The Clinical Significance of Cerebral Microbleeds in Infective Endocarditis Patients

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We assessed the clinical features of cerebral microbleeds (CMBs) and their association with clinical outcomes in active infective endocarditis patients. From January 2009 to June 2015, 132 active IE patients diagnosed per the modified Duke's criteria were retrospectively reviewed. Brain magnetic resonance imaging was performed in 102 patients, and 74 patients whose image data were available to assess CMBs were enrolled. CMBs were defined as hypointense lesion <10 mm in diameter, seen on T2* or susceptibility-weighted imaging. Forty patients had CMB and 34 did not. Patients with CMB were older, and the proportion of prior antiplatelet therapy, staphylococcal infection, and prosthetic valve endocarditis were higher than in patients without CMB. Surgery was performed in 25 (63%) patients with CMB and 24 (71%) patients without CMB. There was no significant difference in the de novo stroke incidence postoperatively (16% vs 17%, $P = 0.95$). Although all-cause mortality rate tended to be higher in patients with CMB, there were no significant differences in the in-hospital mortality rate and estimated 1-year major adverse event rate between the 2 groups (13% vs 12%, $P = 0.92$; 20% vs 19%, $P = 0.35$). Cox regression analysis adjusting age and operative risk did not show that CMB was a significant risk factor for all-cause death and major adverse event. Patients with CMB were older than those without, and microbleeds were associated with antiplatelet therapy, staphylococcal infection, and prosthetic valve endocarditis. However, the mid-term clinical outcomes of patients with CMB and those without were comparable.

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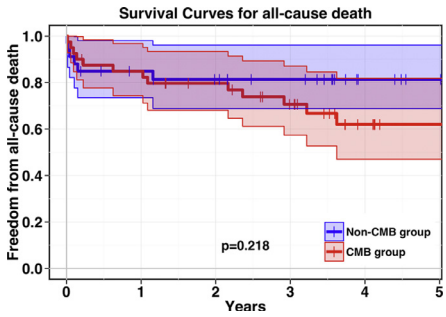
Keywords: Infective endocarditis, Cerebral microbleeds, Surgery



Freedom from all-cause death in patients with/without cerebral microbleeds.

Central Message

The mid-term prognosis of patients with and without cerebral microbleeds was comparable, suggesting that it might not have a major impact on clinical outcomes in patients with infective endocarditis.

Perspective Statement

Although cerebral microbleed is suggested to predict the development of intracranial hemorrhage in patients with infective endocarditis, the clinical significance is not well known. This study showed that the mid-term prognosis of patients with and without cerebral microbleeds was comparable, suggesting that it might not have a major impact on clinical outcomes in patients with infective endocarditis.

Abbreviations: CMB, cerebral microbleeds; Cr, creatinine; DM, diabetes mellitus; DWI, diffusion-weighted imaging; EuroSCORE, European System for Cardiac Operative Risk Evaluation; HT, hypertension; IE, infective endocarditis; IQR, interquartile range; MACE, major adverse cardiac events; MRI, magnetic resonance imaging; NIHSS, National Institutes of Health Stroke Scale; PVE, prosthetic valve endocarditis; RSIE, right-sided infective endocarditis; SWI, susceptibility-weighted imaging; T2*WI, T2-star-weighted imaging

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INTRODUCTION

Cerebrovascular complications occur in 20–40% of patients with active infective endocarditis (IE).[1–4] Symptomatic neurologic complications are associated with poorer clinical outcomes.[5] To prevent systemic emboli and improve mortality, early surgical intervention in patients with active IE can be beneficial.[6] However, there is controversy about early surgery in patients with cerebral complications, especially those with cerebral hemorrhage, concerning deterioration of neurologic complications postoperatively.[7] The current guidelines recommend postponing cardiac surgery for more than 1 month in patients with cerebral hemorrhage.[8]

Brain magnetic resonance imaging (MRI) is thought to be a useful tool for diagnosing IE and deciding treatment

