We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,300

Open access books available

172,000

International authors and editors

190M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Complications of Endocarditis

Yongping Wang and Aifeng Wang

Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/56091>

1. Introduction

Infective endocarditis (IE) is an endovascular infection and inflammation with vegetation formation, usually caused by infectious agents. Bacterial infection is most common for IE, but other pathogens such as fungi, rickettsia, chlamydia and virus, can also cause IE [1]. The vegetations vary in size and shape and are composed of platelet and fibrin blocks with plenty of microbial and small amounts of inflammatory cells inside [2]. IE occurs mostly in the patients with cardiac abnormalities or lesions including rheumatic heart disease, ventricular septal defect, patent ductus arteriosus, valvular stenosis or incompetence [3]. Nevertheless, IE can also be seen in other conditions, such as valve replacement, pacemaker implantation, intravenous drug users and a few people without cardiac lesions. Most patients are young and clinically exhibit low to mild fever, progressive anemia, asthenia, night sweat, hepatosplenomegaly and clubbed finger (toe) [1-3].

The incidence of IE is 30 per million persons per year [4]. Despite major improvements in diagnosis and treatment, the mortality of IE still remains high at 14% of in-hospital and even higher at 20% to 30% complicated with age and heart failure, especially in developing countries [5,6]. Based on different criteria, IE can be divided by several classifications. By duration, IE can be classified to subacute bacterial endocarditis (SBE) and acute bacterial endocarditis (ABE); By culture result, IE can be divided by *staphylococcal* endocarditis, *streptococcal* endocarditis, *enterococcal* endocarditis, *fungus* endocarditis; By individual valve type, IE can be classified by native valve endocarditis, prosthetic valve endocarditis and endocarditis in intravenous drug abusers [2-4]. As reported in a study with an IE population of 223 episodes, complications occurred in 74% patients, including cardiac, neurological, septic, renal, embolism and infarction/ abscess [7]. It also suggests that neurologic and septic complications are the leading causes of death in IE patients [7]. With the improvement of diagnosis and therapy, the frequencies of IE complications have changed. For example, septic and embolic symptoms are relatively rare because of early and adequate dosage of antibiotic therapy [4]. On the contrary, there are increasing

complications in prosthetic valve IE and intravenous drug abuse patients which may bring new challenge for clinical diagnosis and therapy. Since there are different diagnostic criteria for IE, it is difficult to conclude the true incidence of IE [8]. Overall, diagnosis is missed until autopsy in 38.2% of cases, especially when the patients are absence of fever, cardiac murmurs and other typical symptoms of IE [9]. In this chapter, several most common complications and related pathophysiologic process in IE patients will be summarized.

2. Intracardiac infection and local spread

Cardiac complications are the most common complications seen in IE patients, occurring in one-third to one-half of patients in most recent case series [10, 11].

2.1. Congestive Heart Failure (CHF)

CHF is a leading cause of death in IE patients [12]. The size of nodular or polypoid-like vegetation varies from 1 micrometer to several centimeters which can block the valve entrance [13]. The vegetation can cause valvular perforation and lead to prolapse. When the infection is controlled, valve lesions may still go on with fibrosis and contracture in some cases. All of these pathologic changes induce valvular insufficiency, mainly involved in aortic valve (50%), mitral valve (40%) and tricuspid valve (6%) [14]. A study on 511 IE patients show that moderate or severe congestive heart failure accounts for 44% in total complications [15]. According to the report enrolling 4166 IE patients from multi centers in 28 countries, 33.4% patients have heart failure and 66.7% of the heart failure patients are classified as New York Heart Association class III or IV symptom status [16]. The total in-hospital mortality is 29.7% for the CHF cohort. They conclude that the severities of CHF in IE patients are associated with surgical status and mortality [16]. Based on the seriousness of CHF in the course of IE patients, the timing for early prophylaxis and treatment will influence its prognostic significance.

2.2. Cardiac abscess

Abscess is most common in acute patients and may happen anywhere in the heart [17]. The most frequent cardiac abscess occurs in aortic root of 56.5% of (26/46) IE patients, and the infecting organism is staphylococcus (52.3%) in patients with abscesses more often than in those without abscesses (16.2%) [17]. Occasionally, cardiac abscess can cause papillary muscle rupture, ventricular septal perforation, and even purulent pericarditis when the infection infiltrates the myocardial wall [13]. Moreover, the infection beyond the valve annulus can spread to adjacent structures and cause an emergency and higher mortality which can be observed in 88% of cases [18]. Another study on a large number of cases with valvular ring abscesses suggests that early identification of abscesses is particularly important to improve the outcome after timely surgery [19]. They also conclude that the overall operative mortality is not correlated with patient's age, *staphylococcal* infection or abscess fistulization [19]. However, the diagnosis is difficult, especially for small abscesses located on the anterior aortic wall and mitral abscesses.

