *Tropheryma whippelii* tricuspid endocarditis: a case report and review of the literature. *J Med Case Rep* 2010; 4: 245.
- [49] Garbati MA, Tleyjeh IM and Abba AA. Complicated community-acquired *Staphylococcus* endocarditis and multiple lung abscesses: case report and review of literature. *Case Rep Infect Dis* 2011; 2011: 981316.
- [50] Gokaslan G, Deniz H, Ozcaliskan O, Yasim A and Ustunsoy H. Pericardial monocusp reconstruction for pulmonary valve vegetation secondary to patent ductus arteriosus. *J Card Surg* 2011; 26: 650-652.
- [51] Gonçalves AM, Correia A and Falcão LM. Tricuspid valve endocarditis in a patient with congenital heart disease. *Rev Port Cardiol* 2013; 32: 53-58.
- [52] Gullu AU, Akcar M, Arnaz A and Kizilay M. *Candida parapsilosis* tricuspid native valve endocarditis: 3-year follow-up after surgical treatment. *Interact Cardiovasc Thorac Surg* 2008; 7: 513-514.
- [53] Gupta K, Das A, Joshi K, Singh N, Aggarwal R and Prakash M. *Aspergillus* endocarditis in a known case of allergic bronchopulmonary aspergillosis: an autopsy report. *Cardiovasc Pathol* 2010; 19: e137-e139.
- [54] Gupta S, Mittal A and Gupta S. Tricuspid endocarditis in hyper-IgE syndrome. *J Postgrad Med* 2010; 56: 143-145.
- [55] Hatemi AC, Gursoy M, Tongut A, Bicakhan B, Guzeltas A, Cetin G and Kansiz E. Pulmonary stenosis as a predisposing factor for infective endocarditis in a patient with Noonan syndrome. *Tex Heart Inst J* 2010; 37: 99-101.
- [56] Hayani O, Higginson LA, Toye B and Burwash IG. Man's best friend? Infective endocarditis due to *Capnocytophaga canimorsus*. *Can J Cardiol* 2009; 25: e130-e132.
- [57] Higgins J, Shayan H and Fradet G. Right-sided endocarditis secondary to a peritoneovenous shunt. *Can J Cardiol* 2010; 26: e280-e281.
- [58] Hottkowitz C, Ammann P, Kleger GR, Künzli A, Hack D, Rickli H and Maeder MT. Successful management of a case of electrical storm due to invasive endocarditis. *Can J Cardiol* 2012; 28: 245: e13-e15.
- [59] Ikeuchi M, Hisano K, Monden Y and Urabe Y. Isolated pulmonary valve endocarditis detected on multislice CT. *Intern Med* 2013; 52: 835.
- [60] Irving CA, Kelly D, Gould FK and O'Sullivan JJ. Successful medical treatment of bioprosthetic pulmonary valve endocarditis caused by methicillin-resistant *Staphylococcus aureus*. *Pediatr Cardiol* 2010; 31: 553-555.
- [61] Jeppson PC, Park A and Chen CC. Multivalvular bacterial endocarditis after suction curettage abortion. *Obstet Gynecol* 2008; 112: 452-455.
- [62] Johri AM, Kovacs KA and Kafka H. An unusual case of infective endocarditis: extension of a

Right-sided infective endocarditis

- tricuspid valve vegetation into the left atrium through a patent foramen ovale. *Can J Cardiol* 2009; 25: 429-431.
- [63] Joskowiak D, Kappert U, Matschke K and Tugtekin SM. Ruptured pulmonary artery caused by isolated pulmonary valve endocarditis: case report. *Clin Res Cardiol* 2010; 99: 471-473.
- [64] Jung TE, Kim JH, Do HD and Lee DH. Simultaneous aortic and tricuspid valve endocarditis due to complication of sinus of valsalva rupture. *Korean J Thorac Cardiovasc Surg* 2011; 44: 240-242.
- [65] Karabulut A, Surgit O, Akgul O and Bakir I. "Removal without replacement" strategy for uncontrolled prosthetic tricuspid valve endocarditis associated with abortion sepsis. *Heart Surg Forum* 2011; 14: E357-E359.
- [66] Karabinos IK, Kokladi M and Katritsis D. Fungal endocarditis of the superior vena cava: the role of transesophageal echocardiography. *Hellenic J Cardiol* 2010; 51: 538-539.
- [67] Karaci AR, Aydemir NA, Harmandar B, Sasmazel A, Saritas T, Tuncel Z and Yekeler I. Surgical treatment of infective valve endocarditis in children with congenital heart disease. *J Card Surg* 2012; 27: 93-98.
- [68] Kassai I, Friedrich O, Ratnatunga C, Betts TR, Mihálcz A and Szili-Török T. Feasibility of percutaneous implantation of transapical endocardial left ventricular pacing electrode for cardiac resynchronization therapy. *Europace* 2011; 13: 1653-1657.