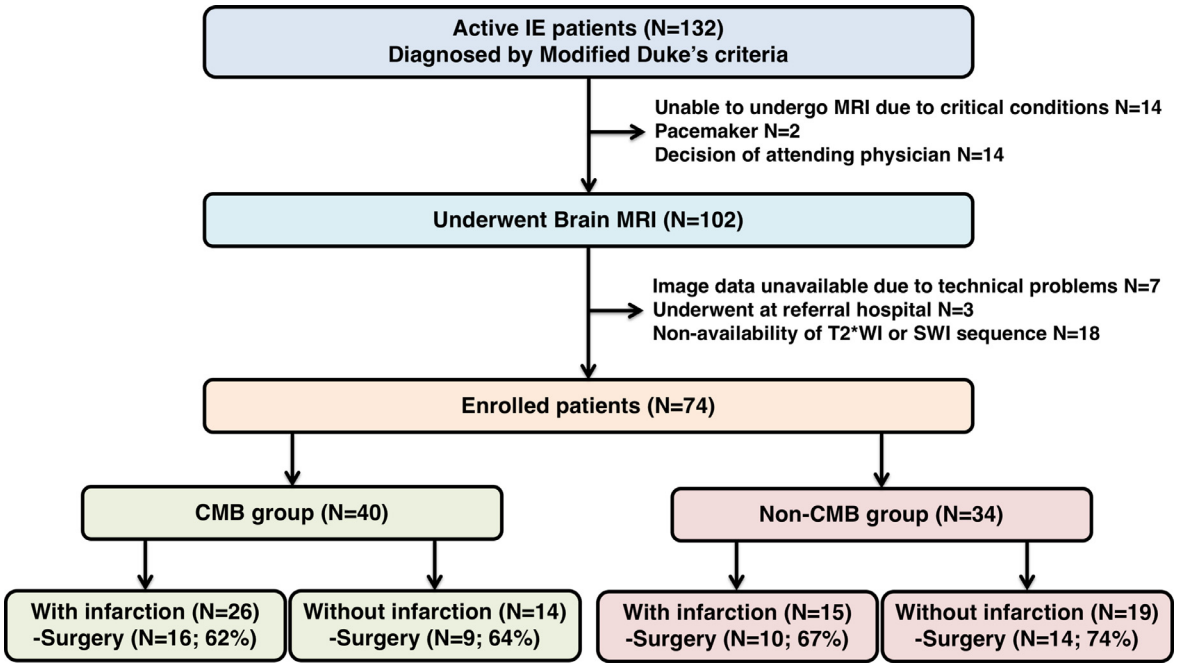

Figure 1. Flow diagram of study enrollment. CMB, cerebral microbleeds; IE, infective endocarditis; MRI, magnetic resonance imaging; T2*WI, T2-star-weighted image.

strategies.[9] Brain MRI has excellent sensitivity in diagnosing acute ischemic infarction, even if the lesion is small or asymptomatic. In addition to detecting ischemic lesions, MRI reveals asymptomatic, small, cerebral hemorrhages that are called cerebral microbleeds (CMBs). CMB is the most frequent abnormality that is seen on brain MRI in active IE patients[10,11] and is thought to be an alternative sign of the formation of mycotic aneurysms.[12] Additionally, CMB formation is suggested to predict the development of intracranial hemorrhage during the clinical course of active IE.[13] Although the pathophysiology of CMB formation in IE patients is unclear, many investigators hypothesized that CMB may represent vascular vulnerability. However, the clinical significance and impact of CMB in patients with active IE have not yet been established. CMB might be associated with poorer in-hospital and clinical outcomes in patients with active IE. Accordingly, the purpose of our study was to clarify the association of CMB and its clinical characteristics with their impact on clinical outcomes in patients with active IE.

METHODS

Ethical Considerations

This study was approved by the institutional review board of Kobe City Medical Center General Hospital. The need to obtain informed consent was waived because of the retrospective nature of the study.