3. Embolic

Embolization is frequently observed in IE patients and can even occur in patients undergoing therapies. IE patients have high prevalence of embolic complications for 13-49% [20]. In a study of 65 IE patients, a total of 37 (56.9%) patients are diagnosed with a cerebral embolism (overt 13, clinically silent 24) by blood test, cultures, echocardiography, and MRI/CT imaging [21]. Their results suggest that both overt and clinical silent central nervous system embolism are common complications of IE patients and silent embolism needs further imaging tools for examination [21]. Recently, their group uses some new biological markers, such as S-100B, to predict embolism in central nervous system during the course of IE [22]. Since most of the S-100B protein are synthesized by astrocytes and released from damaged neural tissues, the biomarker provides another specific method for screening CNS stroke in IE patients. An early study shows that the microorganism, but not vegetations on echocardiography, is associated with a significantly higher risk for embolus in patients with left-sided IE [23]. With transthoracic echocardiography in predicting embolic events, another group found that the vegetations bigger than 10 mm were associated with a 50% incidence of embolic events, while vegetations less than 10 mm had a 42% incidence of emboli in IE patients [24]. Similarly, in the patients who are diagnosed with acute IE and have no confirmed or suspected embolism before, 44% (25 in 57) have embolic events by using both transthoracic and transesophageal echocardiograms [25]. They also suggest the characteristics of vegetations identified by echocardiograms are not helpful in predicting embolic risk in IE patients [25]. In combination with clinical antibiotic therapy, embolism occurred in 34.1% (131 in 384) patients before/after IE diagnosis and in 7.3% (28 in 384) patients after initiation of therapy [26].

Besides neurological embolism, other organs such as spleen, kidney, lung and limbs, are also involved in rare cases [27, 28]. According to a report by Luaces-Méndez *et al*, there are 10% hepatosplenic and renal embolisms infarctions in left-sided IE patients with characteristic clinical features [29]. But in fungi caused IE, emboli occurs in 25%(41 in 162) patients and symptomatic embolization appears to be more common of 17% (7 in 41) in peripheral limb, 7% (3 in 41) in pulmonary, 5% (2 in 41) in mesenteric [30].

The characteristics of bacterial embolus are multiple, fragile and movable, so in many cases IE patients present with stroke, meningitis, brain abscess and bacterial aneurysm.

3.1. Stroke

Ischemic and hemorrhagic strokes are important neurological complications and are frequent in IE patients during uncontrolled infection. There are about 21% complicated by stroke in 212 IE patients in a study between 1978 and 1986 [31]. In a population of 214 IE patients undergoing cardiac surgery, the prognosis for patients with uncomplicated ischemic stroke are better than patients with complicated stroke (meningitis, hemorrhage, or brain abscess) after 20 years following up [32]. In order to prevent cardioembolic stroke, additional diagnostic tools such as echocardiography and cardiac magnetic resonance imaging, can be applied to identify the sources of cardiac embolism. Another investigation in 707 patients who are diagnosed with

possible IE, strokes occur in 9.6% of total cases, which is lower than previous reports (21 to 39%) [33]. In order to study the relationship between vegetation 2-dimensional size and stroke in those IE patients, researchers use Duke Endocarditis Database to examine 145 IE patients and find 23.4% (34 in 145) complicated by stroke, suggesting vegetation 2-dimensional size and characteristics as predictors for stroke and mortality [34].

3.2. Mycotic aneurysm

If the artery is blocked by the septic embolus, the wall may be necrosised and destroyed, and then develop bacterial aneurysm. Mycotic aneurysm is rare, about 4% (23 in 513), in IE patients [15]. Aorta, brain, viscera and limbs can become involved in turn [35]. Patients can show throbbing lump during the late stage. The disease is easily diagnosed while occurring at peripheral vessels. However, when the lesion happens in deep arteries such as brain and mesentery, aneurysm is always ignored until it is broken and bleeding [36]. Mycotic aneurysm has a high mortality rate for its potential catastrophic rupture but can be prevented by early diagnostic imaging techniques [37]. Cerebral mycotic aneurysms tend to occur in the more distal portions of the middle cerebral artery, especially in the region of the sylvian fissure, which clinically is different from berry aneurysms occurring near the Willis circle [38].