- [69] Kaul P, Adluri K, Javangula K and Baig W. Successful management of multiple permanent pacemaker complications—infection, 13 year old silent lead perforation and exteriorisation following failed percutaneous extraction, superior vena cava obstruction, tricuspid valve endocarditis, pulmonary embolism and prosthetic tricuspid valve thrombosis. *J Cardiothorac Surg* 2009; 4: 12.
- [70] Kervan Ü, Altintas G, Ozen A, Durukan M, Guray Ü and Özatik MA. Implantable cardioverter defibrillator lead endocarditis causing diffuse right atrial abscess and pulmonary artery embolism. *Pacing Clin Electrophysiol* 2011; 34: e115-e117.
- [71] Kim JH, Kim YS, Yoon YH, Kim JT, Kim KH and Baek WK. Quadrangular resection of the tricuspid valve. *Korean J Thorac Cardiovasc Surg* 2013; 46: 60-62.
- [72] Kim WS, Kang SH, Lee SA, Ryu MS and Park SH. A case of *staphylococcal* tricuspid valve endocarditis with para-aortic abscess in a patient with bicuspid aortic valve. *Korean Circ J* 2011; 41: 482-485.
- [73] Konstantinov IE. Total resection and complete reconstruction of the tricuspid valve in acute infective endocarditis. *J Thorac Cardiovasc Surg* 2008; 136: 531-532.
- [74] Koruk ST, Erdem H, Koruk I, Erbay A, Tezer-Tekce Y, Erbay AR, Dayan S, Deveci O, Inan A, Engin DO, Guner R, Dikici N, Doyuk-Kartal E, Kurtaran B, Pehlivanoglu F, Sipahi OR, Yalci A, Yemisen M, Alp-Cavus S, Gencer S, Guzel G, Oncul O, Parlak M, Kazak E, Tulek N, Ulcay A and Savasci U. Management of *Brucella* endocarditis: results of the Gulhane study. *Int J Antimicrob Agents* 2012; 40: 145-150.
- [75] Kothari A, Pillai BS and Bhan A. Pacing lead endocarditis due to *Aspergillus fumigatus*. *Indian J Med Microbiol* 2010; 28: 72-73.
- [76] Kumar P, Muranjan MN, Tullu MS, Vaideeswar P, Kher A and Lahiri KR. *Candida tropicalis* endocarditis: treatment in a resource-poor setting. *Ann Pediatr Cardiol* 2010; 3: 174-177.
- [77] Lacalzada J, Enjuanes C, Izquierdo MM, Baragán Acea A, De La Rosa A and Laynez I. Pulmonary valve infective endocarditis in an adult patient with severe congenital pulmonary stenosis and ostium secundum atrial septal defect. *Cardiol Res Pract* 2010; 2010: 798956.
- [78] Lax D, Bhatt RD, Klewer SE and Sorrell VL. Are all ventricular septal defects created equal? *J Am Soc Echocardiogr* 2010; 23: 791, e5-e7.
- [79] Le Gloan L, Leduc L, O'Meara E, Khairy P and Dore A. Right ventricular endocarditis in a pregnant woman with a restrictive ventricular septal defect. *Congenit Heart Dis* 2011; 6: 638-640.
- [80] Lee LC, Wong R, Raju GC, Khor C and Yip J. Protein-losing enteropathy post-valvular surgery with severe tricuspid regurgitation in Subutex-related endocarditis. *Singapore Med J* 2009; 50: e124-e126.
- [81] Lin T, Santos M, Aboltins C, Chiu H, Van Gaal W and Wong C. A case of intra-cardiac right-sided mural infective endocarditis associated with ventricular septal defect despite prophylactic antibiotics: a case report. *Heart Lung Circ* 2010; 19: 566-571.
- [82] Liu CY, Wang JL, Huang YT and Hsueh PR. Development of multiple lung abscesses during daptomycin treatment for right-sided endocarditis caused by methicillin-resistant *Staphylococcus aureus*. *Int J Antimicrob Agents* 2008; 32: 544-545.
- [83] Lodha A, Enakpene E, Haran M and Sadiq A. Unusual endocarditis: "rare bug, rare site". *Am J Med Sci* 2012; 343: 328-329.
- [84] Logue MA, Perk G and Kronzon I. Tricuspid prosthesis stenosis associated with endocarditis: a new M-mode finding. *Echocardiography* 2008; 25: 511-513.
- [85] Luther V, Townell J, Rahman H and Loong CY. Fever and haemoptysis in an injecting drug user. *BMJ* 2012; 344: e1568.

Right-sided infective endocarditis

- [86] Maekawa Y, Sakamoto T, Umezu K, Ohashi N and Harada Y. Infective endocarditis in a child caused by *Cardiobacterium hominis* after right ventricular outflow tract reconstruction using an expanded tetrafluoroethylene conduit. *Gen Thorac Cardiovasc Surg* 2011; 59: 429-432.
