

Case Reports & Case Series

Histopathological examination of an embolus in infective endocarditis: Case report and review of the literature[☆]

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ABSTRACT

A 62-year-old woman with history of chronic back pain and vertebral osteomyelitis presented with right hemiparesis and aphasia with last known well 10 h prior to presentation and a CT angiography demonstrating a left M1 occlusion. A thrombectomy was performed with resultant TICI-2b improving NIHSS from 28 pre-thrombectomy to 19 post-thrombectomy. Pathologic examination of the embolus demonstrated abundant bacterial colonies. While echocardiography was negative for valvular vegetations, fluoroscopy demonstrated abnormal mechanical valve motion. Blood cultures grew *Enterococcus faecalis* for which antibiotics were initiated. NIHSS continued to improve to a score of 12. There are rare cases in the literature that discuss diagnosis and initiation of antibiotics assisted by histopathologic examination of the embolus. Routine histopathologic evaluation of emboli may facilitate timely diagnosis and early antibiotic initiation in the setting of septic emboli.

1. Introduction

Infective endocarditis is an infection of the endocardial surface of the heart and occurs in 0.01–0.015% of the population [1,2]. The modified Duke criteria is well-established for the diagnosis of infective endocarditis [3]. One of the diagnostic criteria is echocardiographic visualization of valvular vegetations. Vegetations of endocarditis have the potential to dislodge from cardiac valves and cause cardioembolic stroke [4]. Risk factors include an immunocompromised state, intravenous drug user, and presence of prosthetic valve [5].

Cerebral ischemic events due to septic emboli have a rate of death or disability up to 73% [6]. These ischemic events occur in 10–20% of patients with infective endocarditis and in half of these patients, neurological findings are the only presenting symptom [7,8]. Cases presenting with a cardioembolic stroke have treatment modalities available to them including tissue plasminogen activator (tPA) and thrombectomy [9]. Current guidelines for the use of tPA in the setting of infective endocarditis are not well established; however, the guidelines discourage use in this setting [10,11]. Unfortunately, it is difficult to have foreknowledge of potential bacterial endocarditis in the acute stroke setting. If tPA is used in a patient with infective endocarditis, the

risk of intracranial hemorrhage reaches 20% [6]; therefore, the most recent literature explores thrombectomy as a treatment modality [12].

Although valvular vegetations are important for a diagnosis of infective endocarditis, they are only present in approximately half of patients who present with isolated neurological symptoms [13]. Proper and timely diagnosis is important because delayed administration of antibiotics worsens patient outcomes [4]. Complications of a delayed diagnosis include increased risk for intracranial hemorrhage and risk for recurrence of a cardioembolic stroke [13,14]. While routine histopathologic analysis of emboli after thrombectomy is not standard, this report raises the question as to whether it may alter treatment by facilitating a timely diagnosis.

2. Case presentation

We report a 62-year-old woman with past medical history of mechanical aortic valve on chronic anticoagulation with warfarin, recent cardiac arrest due to coronary artery disease with drug eluting stents (DES) placement, oxygen-dependent chronic obstructive pulmonary disease (COPD), lumbar spine osteomyelitis and *Enterococcus faecalis* bacteremia. The osteomyelitis and *Enterococcus faecalis* bacteremia

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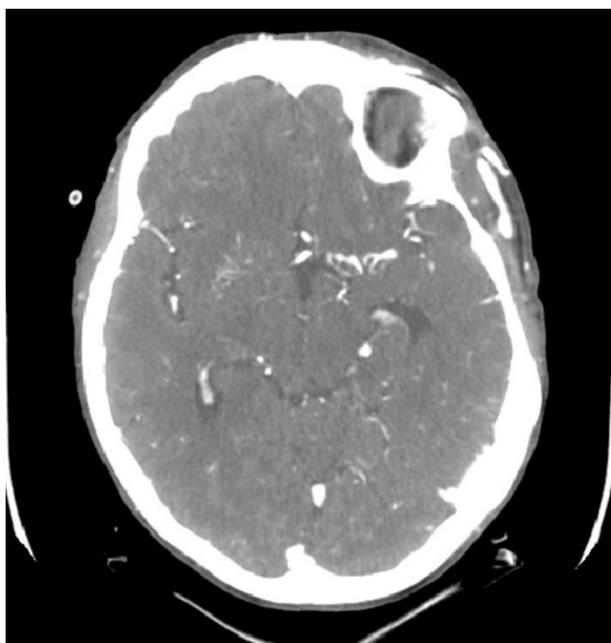


Fig. 1. CT angiography (CTA) of the head showing an occlusion at the left distal M1 segment of the middle cerebral artery.

were successfully treated with long course of antibiotics nine months before presentation. The patient was transferred to our comprehensive stroke center ten hours after onset of right hemiparesis and aphasia. The NIH stroke scale (NIHSS) and the Modified Rankin Scale (mRS) scores were 28 and 3, respectively. The patient was intubated due to rapid clinical decline. On admission, her INR was supratherapeutic at 4.1, her WBC was 6530 cells/mm², and her temperature was 35.4 °C. CT angiography of the head showed occlusion at the left distal M1 segment of the middle cerebral artery (MCA, Fig. 1) and a CT perfusion showed significant penumbra (Fig. 2).