3.3. Cerebral hemorrhage

Cerebral hemorrhage will occur when the vessel is broken in bacterial aneurysm or embolism. It is easy to develop spotted or patched hemorrhage when there is big area of infarction in the brain. There are three different mechanisms for cerebral haemorrhage in IE patients: rupture of a mycotic aneurysm, septic arteritis without aneurysm, spontaneous haemorrhagic transformation of a blank brain infarction [39].

Subarachnoid hemorrhage is a rare but dramatic neurologic complication in IE patients and is always associated with aneurysm rupture in the early phase [40]. Previous reports show that high mortality is related to intracerebral haemorrhage [41, 42]. The species of microorganism seem to have relationship with brain haemorrhage. Data presented in an investigation shows that brain haemorrhages in 40.7% (35 in 86) IE patients are caused by *Staphylococcus aureus* [43].

4. Hematogenous dissemination

4.1. Metastatic abscess

When the vegetation with infecting bacteria drops off, it will migrate with blood and cause embolism in the artery. During a report on 118 IE patients, 44 (37.3 %) patients have 46 definite regions of abscess in total and abscesses present more frequently in endocarditis from aortic-valve than other valves [17]. If the infection is not controlled well, abscesses can develop in the spleen, kidney, brain or soft tissues in IE patients. With the improvement of antibiotic treatment, metastatic abscess is relatively rare but still reported in recent years [44-46].

Splenic abscesses are found in up to 5% IE patients and usually exhibit abdominal pain, pleuritic or shoulder pain as of diaphragmatic irritation, or persistent fever [47]. According to the conclusion from 27 patients with splenic abscess, the cases could not survive without a timely splenectomy surgery [48]. If possible, the patient with IE should be treated first for splenic abscesses and then splenectomy should be performed for the requirements.[49].

Cerebral abscess is rare in IE patients including suppurative encephalitis, chronic granuloma and abscess envelope [50]. The time for envelope formation depends on the types of bacteria and the toxicity, body's resistance and reaction for antibiotic therapy [51]. According to the report on a series of cases, miliary microscopic abscesses are more common than macroscopic cerebral abscess in bacterial endocarditis patients, particularly in patients with acute miliary infection [41]. Cerebral abscesses in some IE patients are suggested to be related with *S. aureus* infection and purulent meningitis [41].

4.2. Toxic encephalopathy

Toxic encephalopathy occurs when plenty of bacteria enter the circulation and cause septicemia. In an investigation of 110 patients, 19.1% (21 in 110) show toxic encephalopathy, which is ranked the second common neurological manifestation of IE patients [52]. Frequently, the patients display a variety of symptoms, such as early stage of headache, dizziness, hypersomnia, nausea, vomit and late stage of hallucination, memory loss, small personality changes, seizures, disturbance of consciousness. The multiple cerebral emboli and multifocal microinfarcts cause formation of microabscesses, which may explain the pathophysiological mechanism for acute encephalopathy [53].

4.3. Purulent meningitis

This complication is uncommon and concomitant with cerebral abscess. Sometimes the intracerebral abscess may enter subarachnoid cavity and cerebral ventricle to invade meninges, which will cause purulent meningitis [54]. If *Streptococcus anaerobius*, *bacteroid*, *Staphylococcus* and mixed bacteria are separated in cerebrospinal fluid, it suggests that there are relationships between meningitis and broken intracerebral abscess.

5. Musculoskeletal complications

The musculoskeletal manifestations include spondylodiscitis, osteomyelitis, septic arthritis and peripheral soft tissue abscess, which occur frequently in up to 44% cases [55]. Because the existing of osteoarticular complications, the patients are at a higher risk of having major embolic events from the central nerve system to lungs [56]. Vertebral osteomyelitis is relatively rare complication in IE patients. Overall, 4.6% (28 in 606) cases in IE patients have pyogenic vertebral osteomyelitis [57]. The patients are needed to exclude IE if they have spondylodiscitis and pre-existing heart disease or microbiologic infection [58]. With MRI as a highly sensitive and specific tool for diagnosing, patients with spondylodiscitis usually can be found early before infection has spread to two vertebral body levels.