Study Patients and Clinical Data

From January 2009 to June 2015, 132 consecutive patients with active IE were admitted to our hospital. Active IE was diagnosed using the modified Dukes' criteria of definite or probable IE.[14] Of these, 102 patients underwent brain MRI. After excluding the patients in whom MR images for evaluating the presence of CMB were not available, 74 patients were finally enrolled in this study. Flow diagram of study patients is shown in Figure 1. Clinical data, images, and outcomes were obtained from the patients' medical records. Concomitant cerebral complications, such as cerebral infarction or major intracranial hemorrhage, were diagnosed using a series of MRI and computed tomography scans.

With regard to treatment strategy, the indications for valve surgery in this study population were as follows: heart failure refractory to medical therapy, persistent infection, repeat embolization, high embolic risk, and presence of perivalvular extension of IE. In addition, the late surgery was indicated when the patients had residual severe regurgitation after conventional treatment and the resolution of infection. In this study, early surgery was defined as surgical intervention within 14 days (2 weeks) after the initial diagnosis of IE.[15] Late surgery was defined as surgical intervention later than 14 days after the initial diagnosis of IE. The timing of surgery was based on the decision of the attending physician.

Neurologic Assessment and Brain MRI

Patients with neurologic symptoms were referred to neurologists and assessed with standardized neurologic examinations. NIHSS and other neurologic scale or scores were assessed when necessary. We also assessed NIHSS retrospectively from the medical record as previously reported[16] in the patients in whom NIHSS was not assessed prospectively by neurologists. Brain MRI was performed using a 1.5 or 3.0

Tesla MR system. We used standard-protocol diffusion-weighted imaging, fluid-attenuated inversion recovery, and MR angiography. In addition, T2-star-weighted imaging or susceptibility-weighted imaging (SWI) sequences were used to assess CMB. CMB was defined as T2* or SWI hypointense lesion less than 10 mm in diameter (Fig. 2).[10,17] Hypointense lesions equal or more than 10 mm in diameter were classified as cerebral hemorrhage. Brain MRI was performed simultaneously with the diagnosis of IE in 31 (42%) patients, previously in 4 (5%) patients at a median of 2 (1−2) days before IE diagnosis, and later in 39 (53%) patients at a median of 2 (1−9) days after IE diagnosis. The median time to obtain a cerebral MR image from the time IE was diagnosed was 1 day (interquartile range [IQR]: 0−3 days).

We divided the patients into 2 groups: the CMB and non-CMB groups, according to the existence of at least 1 CMB. Figure 1 shows the patient flow chart of the 2 groups.

Clinical Outcomes

All of the patients were followed up at the outpatient clinic every 4−8 weeks after discharge. Further follow-up was performed by the referring physician. A total of 11 (15%) patients could not be contacted and were lost to follow-up. The median follow-up period was 37 months ranging from 0 to 87 months (IQR 13−48 months). The primary endpoint was defined as all-cause death. The secondary endpoint was major adverse cardiac events (MACE), defined as a composite outcome of IE-related death, repeat surgery, and recurrence of IE.

Among patients undergoing cardiac surgery, new-onset neurologic symptom and asymptomatic stroke lesion detected by cerebral imaging were regarded as postoperative neurologic events.