- [87] Matt P, Winkler B, Carrel T and Eckstein F. Plicated patch repair for acquired Gerbode defect involving the tricuspid valve. *Ann Thorac Surg* 2010; 89: 643-645.
- [88] McCaughan JA, Purvis JA and Sharkey RA. Embolisation of vegetation to the liver in right sided infective endocarditis. *Eur J Intern Med* 2009; 20: e32-e33.
- [89] McKenna T and O'Brien K. Case report: group B streptococcal bacteremia and sacroiliitis after mid-trimester dilation and evacuation. *J Perinatol* 2009; 29: 643-645.
- [90] Melina G, El-Hamamsy I, Sinatra R and Yacoub MH. Late fulminant pulmonary valve endocarditis after the Ross operation. *J Thorac Cardiovasc Surg* 2010; 139: e99-e100.
- [91] Misra S, Koshy T, Sinha PK, Misra M and Bijulal S. An additional mass in the aortic root in a patient with infective endocarditis scheduled for excision of a tricuspid valve mass? *J Cardiothorac Vasc Anesth* 2008; 22: 495-496.
- [92] Mitchell ME, McManus M, Dietz J, Camitta BM, Szabo S and Havens P. *Absidia corymbifera* endocarditis: survival after treatment of disseminated mucormycosis with radical resection of tricuspid valve and right ventricular free wall. *J Thorac Cardiovasc Surg* 2010; 139: e71-e72.
- [93] Moreira D, Correia E, Rodrigues B, Santos L, Capelo J, Abreu L, Nunes L and Oliveira-Santos J. Isolated pulmonary valve endocarditis in a normal heart. *Rev Port Cardiol* 2012; 31: 615-617.
- [94] Morin JF, Sheppard R and Chamoun P. Unusual case of colonized pacemaker lead presenting with endocarditis, hemoptysis and tricuspid valve stenosis. *Can Respir J* 2011; 18: e48-e49.
- [95] Morokuma H, Minato N, Kamohara K and Minematsu N. Three surgical cases of isolated tricuspid valve infective endocarditis. *Ann Thorac Cardiovasc Surg* 2010; 16: 134-138.
- [96] Mousavi N, Bhagirath K, Ariyaratna V, Fang T, Ahmadie R, Lytwyn M, Jassal DS and Seifer C. Chiari network endocarditis: not just an innocent bystander. *Echocardiography* 2008; 25: 642-645.
- [97] Moysakakis I, Kazazis A, Lionakis N, Vlahodimitris I, Petrikos G and Votteas V. Late recurrence of pulmonic valve endocarditis in an adult patient with ventricular septal defect: a case report. *J Heart Valve Dis* 2009; 18: 167-169.
- [98] Murillo J, McMahon C and Starr J. Large vegetations in *Staphylococcus lugdunensis* endocarditis. *Heart Lung* 2008; 37: 479-480.
- [99] Mutlu H, Babar J and Maggiore PR. Extensive *Salmonella* enteritidis endocarditis involving mitral, tricuspid valves, aortic root and right ventricular wall. *J Am Soc Echocardiogr* 2009; 22: 210, e1-e3.
- [100] Naba MR, Araj GF, Kanafani ZA and Kanj SS. First case of *Pasteurella multocida* endocarditis of the tricuspid valve: a favorable outcome following medical treatment. *Int J Infect Dis* 2009; 13: e267-e269.
- [101] Naqvi TZ, Rafie R and Ghalichi M. Real-time 3D TEE for the diagnosis of right-sided endocarditis in patients with prosthetic devices. *JACC Cardiovasc Imaging* 2010; 3: 325-327.
- [102] Natsag J, Min Z, Hamad Y, Alkhalil B, Rahman A and Williams R. A mysterious gram-positive rods. *Case Rep Infect Dis* 2012; 2012: 841834.
- [103] Negi SI and Anand A. Widespread systemic embolization with isolated tricuspid valve endocarditis. *Heart Lung* 2012; 41: 387-389.
- [104] Nishanth KR, Seshadri S, Pandit V and Krishnanand N. Isolated pulmonary valve endocarditis in a patient with aplastic anaemia. *BMJ Case Rep* 2013; 2013. pii: bcr2013008769.
- [105] Nishida K, Fukuyama O and Nakamura DS. Pulmonary valve endocarditis caused by right ventricular outflow obstruction in association with sinus of valsalva aneurysm: a case report. *J Cardiothorac Surg* 2008; 3: 46.
- [106] Nordbeck P, Bauer WR and Ritter O. Ablation of atrial flutter in a patient with a tricuspid valve replacement after endocarditis. *Pacing Clin Electrophysiol* 2009; 32: 1237-1239.