In IE patients, the percentages of osteomyelitis and septic arthritis are up to 4.3%, and they occur more frequently in patients with tricuspid valve involvement [56]. *S. aureus* are reported to have higher frequencies than other microorganisms for causing osteomyelitis complication in IE patients [59]. The infections usually occur at large joints and involve one or more joints, including the knee, shoulder, elbow, hip and sacroiliac joints [56]. If the patients are infected with multiple joints but don't have joint infection or trauma, they are suspicious for septic arthritis with IE.

6. Immune-mediated damage

With persistent bacteremia existing in IE patients, clinical manifestations, such as splenomegaly, glomerulonephritis and arthritis, may present because of cellular and humoral-mediated immune response [60]. Patients with splenomegaly occur in 20% of cases and are more likely in patients who have been ill for months rather than for days or weeks. Glomerulonephritis are most common in *S. aureus* caused ABE and *S. viridans* caused SBE with histologic immune deposits in the glomerular capillary wall [61]. With antibiotic prophylaxis and therapy in IE patients, the incidence of glomerulonephritis decreases to about 4.5% (9 in 198) in an investigation [62]. Under rare conditions, patients with glomerulonephritis will develop diffuse proliferative glomerulonephritis and extensive crescent formation with renal failure [63].

Other clinical manifestations, such as arthritis, pericarditis and micro-vessel vasculitis are also found in IE patients. Vasculitis may cause unspecific signs on skin and mucosa, including subconjunctival and soft palate petechiae, hemorrhages within the nail beds (splinter hemorrhages), oval retinal bleeding spots with white center (Roth spots), painful subcutaneous nodules on the palms or soles (Osler's nodes), painless bleeding spots on the palms and soles with diameter of 1 to 4 mm (Janeway lesions) [1]. The pathogenesis for above lesions may be caused by microemboli and microabscesses in the small vessels of dermis.

In summary, multidisciplinary approaches including clinician microbiologists, radiology, cardiology and surgery are necessary for treatment of IE with complications. In order to get better understanding, the complications are classified into several categories although some features are overlapping or broad. For example, the IE patients with embolic complication can have metastatic abscess, mycotic aneurysm or cerebral hemorrhage; patients with hematogenous dissemination can simultaneously have embolic and musculoskeletal symptoms. This content tries to describe the frequency of IE complications and may help for better prophylaxis and therapy.

Acknowledgements

We are grateful to Zhijian Duan at UC Davis for reviewing and editing this chapter. Yongping Wang is supported by grant from the Shriners Hospital for Children [84204 to Y.P.W].

Author details

Yongping Wang^{1,2*} and Aifeng Wang^{3,4}

*Address all correspondence to: wypwang@ucdavis.edu

1 Department of Cell Biology and Human Anatomy, University of California, Davis, USA

2 Institute of Pediatric Regenerative Medicine, Shriners Hospital for Children-North California, University of California, Davis, USA

3 Department of Biochemistry and Molecular Medicine, University of California, Davis, USA

4 Department of Forensic Medicine, Preclinical Medical College, Southern Medical University, Guangzhou, Guangdong, China

References

- [1] Baddour LM, Wilson WR, Bayer AS, Fowler VG Jr, Bolger AF, Levison ME, et al. Council on Cardiovascular Disease in the Young; Councils on Clinical Cardiology, Stroke, and Cardiovascular Surgery and Anesthesia; American Heart Association; Infectious Diseases Society of America. Infective endocarditis: diagnosis, antimicrobial therapy, and management of complications: a statement for healthcare professionals from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, and the Councils on Clinical Cardiology, Stroke, and Cardiovascular Surgery and Anesthesia, American Heart Association: endorsed by the Infectious Diseases Society of America. *Circulation* 2005, 111: e394-434.
- [2] Bayer AS, Bolger AF, Taubert KA, Wilson W, Steckelberg J, Karchmer AW, et al. Diagnosis and management of infective endocarditis and its complications. *Circulation* 1998, 98: 2936-2948.
- [3] Horstkotte D, Follath F, Gutschik E, Lengyel M, Oto A, Pavie A, et al. Guidelines on prevention, diagnosis and treatment of infective endocarditis: executive summary; the Task Force on Infective Endocarditis of the European Society of Cardiology. *Eur Heart J* 2004, 25: 267-276.
- [4] Mylonakis E, Calderwood SB. Infective endocarditis in adults. *N Engl J Med* 2001, 345: 1318-1330.
- [5] Tariq M, Alam M, Munir G, Khan MA, Smego RA Jr. Infective endocarditis: a five-year experience at a tertiary care hospital in Pakistan. *Int J Infect Dis* 2004, 8: 163-170.