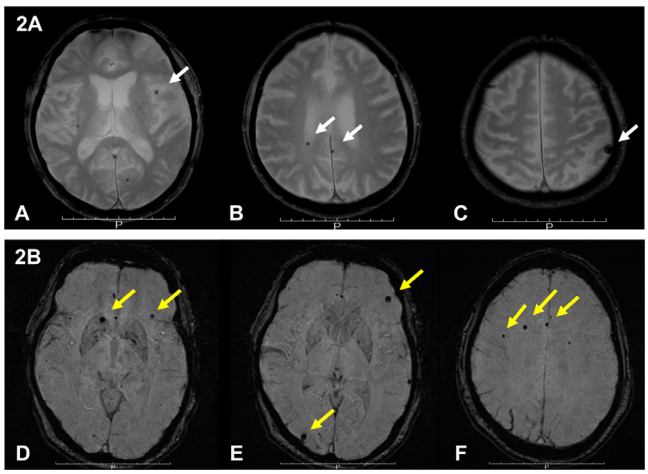

Figure 2. (A−C) Multiple microbleeds (white arrows) on a T2-star-weighted image in a patient with aortic prosthetic valve endocarditis. Microbleeds were located in the cortical area and around the lateral ventricles (white arrows). (D and E) Multiple microbleeds (yellow arrows) on a susceptibility-weighted image in another patient with aortic prosthetic valve endocarditis. Microbleeds were located in the cortical area, subcortical area, and basal ganglia (yellow arrows). (Color version of figure is available online.)

Statistical Analysis

Continuous variables are expressed as the mean and standard deviation or median and IQR. Categorical variables are expressed as numbers and percentages. We used a *t*-test or Wilcoxon's test for a univariate analysis of continuous variables, and a Chi-square test or Fisher's exact test for categorical variables. A survival analysis was performed with a Kaplan-Meier analysis, and differences between 2 groups were assessed with a log-rank test. The multivariate Cox proportional hazards model was used to assess the hazard risk of CMB. In all analysis, a P value of <0.05 was considered significant. All statistical analyses were performed using SPSS Statistics version 25.0 (IBM Corp., Armonk, NY) or R software package version 3.3.0.

RESULTS

Of the 74 patients in this study, 40 (54%) had at least 1 CMB (CMB group) and 34 (46%) did not have any CMB (non-CMB group). No patient had T2* or SWI hypointense lesions more than 10 mm in diameter. The median time to obtain a cerebral MR image from the time IE was diagnosed was 1 day (IQR: 0−3 days). Only 1 patient underwent MRI at referral hospital 1 day before admission. The other patient underwent MRI during index hospitalization. Among the patients undergoing cardiac surgery ($N = 49$), 46 patients underwent MRI before surgery (median 7 days, IQR 3−32), 1 underwent 14 days after surgery, and 2 underwent the day of surgery.

Cerebral Microbleeds and Clinical Background

Table 1 shows the baseline characteristics and concomitant neurologic findings of patients in the CMB group and those in the non-CMB group. Patients with CMB were older than non-CMB patients and more patients with CMB had prior antiplatelet therapy than did non-CMB patients. There was a significantly higher percentage of prosthetic valve endocarditis and staphylococcal endocarditis in the CMB group than in the non-CMB group. The percentages of concomitant cerebral infarction, intracranial hemorrhage, and angiographic aneurysm did not differ between the 2 groups. Of the 74 patients, only 1 had a mycotic aneurysm that was seen on MR angiography. However, of the 17 patients undergoing cerebral angiography, 5 had a concomitant mycotic aneurysm (Table 2).

Surgical Intervention and Neurologic Events

Table 3 shows the details of surgical interventions including indications for operations and operative risks between the 2 groups. A total of 44 patients (24 in the CMB group and 20 in the non-CMB group) underwent cardiac surgery during the initial admission period. Of these, 30 patients (17 in the CMB group and 13 in the non-CMB group) underwent early surgery within 2 weeks after the diagnosis of IE. There were no

Table 1. Baseline Characteristics and Clinical Outcomes According to Patient Group