- [107] Nunes Mdo C, Barbosa FB, Gelape CL, Leduc LR, Castro LR, Gresta LT and Ferrari TC. Piercing-related endocarditis presenting with multiple large masses in the right-side chamber of the heart. *J Am Soc Echocardiogr* 2008; 21: 776, e1-e3.
- [108] Nwaohiri N, Urban C, Gluck J, Ahluwalia M and Wehbeh W. Tricuspid valve endocarditis caused by *Haemophilus parainfluenzae*: a case report and review of the literature. *Diagn Microbiol Infect Dis* 2009; 64: 216-219.
- [109] Odieta O, Akinwande O, Murray JJ and Akamah J. *Pneumococcal* tricuspid valve endocarditis in a young African American: a case for inclusion of African Americans in pneumococcal vaccine criteria. *Case Rep Med* 2010; 2010. pii: 982521.
- [110] Ou TY, Chen RF, Hsu CS, Kao PF, Yu FL, Teng SO and Lee WS. Pulmonary valve endocarditis in a pregnant woman with a ventricular septal defect. *J Microbiol Immunol Infect* 2009; 42: 92-95.
- [111] Pagni S, Dempsey A and Austin EH 3rd. Tricuspid and aortic valve and ventricular septal defect endocarditis: an unusual presentation of

Right-sided infective endocarditis

- acute Q fever. *Ann Thorac Surg* 2009; 88: 2027-2029.
- [112] Palma G, Giordano R, Russolillo V and Vosa C. Percutaneous pulmonary valve implantation after endocarditis of Contegra® valved conduit: a case report. *Thorac Cardiovasc Surg* 2011; 59: 123-125.
- [113] Panduranga P, Al-Mukhaini M, Sulaiman K and Al-Abri S. Tricuspid valve endocarditis in an intravenous drug abuser masquerading as pulmonary tuberculosis. *Heart Views* 2010; 11: 121-124.
- [114] Papanikolaou J, Karakitsos D, Yang C, Saranteas T and Karabinis A. Transesophageal echocardiography in detecting tricuspid valve pathology in an intensive care unit patient. *J Cardiothorac Vasc Anesth* 2010; 24: 211-213.
- [115] Parish LM, Liu L and Woo YJ. Endocarditis with massive aortic root abscess and atrioventricular septal destruction. *Interact Cardiovasc Thorac Surg* 2009; 8: 280-282.
- [116] Park HE, Cho GY, Kim HK, Kim YJ and Sohn DW. Pulmonary valve endocarditis with septic pulmonary thromboembolism in a patient with ventricular septal defect. *J Cardiovasc Ultrasound* 2009; 17: 138-140.
- [117] Partridge DG, O'Brien E and Chapman AL. Outpatient parenteral antibiotic therapy for infective endocarditis: a review of 4 years' experience at a UK centre. *Postgrad Med J* 2012; 88: 377-381.
- [118] Patil R, Patil T and Hussain KM. *Staphylococcus lugdunensis* native tricuspid valve endocarditis: a case report and review of literature. *J Gen Intern Med* 2011; 26: 1209-1211.
- [119] Peng H, Chen WF, Wu C, Chen YR, Peng B, Paudel SD and Lou TQ. Culture-negative subacute bacterial endocarditis masquerades as granulomatosis with polyangiitis (Wegener's granulomatosis) involving both the kidney and lung. *BMC Nephrol* 2012; 13: 174.
- [120] Pérez Baztarrica G, Gariglio L, Salvaggio F, Reolón E, Blanco N, Mazzetti H, Villecco S, Botbol A and Porcile R. Transvenous extraction of pacemaker leads in infective endocarditis with vegetations ≥ 20 mm: our experience. *Clin Cardiol* 2012; 35: 244-249.
- [121] Pfannmüller B, Moz M, Misfeld M, Borger MA, Funkat AK, Garbade J and Mohr FW. Isolated tricuspid valve surgery in patients with previous cardiac surgery. *J Thorac Cardiovasc Surg* 2013; 146: 841-847.
- [122] Pillai V, Menon S, Kottayil B and Karunakaran J. Tricuspid endocarditis with indirect Gerbode: septal translocation of posterior leaflet. *Heart Lung Circ* 2011; 20: 362-364.
- [123] Poh KK, Chong E and Yeo TC. Disappearing left atrial mass with drainage of pericardial effusion. *Eur Heart J* 2009; 30: 97.
- [124] Pottecher J, Ludes PO, Kuhnert C, Petit-Eisenmann H and Mommerot A. Focal neurological deficit with tricuspid endocarditis and patent foramen ovale. *Intensive Care Med* 2012; 38: 2081-2082.
- [125] Pretorius V, Jones A, Taylor D, Coe Y and Ross DB. Percutaneous valved stent repair of a failed homograft: implications for the Ross procedure. *Can J Cardiol* 2008; 24: e54-e55.
- [126] Quiñones JN, Campbell F, Coassolo KM, Pylewski G and Maran P. Tricuspid valve endocarditis during the second trimester of pregnancy. *Obstet Med* 2010; 3: 78-80.