- [6] Fedeli U, Schievano E, Buonfrate D, Pellizzer G, Spolaore P. Increasing incidence and mortality of infective endocarditis: a population-based study through a record-linkage system. *BMC Infect Dis* 2011, 11: 48.
- [7] Mansur AJ, Grinberg M, da Luz PL, Bellotti G. The complications of infective endocarditis. A reappraisal in the 1980s. *Arch Intern Med* 1992, 152: 2428-2432.
- [8] Heiro M, Nikoskelainen J, Hartiala JJ, Saraste MK, Kotilainen PM. Diagnosis of infective endocarditis. Sensitivity of the Duke vs von Reyn criteria. *Arch Intern Med* 1998, 158: 18-24.
- [9] Fernández Guerrero ML, Álvarez B, Manzarbeitia F, Renedo G. Infective endocarditis at autopsy: a review of pathologic manifestations and clinical correlates. *Medicine (Baltimore)* 2012, 91: 152-164.
- [10] Millaire A, Van Belle E, de Groote P, Leroy O, Ducloux G. Obstruction of the left main coronary ostium due to an aortic vegetation: survival after early surgery. *Clin Infect Dis* 1996, 22: 192-193.
- [11] Nadji G, Rusinaru D, Réyadi JP, Jeu A, Sorel C, Tribouilloy C. Heart failure in left-sided native valve infective endocarditis: characteristics, prognosis, and results of surgical treatment. *Eur J Heart Fail* 2009, 11: 668-675.
- [12] Delahaye F, Alla F, Béguinot I, Bruneval P, Doco-Lecompte T, Lacassin F, et al. In-hospital mortality of infective endocarditis: prognostic factors and evolution over an 8-year period. *Scand J Infect Dis* 2007, 39: 849-857.
- [13] Bashore TM, Cabell C, Fowler V Jr. Update on infective endocarditis. *Curr Probl Cardiol* 2006, 31: 274-352.
- [14] Lalani T, Cabell CH, Benjamin DK, Lasca O, Naber C, Fowler VG Jr, et al. International Collaboration on Endocarditis-Prospective Cohort Study (ICE-PCS) Investigators. Analysis of the impact of early surgery on in-hospital mortality of native valve endocarditis: use of propensity score and instrumental variable methods to adjust for treatment-selection bias. *Circulation* 2010, 121: 1005-1013.
- [15] Hasbun R, Vikram HR, Barakat LA, Buenconsejo J, Quagliarello VJ. Complicated left-sided native valve endocarditis in adults: risk classification for mortality. *JAMA* 2003, 289: 1933-1940.
- [16] Kiefer T, Park L, Tribouilloy C, Cortes C, Casillo R, Chu V, et al. Association between valvular surgery and mortality among patients with infective endocarditis complicated by heart failure. *JAMA* 2011, 306: 2239-2247.
- [17] Daniel WG, Mugge A, Martin RP, Lindert O, Hausmann D, Nonnast-Daniel B, et al. Improvement in the diagnosis of abscesses associated with endocarditis by transesophageal echocardiography. *N Engl J Med* 1991, 324: 795-800.