	Non-CMB Group (*N* = 34)	CMB Group (*N* = 40)	*P*
Background			
Age (y), median (IQR)	59 (41–79)	70 (63–79)	0.008
Male, *n* (%)	23 (68)	21 (53)	0.186
HT, *n* (%)	11 (32)	18 (45)	0.267
DM, *n* (%)	5 (15)	8 (20)	0.551
Smoking history, *n* (%)	13 (38)	12 (30)	0.455
Renal failure (Cr > 2.0 mg/dL), *n* (%)	4 (12)	4 (10)	0.808
Dialysis, *n* (%)	3 (9)	5 (13)	0.612
History of IE, *n* (%)	3 (9)	3 (8)	0.835
History of PVE, *n* (%)	0 (0)	1 (3)	0.353
EuroSCORE logistic %, median (IQR)	12 (5–31)	24 (16–37)	0.014
Definite IE, *n* (%)	29 (85)	35 (88)	0.782
PVE, *n* (%)	2 (6)	11 (28)	0.015
RSIE, *n* (%)	5 (15)	1 (3)	0.055
Positive culture, *n* (%)	28 (82)	33 (83)	0.987
Staphylococcus spp., *n* (%)	4 (12)	13 (33)	0.035
Streptococcus spp., *n* (%)	20 (59)	15 (38)	0.067
Abscess, *n* (%)	7 (21)	7 (18)	0.735
Valve rupture, *n* (%)	7 (21)	10 (25)	0.653
Vegetation size >10 mm, *n* (%)	19 (56)	25 (63)	0.563
Aortic valve, *n* (%)	15 (44)	18 (45)	0.939
Mitral valve, *n* (%)	20 (59)	23 (58)	0.908
Aortic and mitral valve, *n* (%)	5 (15)	5 (13)	0.782
Systemic complications			
Systemic emboli, *n* (%)	15 (44)	11 (28)	0.136
Splenic infarction, *n* (%)	8 (24)	9 (23)	0.916
Renal infarction, *n* (%)	5 (15)	5 (13)	0.782
Heart failure, *n* (%)	6 (18)	6 (15)	0.758
Serum inflammation marker			
C-reactive protein, mg/dL, median (IQR)	8.1 (4.0–13.7)	5.3 (3.5–8.5)	0.300
Leukocyte count, μL, median (IQR)	9800 (7025–16,050)	8950 (6725–11,500)	0.229
Medication on admission			
Antiplatelet therapy	1 (3)	13 (33)	0.001
Oral anticoagulant therapy	3 (9)	8 (20)	0.178
Clinical outcomes			
All-cause death, *n* (%)	6 (18)	14 (35)	0.094
In-hospital death, *n* (%)	4 (12)	5 (13)	0.923
MACE, *n* (%)	8 (24)	15 (38)	0.196
IE death, *n* (%)	6 (18)	8 (20)	0.797
IE recurrence, *n* (%)	1 (3)	3 (8)	0.387
Repeat surgery, *n* (%)	2 (6)	2 (5)	0.867

CMB, cerebral microbleeds; Cr, creatinine; DM, diabetes mellitus; EuroSCORE, European System for Cardiac Operative Risk Evaluation; HT, hypertension; IE, infective endocarditis; IQR, interquartile range; MACE, major adverse cardiac events; PVE, prosthetic valve endocarditis; RSIE, right-sided IE, SD: standard deviation.

significant differences in postoperative neurologic events after early and late cardiac surgery between the 2 groups. Thirteen patients had CMB without cerebral infarction or hemorrhage. Of these, 8 patients underwent early or late surgery and did not have new onset or worsening of neurologic symptoms postoperatively. On the other hand, 26 patients had CMB and cerebral infarction, and 16 patients of them underwent cardiac surgery. Of these, neurologic worsening events occurred after cardiac surgery in 4 patients. Among 10 patients with CMB and cerebral hemorrhage, 2 of 8 patients undergoing cardiac surgery experienced neurologic worsening event postoperatively.

Cerebral Microbleeds and Clinical Outcomes

The clinical outcomes of the patients in each group are shown in Table 1. During the follow-up period, 14 (35%) patients in the CMB group and 6 (18%) in the non-CMB group died. In-hospital death and IE-related death occurred in 5 (13%) and 8 (20%) patients, respectively, in the CMB group, and in 4 (12%) and 6 (18%), respectively, in the non-CMB group. MACE occurred in 15 (38%) patients in the CMB group and 8 (24%) in the non-CMB group.