- [127] Rainer PP, Schmidt A, Anelli-Monti M, Kleinert R, Pieske BM and Maier RM. A swinging pacemaker lead promoting endocarditis and severe tricuspid regurgitation. *J Am Coll Cardiol* 2012; 59: e45.
- [128] Regules JA, Glasser JS, Wolf SE, Hospenthal DR and Murray CK. Endocarditis in burn patients: clinical and diagnostic considerations. *Burns* 2008; 34: 610-616.
- [129] Reyes MP, Ali A, Mendes RE and Biedenbach DJ. Resurgence of *Pseudomonas* endocarditis in Detroit, 2006-2008. *Medicine (Baltimore)* 2009; 88: 294-301.
- [130] Rohn V, Slais M, Kotulák T and Psenicka M. Pulmonary valve replacement for pacing electrodes related bacterial endocarditis. *Ann Thorac Surg* 2008; 85: 2128-2130.
- [131] Rostagno C, Carone E, Rossi A, Gensini GF and Stefano PL. Surgical treatment in active infective endocarditis: results of a four-year experience. *ISRN Cardiol* 2011; 2011: 492543.
- [132] Sahin T, Bildirici U, Kandemir C, Celikyurt U, Ural D and Komsuoglu B. Infective endocarditis in the setting of infundibular-valvular pulmonary stenosis with incomplete cor triatriatum dextrum and patent foramen ovale. *Int J Cardiol* 2008; 127: e129-e131.
- [133] Sai S, Adachi O, Saiki Y, Konishi A and Tabayashi K. Relapsing infective endocarditis following closure of ventricular septal defect. *Asian Cardiovasc Thorac Ann* 2012; 20: 58-60.
- [134] Salhiyyah K, Senanayake E and Cooper GJ. Successful surgical repair of tricuspid valve endocarditis. *J Card Surg* 2010; 25: 153-155.
- [135] Salmi D, Bhat A, Corman L, Raff G and Satake N. Diagnostic challenges in native valve fungal endocarditis producing a massive septic pulmonary embolus. *Nihon Ishinkin Gakkai Zasshi* 2010; 51: 207-210.
- [136] Sani AT, Mojtabavi M and Bolandnazar R. Effect of vegetation size on the outcome of infective endocarditis in intravenous drug users. *Iran J Clin Infect Dis* 2009; 4: 129-134.
- [137] Saravu K, Mukhopadhyay C, Satyanarayanan V, Pai A, Komaranchath AS, Munim F, Shastry BA and Tom D. Successful treatment of right-

Right-sided infective endocarditis

- sided native valve methicillin-resistant *Staphylococcus aureus* endocarditis and septicaemia with teicoplanin and rifampicin: a case report. *Scand J Infect Dis* 2012; 44: 544-547.
- [138] Sarkar A, Ahmed I, Chandra N and Pande A. Pulmonary endarteritis, cerebral abscesses, and a single ventricle: An uncommon combination. *J Cardiovasc Dis Res* 2012; 3: 236-239.
- [139] Schnell D, Beyler C, Lanternier F, Lucron H, Lebeaux D, Bille E, Bonnet D, Lortholary O, Nassif X, Zahar JR and Bajorolle F. Nontoxigenic corynebacterium diphtheriae as a rare cause of native endocarditis in childhood. *Pediatr Infect Dis J* 2010; 29: 886-888.
- [140] Schnoering H, Sachweh JS, Muehler EG and Vazquez-Jimenez JF. Pancarditis in a five-year-old boy affecting tricuspid valve and ventricular septum. *Eur J Cardiothorac Surg* 2008; 34: 1115-1117.
- [141] Seeburger J, Groesdonk H, Borger MA, Merk D, Ender J, Falk V, Mohr FW and Doll N. Quadruple valve replacement for acute endocarditis. *J Thorac Cardiovasc Surg* 2009; 137: 1564-1565.
- [142] Seif D, Meeks A, Mailhot T and Perera P. Emergency department diagnosis of infective endocarditis using bedside emergency ultrasound. *Crit Ultrasound J* 2013; 5: 1.
- [143] Shaikh Q and Mahmood F. Triple valve endocarditis by mycobacterium tuberculosis: a case report. *BMC Infect Dis* 2012; 12: 231.
- [144] Shetty RK, Vivek G, Naha K and Bekkam S. Right-sided infective endocarditis presenting with purpuric skin rash and cardiac failure in a patient without fever. *BMJ Case Rep* 2013; 2013. pii: bcr2012007841.
- [145] Shiokawa Y, Nakashima A, Tanoue Y and Tomi-naga R. Successful surgical treatment for methicillin-resistant *Staphylococcus aureus* endocarditis on the ventricular rerouting patch after a Rastelli operation. *Gen Thorac Cardiovasc Surg* 2011; 59: 483-484.