- [18] Sampedro MF, Patel R. Infections associated with long-term prosthetic devices. *Infect Dis Clin North Am* 2007, 21: 785-819.
- [19] Choussat R, Thomas D, Isnard R, Michel PL, Iung B, Hanania G, et al. Perivalvular abscesses associated with endocarditis; clinical features and prognostic factors of overall survival in a series of 233 cases. Perivalvular Abscesses French Multicentre Study. *Eur Heart J* 1999, 20: 232-241.
- [20] Habib G. Embolic risk of subacute bacterial endocarditis: role of transesophageal echocardiography. *Curr Cardiol Rep* 2003, 5: 129-136.
- [21] Grabowski M, Hryniewiecki T, Janas J, Stępińska J. Clinically overt and silent cerebral embolism in the course of infective endocarditis. *J Neurol* 2011, 258: 1133-1139.
- [22] Grabowski M, Hryniewiecki T, Stępińska J. Novel markers of cerebral embolism in the course of infective endocarditis. *Int J Cardiol* 2012, 154: 90-92.
- [23] Steckelberg JM, Murphy JG, Ballard D, Bailey K, Tajik AJ, Taliencio CP, et al. Emboli in infective endocarditis: the prognostic value of echocardiography. *Ann Intern Med* 1991, 114: 635-640.
- [24] Heinle S, Wilderman N, Harrison JK, Waugh R, Bashore T, Nicely LM, et al. Value of transthoracic echocardiography in predicting embolic events in active infective endocarditis. Duke Endocarditis Service. *Am J Cardiol* 1994, 74: 799-801.
- [25] De Castro S, Magni G, Beni S, Cartoni D, Fiorelli M, Venditti M, et al. Role of transthoracic and transesophageal echocardiography in predicting embolic events in patients with active infective endocarditis involving native cardiac valves. *Am J Cardiol* 1997, 80: 1030-1034.
- [26] Thuny F, Di Salvo G, Belliard O, Avierinos JF, Pergola V, Rosenberg V, et al. Risk of embolism and death in infective endocarditis: prognostic value of echocardiography: a prospective multicenter study. *Circulation* 2005, 112: 69-75.
- [27] Pessinaba S, Kane A, Ndiaye MB, Mbaye A, Bodian M, Dia MM, et al. Vascular complications of infective endocarditis. *Med Mal Infect* 2012, 42: 213-217.
- [28] Ting W, Silverman NA, Arzouman DA, Levitsky S. Splenic septic emboli in endocarditis. *Circulation* 1990, 82: IV105-109.
- [29] Luaces Méndez M, Vilacosta I, Sarriá C, Fernández C, San Román JA, Sanmartín JV, et al. Hepatosplenic and renal embolisms in infective endocarditis. *Rev Esp Cardiol* 2004, 57: 1188-1196.
- [30] Ellis ME, Al-Abdely H, Sandridge A, Greer W, Ventura W. Fungal endocarditis: evidence in the world literature, 1965-1995. *Clin Infect Dis* 2001, 32: 50-62.
- [31] Hart RG, Foster JW, Luther MF, Kanter MC. Stroke in infective endocarditis. *Stroke* 1990, 21: 695-700.

- [32] Ruttmann E, Willeit J, Ulmer H, Chevtchik O, Höfer D, Poewe W, et al. Neurological outcome of septic cardioembolic stroke after infective endocarditis. *Stroke* 2006, 37: 2094-2099.
- [33] Anderson DJ, Goldstein LB, Wilkinson WE, Corey GR, Cabell CH, Sanders LL, et al. Stroke location, characterization, severity, and outcome in mitral vs. aortic valve endocarditis. *Neurology* 2003, 61:1341-1346.
- [34] Cabell CH, Pond KK, Peterson GE, Durack DT, Corey GR, Anderson DJ, et al. The risk of stroke and death in patients with aortic and mitral valve endocarditis. *Am Heart J* 2001, 142: 75-80.
- [35] Dubois M, Daenens K, Houthoofd S, Peetermans WE, Fourneau I. Treatment of mycotic aneurysms with involvement of the abdominal aorta: single-centre experience in 44 consecutive cases. *Eur J Vasc Endovasc Surg* 2010, 40: 450-456.
- [36] Ziment, I. Nervous system complications in bacterial endocarditis. *Am J Med* 1969; 47:593.
- [37] Shaikholeslami R, Tomlinson CW, Teoh KH, Molot MJ, Duke RJ. Mycotic aneurysm complicating staphylococcal endocarditis. *Can J Cardiol* 1999, 15: 217-222.
- [38] Jones HR Jr, Siekert RG. Neurological manifestations of infective endocarditis: Review of clinical and therapeutic challenges. *Brain* 1989, 112: 1295-1315.
- [39] Hart RG, Kagan-Hallet K, Joerns SE. Mechanisms of intracranial hemorrhage in infective endocarditis. *Stroke* 1987, 18: 1048-1056.
- [40] Kanter MC, Hart RG. Neurologic complications of infective endocarditis. *Neurology* 1991, 41: 1015-1020.
- [41] Pruitt AA, Rubin RH, Karchmer AW, Duncan GW. Neurologic complications of bacterial endocarditis. *Medicine (Baltimore)* 1978, 57:329-343.
- [42] Heiro M, Nikoskelainen J, Engblom E, Kotilainen E, Marttila R, Kotilainen P. Neurologic manifestations of infective endocarditis: a 17-year experience in a teaching hospital in Finland. *Arch Intern Med* 2000, 160: 2781-2787.
- [43] LeCam B, Guivarch G, Boles JM, Garre M, Cartier F. Neurologic complications in a group of 86 bacterial endocarditis. *Eur Heart J* 1984, 5 (Suppl C): 97-100.
- [44] Wang CC, Lee CH, Chan CY, Chen HW. Splenic infarction and abscess complicating infective endocarditis. *Am J Emerg Med* 2009, 27: 1021.e3-5.
- [45] Wadhwa R, Thakur JD, Nanda A, Guthikonda B. Sterile hemorrhagic brain abscess in infective endocarditis. *Neurol India* 2012, 60: 240-242.
- [46] Kanter MC, Hart RG. Neurologic complications of infective endocarditis. *Neurology*, 1991, 41: 1015-1020.