In the Kaplan-Meier analysis, there were no significant differences in all-cause mortality and MACE between the CMB

Table 2. Cerebral Lesions Observed on MR Imaging and Concurrent Neurologic Features in Each Group

	Non-CMB Group (*N* = 34)	CMB Group (*N* = 40)	*P*
MRI days from IE diagnosis, days median (IQR)	1 (0–3)	0 (0–3)	0.561
No. of CMBs, median (IQR)	–	4 (1–7)	–
CMB only, *n* (%)	–	13 (33)	–
Without significant lesion, *n* (%)	18 (53)	–	–
Concurrent MR findings			
DWI infarction, *n* (%)	15 (44)	26 (65)	0.072
T2* hemorrhage, *n* (%)	3 (9)	10 (25)	0.068
MR angiographic aneurysm, *n* (%)	0 (0)	1 (3)	0.353
Others neurologic complications			
Underwent cerebral angiography, *n* (%)	6 (18)	11 (28)	0.315
Angiographic aneurysms,* *n* (%)	0 (0)	5 (45)	0.050
Meningitis, *n* (%)	3 (9)	1 (3)	0.236
Brain abscess, *n* (%)	0 (0)	2 (5)	0.186
Clinical evaluation of neurologic symptom			
Symptomatic CNS complication, *n* (%)	10 (29)	11 (28)	0.858
NIHSS, median (IQR)	0 (0–4)	0 (0–1)	0.749

CMB, cerebral microbleeds; CNS, central nervous system; DM, diabetes mellitus; DWI, diffusion-weighted imaging, HT, hypertension; IE, infective endocarditis; MR, magnetic resonance; NIHSS, National Institutes of Health Stroke Scale; PVE, prosthetic valve endocarditis; RSIE, right-sided IE.
*In patients who underwent cerebral angiography (*N* = 6 vs 11).

Table 3. Surgical Intervention and In-hospital Neurologic Events According to Patient Groups

	Non-CMB Group (*N* = 34)	CMB Group (*N* = 40)	*P*
Surgical intervention			
Cardiac surgery, *n* (%)	24 (71)	25 (63)	0.464
EuroSCORE logistic %, median (IQR)	12 (5–31)	24 (16–37)	0.014
Operation during initial admission, *n* (%)	20 (59)	24 (60)	0.918
Early surgery (within 14 d), *n* (%)	13 (38)	17 (43)	0.710
Late surgery (after 14 d), *n* (%)	11 (32)	8 (20)	0.225
Days from IE diagnosis, median (IQR)*	11 (3–39)	8 (4–21)	0.645
Days from MRI, median (IQR)*	6 (3–19)	7 (3–38)	0.718
Operative procedures			
Aortic valve replacement, *n* (%)*	8 (35)	11 (46)	0.440
Mitral valve replacement, *n* (%)*	3 (13)	10 (42)	0.028
Mitral valve repair, *n* (%)*	12 (52)	9 (38)	0.312
Indications for surgery			
Heart failure unresponsive to OMT	6 (18)	6 (15)	0.758
Persistent infection	0 (0)	0 (0)	–
Repeat embolization or high embolic risk	17 (50)	18 (45)	0.668
Perivalvular extension	7 (21)	7 (18)	0.735
Residual valvular regurgitation	10 (29)	12 (30)	0.956
Postoperative neurologic event			
Neurologic event after operation, *n* (%)*	4 (17)	4 (16)	0.950
Cerebral infarction, *n* (%)*	1 (4)	1 (4)	0.977
Intracranial hemorrhage, *n* (%)*	3 (13)	3 (12)	0.957
Neurologic event with symptom, *n* (%)*	1 (4)	1 (4)	0.977

CMB, cerebral microbleeds; IE, infective endocarditis; IQR, interquartile range.
*In patients who underwent cardiac surgery (*n* = 49).

and non-CMB groups ($P = 0.218$ and $P = 0.345$, respectively; log-rank test; Fig. 3A and B). Multivariate Cox regression analysis adjusting age and operative risk (EuroSCORE) did not show that CMB was a significant risk factor for all-cause death and MACE (Table 4).