- [146] Shrestha BM, Fukushima S, Vrtik M, Chong IH, Sparks L, Jalali H and Pohlner PG. Partial replacement of tricuspid valve using cryopreserved homograft. *Ann Thorac Surg* 2010; 89: 1187-1194.
- [147] Sibal AK, Lin Z and Jogia D. Coagulase-negative *Staphylococcus* endocarditis: *Staphylococcus lugdunensis*. *Asian Cardiovasc Thorac Ann* 2011; 19: 414-415.
- [148] Singh NK, Godara M and Agrawal V. Rupture of non-coronary sinus of valsalva with infective endocarditis of naive tricuspid valve presenting with recurrent ill-sustained ventricular tachycardia. *J Assoc Physicians India* 2011; 59: 184-186.
- [149] Sohail MR, Gray AL, Baddour LM, Tleyjeh IM and Virk A. Infective endocarditis due to *Propionibacterium* species. *Clin Microbiol Infect* 2009; 15: 387-394.
- [150] Sözüner ZC, Kayal A, Atasoy C, Kilickap M, Numanoglu N and Savas I. Septic pulmonary embolism: three case reports. *Monaldi Arch Chest Dis* 2008; 69: 75-77.
- [151] Spoladore R, Agricola E, D'Aamato R, Durante A, Fragasso G and Margonato A. Isolated native tricuspid valve endocarditis due to group A β -hemolytic *Streptococcus* without drug addiction. *J Cardiovasc Med (Hagerstown)* 2010 Jul 27; [Epub ahead of print].
- [152] Sreeram N, Ben Mime L and Bennink G. Pericardial patch valve in the tricuspid position in an infant. *Cardiol Young* 2011; 21: 458-459.
- [153] Stöllberger C, Thalmann M and Finsterer J. Myasthenia, spondylitis and *Enterococcus faecalis* endocarditis. *Arch Immunol Ther Exp (Warsz)* 2012; 60: 221-223.
- [154] Stroup JS, Wagner J and Badzinski T. Use of daptomycin in a pregnant patient with *Staphylococcus aureus* endocarditis. *Ann Pharmacother* 2010; 44: 746-749.
- [155] Sundaram PS, Bijulal S, Tharakan JA and Antony M. *Kodamaea ohmeri* tricuspid valve endocarditis with right ventricular inflow obstruction in a neonate with structurally normal heart. *Ann Pediatr Cardiol* 2011; 4: 77-80.
- [156] Tembe AG, Kharbanda P, Dalal JJ, Vaishnav G and Joshi VR. Infective endocarditis—a tale of two cases and the lessons (re)learned. *J Assoc Physicians India* 2010; 58: 319-322.
- [157] Thakar S, Janga KC, Tolchinsky T, Greenberg S, Sharma K, Sadiq A, Lichstein E and Shani J. Superior vena cava and right atrium wall infective endocarditis in patients receiving hemodialysis. *Heart Lung* 2012; 41: 301-307.
- [158] Tirmizi A, Butt S and Molitoris S. First reported case of *Pasteurella pneumotropica* tricuspid valve endocarditis. *Int J Cardiol* 2012; 161: e44-e45.
- [159] Tsutsumi T, Hiraoka E, Kanazawa K, Akita H and Eron LJ. Diagnosis of *E. coli* tricuspid valve endocarditis: a case report. *Hawaii Med J* 2010; 69: 286-288.
- [160] Vaideeswar P, Jawale RM and Tullu M. Isolated infective endocarditis of the pulmonary valve: an autopsy analysis of nine cases. *Cardiovasc Pathol* 2009; 18: 231-235.
- [161] Vellinga S, Timmermans P and Wollaert B. A young man with chest pain and fever. *Acta Cardiol* 2010; 65: 707-708.
- [162] Vernadakis S, Saner FH, Rath PM, Kaiser GM, Mathe Z, Treckmann J and Paul A. Successful salvage therapy with daptomycin after linezolid and vancomycin failure in a liver transplant recipient with methicillin-resistant *Staphylococcus aureus* endocarditis. *Transpl Infect Dis* 2009; 11: 346-348.

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- [163] Vincent P, Davis R and Roy D. Group B *streptococcus tricuspid* endocarditis presenting with arthralgia in a postpartum woman: a case report. *J Med Case Rep* 2012; 6: 242.
- [164] Vohra S, Taylor R and Aronowitz P. The tell-tale heart: *Aspergillus fumigatus* endocarditis in an immunocompetent patient. *Hosp Pract* (1995) 2013; 41: 117-121.
- [165] Vu M, Harrison BA, DeStephano C and Odell J. Endocarditis, vegetation, and perforation of the pulmonary valve. *J Cardiothorac Vasc Anesth* 2008; 22: 261-262.
- [166] Waldman JD, Berman W Jr and McCullough G. Endocarditis after balloon dilation of congenital pulmonary valve stenosis. *Pediatr Cardiol* 2012; 33: 1461-1462.