- [47] Mansur AJ, Grinberg M, da Luz PL, Bellotti G. The complications of infective endocarditis. A reappraisal in the 1980s. *Arch Intern Med* 1992, 152: 2428–2432.
- [48] Robinson, SL, Saxe, JM, Lucus, CE, Arbulu A, Ledgerwood AM, Lucas WF. Splenic abscess associated with endocarditis. *Surgery* 1992, 112: 781-786.
- [49] Bayer AS, Bolger AF, Taubert KA, Wilson W, Steckelberg J, Karchmer AW, et al. Diagnosis and management of infective endocarditis and its complications. *Circulation* 1998, 98: 2936-2948.
- [50] Muzumdar D, Jhawar S, Goel A. Brain abscess: an overview. *Int J Surg* 2011, 9: 136-144. Epub 2010 Nov 16.
- [51] Mathisen GE, Johnson JP. Brain abscess. *Clin Infect Dis* 1997, 25: 763-779.
- [52] Jones HR Jr, Siekert RG, Geraci JE. Neurologic manifestations of bacterial endocarditis. *Annals of Internal Medicine* 1969, 71: 21-28.
- [53] Jones HR Jr, Siekert RG. Neurological manifestations of infective endocarditis. Review of clinical and therapeutic challenges. *Brain* 1989, 112: 1295-1315.
- [54] Heiro M, Nikoskelainen J, Engblom E, Kotilainen E, Marttila R, Kotilainen P. Neurologic manifestations of infective endocarditis. *Arch Intern Med* 2000, 160: 2781–2787.
- [55] Churchill MA Jr, Geraci JE, Hunder GG. Musculoskeletal manifestations of bacterial endocarditis. *Ann Intern Med* 1977, 87: 754–759.
- [56] Lamas C, Boia M, Eykyn SJ. Osteoarticular infections complicating infective endocarditis: a study of 30 cases between 1969 and 2002 in a tertiary referral centre. *Scand J Infect Dis* 2006, 38: 433–440.
- [57] Pigrau C, Almirante B, Flores X, Falco V, Rodripuez D, Gasser I, et al. Spontaneous pyogenic vertebral osteomyelitis and endocarditis: incidence, risk factors, and outcome. *Am J Med* 2005, 118:1287.
- [58] Morelli S, Carmenini E, Caporossi AP, Aguglia G, Bernardo ML, Gurgo AM. Spondylodiscitis and infective endocarditis: case studies and review of the literature. *Spine* 2001, 26: 499–500.
- [59] Di Salvo G, Habib G, Pergola V, Avierinos JF, Philip E, Casalta JP. Echocardiography predicts embolic events in infective endocarditis. *J Am Coll Cardiol* 2001, 37: 1069-1076.
- [60] Burton-Kee J, Morgan-Capner P, Mowbray JF. Nature of circulating immune complexes in infective endocarditis. *J Clin Pathol* 1980, 33: 653-659.
- [61] Neugarten J, Baldwin DS. Glomerulonephritis in bacterial endocarditis. *Am J Med.* 1984, 77: 297-304.

- [62] Garg N, Kandpal B, Garg N, Tewari S, Kapoor A, Goel P, et al. Characteristics of infective endocarditis in a developing country-clinical profile and outcome in 192 Indian patients, 1992-2001. *Int J Cardiol* 2005, 98: 253-260.
- [63] Kannan S, Mattoo TK. Diffuse crescentic glomerulonephritis in bacterial endocarditis. *Pediatr Nephrol* 2001, 16: 423-428.

IntechOpen

IntechOpen