DISCUSSION

This study demonstrated the clinical features and outcomes of patients with active IE and CMB in association with cardiac surgery. Patients with CMB were older, took antiplatelet therapy more frequently, and showed a higher prevalence of

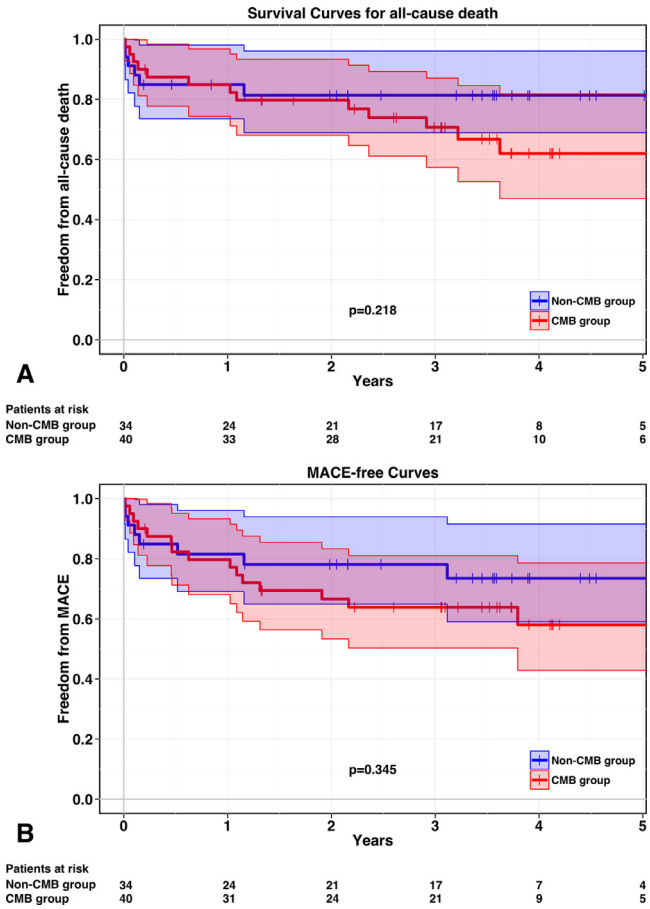

Figure 3. (A) Kaplan-Meier curves for freedom from all-cause death between patients with microbleeds and those without. (B) Kaplan-Meier curves for freedom from MACE between patients with microbleeds and those without. CMB, cerebral microbleeds; MACE, major adverse cardiac events.

Table 4. Multivariate Analysis

	Hazard Ratio (95% CI)	*P*
All-cause death		
Age	1.049 (0.996–1.105)	0.071
EuroSCORE (logistic)	1.026 (1.003–1.051)	0.029
CMB	0.797 (0.286–2.226)	0.665
MACE		
Age	1.011 (0.976–1.048)	0.542
EuroSCORE (logistic)	1.032 (1.011–1.055)	0.003
CMB	0.761 (0.292–1.983)	0.576

CMB, cerebral microbleeds; MACE, major adverse cardiac events.

staphylococcal infection and prosthetic valve endocarditis than those without CMB. However, the mid-term clinical outcomes of patients with CMB were comparable to those of patients without. Further large-scale prospective studies are warranted to elucidate the clinical importance of CMB in patients with IE.

Klein et al reported that CMB occurs frequently in patients with IE, and the prevalence of any CMB in IE patients was 57%.[18] In another study, CMB was observed in 58% of patients with IE.[19] The results of the current study agreed with the results of these previous studies with respect to the high prevalence of CMB in patients with active IE. Considering the high frequency of CMB in patients with active IE, the impact of CMB on clinical outcomes should be clarified during decision-making for the management of active IE.