- [167] Wei HH, Wu KG, Sy LB, Chen CJ and Tang RB. Infectious endocarditis in pediatric patients: analysis of 19 cases presenting at a medical center. *J Microbiol Immunol Infect* 2010; 43: 430-437.
- [168] Weymann A, Schmack B, Rosendal C, Rauch H, Karck M, Tochtermann U and Szabó G. Tricuspid valve endocarditis with septic pulmonary emboli in a drug addict. *Ann Thorac Cardiovasc Surg* 2012; 18: 481-484.
- [169] Wilder T, Shah S, Mello D and Setty SP. Double-chambered right ventricle with cough, endocarditis, and lung mass. *Ann Thorac Surg* 2010; 89: 1299-300.
- [170] Williamson DA and McBride SJ. A case of tricuspid valve endocarditis due to *Burkholderia cepacia* complex. *N Z Med J* 2011; 124: 84-86.
- [171] Wu W, He P, Xiong G, Wang HD, Liao KL, Zhang W and Yang K. Bioprosthetic tricuspid valve implantation for active tricuspid valve endocarditis in an adult burn patient. *J Heart Valve Dis* 2010; 19: 674-675.
- [172] Xhabija N, Prifti E, Allajbeu I and Sula F. Gerbode defect following endocarditis and misinterpreted as severe pulmonary arterial hypertension. *Cardiovasc Ultrasound* 2010; 8: 44.
- [173] Yamamoto Y, Shiohita K, Takazono T, Seki M, Izumikawa K, Kakeya H, Yanagihara K, Tashiro T, Otsuka Y, Ohkusu K and Kohno S. *An autopsy case of Erysipelothrix rhusiopathiae* endocarditis. *Intern Med* 2008; 47: 1437-1440.
- [174] Yamamoto K, Ito H and Hiraiwa T. Perforation of a tricuspid pouch caused by infective endocarditis. *Ann Thorac Surg* 2008; 86: 1670-1672.
- [175] Yang EH, Poon K, Pillutla P, Budoff MJ and Chung J. Pulmonary embolus caused by *Suttonella indologenes* prosthetic endocarditis in a pulmonary homograft. *J Am Soc Echocardiogr* 2011; 24: 592, e1-e3.
- [176] Yasar KK, Pehlivanoglu F, Gursoy S and Sengoz G. Tricuspid endocarditis and septic pulmonary embolism in an intravenous drug user with advanced HIV infection. *Oman Med J* 2011; 26: 365-367.
- [177] Murdoch DR, Corey GR, Hoen B, Miró JM, Fowler VG Jr, Bayer AS, Karchmer AW, Olaison L, Pappas PA, Moreillon P, Chambers ST, Chu VH, Falcó V, Holland DJ, Jones P, Klein JL, Raymond NJ, Read KM, Tripodi MF, Utili R, Wang A, Woods CW, Cabell CH and International Collaboration on Endocarditis-Prospective Cohort Study (ICE-PCS) Investigators. Clinical presentation, etiology, and outcome of infective endocarditis in the 21st century: the International Collaboration on Endocarditis-Prospective Cohort Study. *Arch Intern Med* 2009; 169: 463-473.
- [178] Nandakumar R and Raju G. Isolated tricuspid valve endocarditis in non addicted patients: a diagnostic challenge. *Am J Med Sci* 1997; 314: 207-212.
- [179] de Alarcón A and Villanueva JL. Endocarditis in parenteral drug addicts. Right-sided endocarditis. Influence of HIV infection. *Rev Esp Cardiol* 1998; 51 Suppl 2: 71-78.
- [180] Pektok E, Sierra J, Cikirikcioglu M, Müller H, Myers PO and Kalangos A. Midterm results of valve repair with a biodegradable annuloplasty ring for acute endocarditis. *Ann Thorac Surg* 2010; 89: 1180-1185.
- [181] Saxena A, Aggarwal N, Gupta P, Juneja R, Kothari SS and Math R. Predictors of embolic events in pediatric infective endocarditis. *Indian Heart J* 2011; 63: 237-240.
- [182] Jain V, Yang MH, Kovacicova-Lezcano G, Juhle LS, Bolger AF and Winston LG. Infective endocarditis in an urban medical center: association of individual drugs with valvular involvement. *J Infect* 2008; 57: 132-138.
- [183] De Rosa FG, Cicalini S, Canta F, Audagnotto S, Cecchi E and Di Perri G. Infective endocarditis in intravenous drug users from Italy: the increasing importance in HIV-infected patients. *Infection* 2007; 35: 154-160.
- [184] del Río A, Anguera I, Miró JM, Mont L, Fowler VG Jr, Azqueta M, Mestres CA and Hospital Clínic Endocarditis Study Group. Surgical treatment of pacemaker and defibrillator lead endocarditis: the impact of electrode lead extraction on outcome. *Chest* 2003; 124: 1451-1459.