Opinions about the association between hemorrhagic complications and CMB after thrombolytic therapy in ischemic stroke patients are conflicting.[20,21] Similarly, whether CMBs are associated with hemorrhagic complications after cardiac surgery in patients with acute IE is still being debated. Okazaki et al reported that the presence of more than 2 CMBs could predict the development of intracranial hemorrhage.[13] Hess et al reported that none of the IE patients who had preoperative CMB in their study experienced clinically significant cerebral complications after cardiac surgery.[10] Our findings are consistent with their results. In our study, postoperative neurologic deterioration was comparable between patients with and without CMB. Thus, our results indicated that CMB was not associated with worse clinical outcomes after cardiac surgery. In contrast, cerebral hemorrhage in patients with IE has been reported to be strongly associated with neurologic deterioration after cardiac surgery.[22] Therefore, CMB might be regarded as a different clinically entity from cerebral hemorrhage.

The potential mechanism of CMB formation is not well understood. Hypertensive microangiopathy and amyloid angiopathy are thought to be possible causes of CMB.[23] Miwa et al reported that serum inflammatory mediators such as high-sensitivity C-reactive protein, interleukin-6, and interleukin-16 are associated with CMB formation in stroke patients.[24] Vascular vulnerability and the inflammatory process may play an important role in the formation of CMB.[25] Considering these hypotheses, the systemic or regional inflammation process of inflammatory cytokines might contribute to CMB formation in patients with active IE. Although our data did not show that there was a significant relationship between inflammatory markers and CMB, more severe vascular inflammation due to a staphylococcal infection might facilitate the formation of CMB.

Study Limitations

Our study had several limitations. First, this study was a single-center retrospective study in a small number of patients. Considering the numbers of all-cause death in the 2 groups, the power calculation including an alpha level of 5%, a beta level of 20%, and a two-tail test revealed that sample size needs at least 105 to show the differences of the primary endpoint. Thus, the sample size of this study was not sufficient to detect statistical differences in primary endpoints between the 2 groups. However, multivariate analysis adjusting age and operative risk could not reveal that CMB is a significant risk factor for all-cause death and MACE. Therefore, considering confounding effects of older age and relatively higher operative risk in CMB group, CMB might not have a major impact on clinical outcomes in patients with IE. Second, an inclusion bias cannot be excluded, because only patients who could undergo

brain MRI were included in the analysis. Therefore, the clinical outcomes and prognosis may have been affected by the patient's clinical situation and severity of their conditions. Third, since not all of the study patients underwent cerebral angiography, small mycotic aneurysms could have been undiagnosed, which might affect clinical outcomes. However, MR angiography has been reported to be highly accurate in diagnosing cerebral aneurysms with the sensitivity of 82−96%.[26] Considering the relatively poor conditions of the study patients, noninvasive approach with MR angiography in detecting mycotic aneurysm might be acceptable. Finally, since the follow-up period was relatively short, we could not clarify the long-term clinical impact of CMB.

CONCLUSIONS

The mid-term outcomes of IE patients with CMB were comparable to those of patients without CMB. The postoperative outcomes of patients with CMB were especially equivalent to those without CMBs. Our results suggest that it might not have a major impact on clinical outcomes in patients with infective endocarditis. However, further prospective studies in a large study population with multicenter settings are necessary to clarify the relationship between CMB and neurologic deterioration after cardiac surgery.

SUPPLEMENTARY MATERIAL

The following is the supplementary data to this article:

Case 72-year-old male

Native mitral valve endocarditis
Iliopsoas muscle abscess
Splenic infarction

Fever and lower back pain
Positive blood culture (*Streptococcus anginosus*)
Asymptomatic cerebral complication
Large vegetation on the mitral valve

Video 1. A 72-year-old male presented with lower back pain. Contrast-enhanced computed tomography revealed splenic infarction and abscess in right iliopsoas muscle. Transthoracic echocardiography revealed large flail vegetation on the mitral valve. Serial sets of blood culture were positive for *Streptococcus anginosus*. Brain MRI was notable for acute ischemic lesion in the cerebellum, subarachnoid hemorrhage in the left hemisphere, and CMB in the right hemisphere. Invasive cerebral angiography showed no evidence of mycotic aneurysm formation. On the next day of infective endocarditis diagnosis, mitral valve repair was performed. His postoperative course was uneventful and he was discharged without any neurologic deficit.

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