- [185] Arbulu A. Trivalvular/bivalvular heart: a philosophical, scientific and therapeutic concept. *J Heart Valve Dis* 2000; 9: 353-357; discussion 357-358.
- [186] Nadji G, Réyadi JP, Coviaux F, Mirode AA, Brahim A, Enriquez-Sarano M and Tribouilloy C. Comparison of clinical and morphological characteristics of *Staphylococcus aureus* endocarditis with endocarditis caused by other pathogens. *Heart* 2005; 91: 932-937.
- [187] Chrissoheris MP, Libertin C, Ali RG, Ghantous A, Bekui A and Donohue T. Endocarditis com-

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- plicating central venous catheter bloodstream infections: a unique form of health care associated endocarditis. *Clin Cardiol* 2009; 32: E48-E54.
- [188] Naidoo DP. Right-sided endocarditis in the non-drug addict. *Postgrad Med J* 1993; 69: 615-620.
- [189] Forrest GN, Arnold RS, Gammie JS and Gilliam BL. Single center experience of a vancomycin resistant *enterococcal* endocarditis cohort. *J Infect* 2011; 63: 420-428.
- [190] Levine DP and Lamp KC. Daptomycin in the treatment of patients with infective endocarditis: experience from a registry. *Am J Med* 2007; 120 Suppl 1: S28-S33.
- [191] scini C, Bongiorno MG, Doria R, Polidori M, Iapoce R, Fondelli S, Tagliaferri E, Soldati E, Di Paolo A, Leonildi A and Menichetti F. Linezolid for endocarditis: a case series of 14 patients. *J Antimicrob Chemother* 2011; 66: 679-682.
- [192] Musci M, Siniawski H, Pasic M, Grauhan O, Weng Y, Meyer R, Yankah CA and Hetzer R. Surgical treatment of right-sided active infective endocarditis with or without involvement of the left heart: 20-year single center experience. *Eur J Cardiothorac Surg* 2007; 32: 118-125.
- [193] Chambers HF, Miller RT and Newman MD. Right-sided *Staphylococcus aureus* endocarditis in intravenous drug abusers: two-week combination therapy. *Ann Intern Med* 1988; 109: 619-624.
- [194] Leitman M, Dreznik Y, Tyomkin V, Fuchs T, Krakover R and Vered Z. Vegetation size in patients with infective endocarditis. *Eur Heart J Cardiovasc Imaging* 2012; 13: 330-338.
- [195] Gotsman I, Meirovitz A, Meizlish N, Gotsman M, Lotan C and Gilon D. Clinical and echocardiographic predictors of morbidity and mortality in infective endocarditis: the significance of vegetation size. *Isr Med Assoc J* 2007; 9: 365-369.
- [196] Bonetti NR, Namdar M, Güenthard HF, Gruner C, Greutmann M, Steffel J, Hürlimann D, Ruef C, Tanner FC, Jenni R and Biaggi P. Early versus late initial echocardiographic assessment in infective endocarditis: similar findings and no difference in clinical outcome. *Cardiovasc Med* 2012; 15: 317-324.
- [197] San Román JA, Vilacosta I, Zamorano JL, Almería C and Sánchez-Harguindey L. Transesophageal echocardiography in right-sided endocarditis. *J Am Coll Cardiol* 1993; 21: 1226-1230.
- [198] David TE, Gavra G, Feindel CM, Regesta T, Armstrong S and Magati MD. Surgical treatment of active infective endocarditis: a continued challenge. *J Thorac Cardiovasc Surg* 2007; 133: 144-149.
- [199] Nakagawa T, Wada H, Sakakura K, Yamada Y, Ishida K, Ibe T, Ikeda N, Sugawara Y, Ako J and Momomura SI. Clinical features of infective endocarditis: comparison between the 1990s and 2000s. *J Cardiol* 2013 Jul 29. pii: S0914-5087(13)00193-7.
- [200] Fernández Guerrero ML, González López JJ, Goyenechea A, Fraile J and de Górgolas M. Endocarditis caused by *Staphylococcus aureus*: A reappraisal of the epidemiologic, clinical, and pathologic manifestations with analysis of factors determining outcome. *Medicine (Baltimore)* 2009; 88: 1-22.
- [201] Ota T, Gleason TG, Salizzoni S, Wei LM, Toyoda Y and Bermudez C. Midterm surgical outcomes of noncomplicated active native multivalve endocarditis: single-center experience. *Ann Thorac Surg* 2011; 91: 1414-1419.
- [202] Martín-Dávila P, Navas E, Fortún J, Moya JL, Cobo J, Pintado V, Quereda C, Jiménez-Mena M and Moreno S. Analysis of mortality and risk factors associated with native valve endocarditis in drug users: the importance of vegetation size. *Am Heart J* 2005; 150: 1099-1106.