Thrombectomy achieved a TICI-2b recanalization and residual left M3 opercular frontal with slow flow. Clinically, she demonstrated significant improvement of right hemiparesis but remained globally aphasic. She was extubated two days after the procedure. Routine workup for embolic stroke included a transthoracic echocardiogram (TTE), which did not show cardiac valvular vegetations. Histopathologic evaluation of the embolus, however, demonstrated that it contained abundant bacterial colonies, up to 50% of the total volume (Fig. 3). Blood cultures grew *Enterococcus faecalis* which, in conjunction with the pathology results, were strongly suggestive of infective endocarditis despite the negative TTE. Fluoroscopy of the mechanical aortic valve ultimately showed abnormal aortic valvular leaflet motion suggesting vegetations that were undetected by echocardiography. Transesophageal echocardiography (TEE) was unable to be obtained due to the patient's critical illness. She was started on ampicillin, ceftriaxone, and ciprofloxacin for bacteremia and septic emboli. Two days later, her NIHSS improved to 12.

The patient's hospital course was complicated by hypoxic respiratory failure secondary to COPD exacerbation, volume overload, aortic valve insufficiency, dysphagia and persistent aphasia. Due to the patient's several comorbidities along with new complicating issues, the grim prognosis with the potential need for repeat intubation, the family opted for palliative care. The patient died seven days after admission to our hospital.

3. Discussion

Infective endocarditis can result in cardiac valvular vegetations that can dislodge and embolize distal vessels including the cerebral vessels.

Neurological complications of infective endocarditis are frequent in critically ill patients (up to 55%) [12]. A cohort analyzed by Anderson et al. [15] noted that cerebral involvement had a high mortality rate with 35% of patients dying during the current admission and 52% of patients dying within one year of diagnosis and that the middle cerebral artery was most commonly affected.

The optimal therapy for acute ischemic stroke due to septic embolism is controversial [12]. Tissue plasminogen activator (tPA) is the standard of treatment for cardioembolic stroke. Current guidelines do not recommend the use of tPA in infective endocarditis and septic embolus due to the increased risk for intracranial hemorrhage (up to 20%) [16]. Possible mechanisms of hemorrhage include rupture of mycotic aneurysm and pyogenic or immune complex-mediated arteritis [12]. Also, due to the high fibrous hyaline content of the septic emboli, thrombolytic therapy is unlikely to degrade the embolus to a clinically significant extent [17]. New intracerebral hemorrhage in the setting of septic emboli increases the morbidity and mortality significantly. Due to the difficulty of determining the etiology prior to neurointervention, tPA has been inadvertently administered to patients with septic emboli [18–20].

Endovascular management of septic emboli is not readily articulated in AHA guidelines. Intracerebral hemorrhage from vessel fragility, presence of mycotic aneurysms, and reperfusion injury are concerns when considering endovascular management [17]. In addition to the therapeutic effect of mechanical thrombectomy, the retrieved clot can also establish the diagnosis of septic embolism. Hernandez-Fernandez et al. [21] reported 4 out of 65 cases in which histopathological and bacteriological studies with Gram staining of the extracted material guided the initiation of early antibiotic therapy even before the results of the blood cultures were available. Several other case reports suggest that thrombectomy is both safe and effective in the setting of septic cardiac emboli and leads to improved outcomes when compared to tPA due to the risk for intracerebral hemorrhage [22].

Performed as part of a routine stroke workup, an echocardiogram can diagnose cardiac valvular vegetations 53% of the time if the patient has neurological symptoms; however, the remaining 47% of cases require additional testing to determine the etiology that necessitates further investigation and prolongs diagnosis [13]. Furthermore, neurologic manifestation is the first presenting symptom in 47% of patients with infective endocarditis [13]. Delay in treatment initiation increases likelihood for a poor prognosis and can potentially result in additional sequelae such as recurrence of cerebral stroke [13,14]. Embolism reportedly occurs in 20–40% of infective endocarditis cases; however, this risk decreases to 9–21% after antibiotic treatment initiation [23], illustrating the importance of early detection in these cases. In patients with infective endocarditis who display a paucity of diagnostic signs or symptoms, histopathologic examination can potentially improve patient morbidity and mortality by providing the information needed to direct further confirmatory testing. The value of the pathology examination was exemplified in our case when the test meant to evaluate for valvular vegetations, the echocardiogram, was negative. The pathology result prompted additional evaluation of the aortic valve via fluoroscopy, resulting in confirmation of the suspected infective endocarditis. While the blood cultures ultimately supported the need for additional work up, a diagnostic delay was avoided while awaiting the results of the blood cultures.

A literature review using PubMed was conducted showing all available septic emboli with treatment modality and patient outcomes. We included patients into the literature review table who had appropriate data points for comparison (presentation, presentation NIHSS, neurointervention, echocardiogram results, clinical outcome, follow-up NIHSS, echocardiogram results). All of this data is presented in Table 1 and Table 2.

Table 1 presents 19 patients that received thrombectomy with or without tPA [12,17,18,21,22,24–32]. Average age was 49 ± 19 (mean ± standard deviation) years old. These patients had sufficient

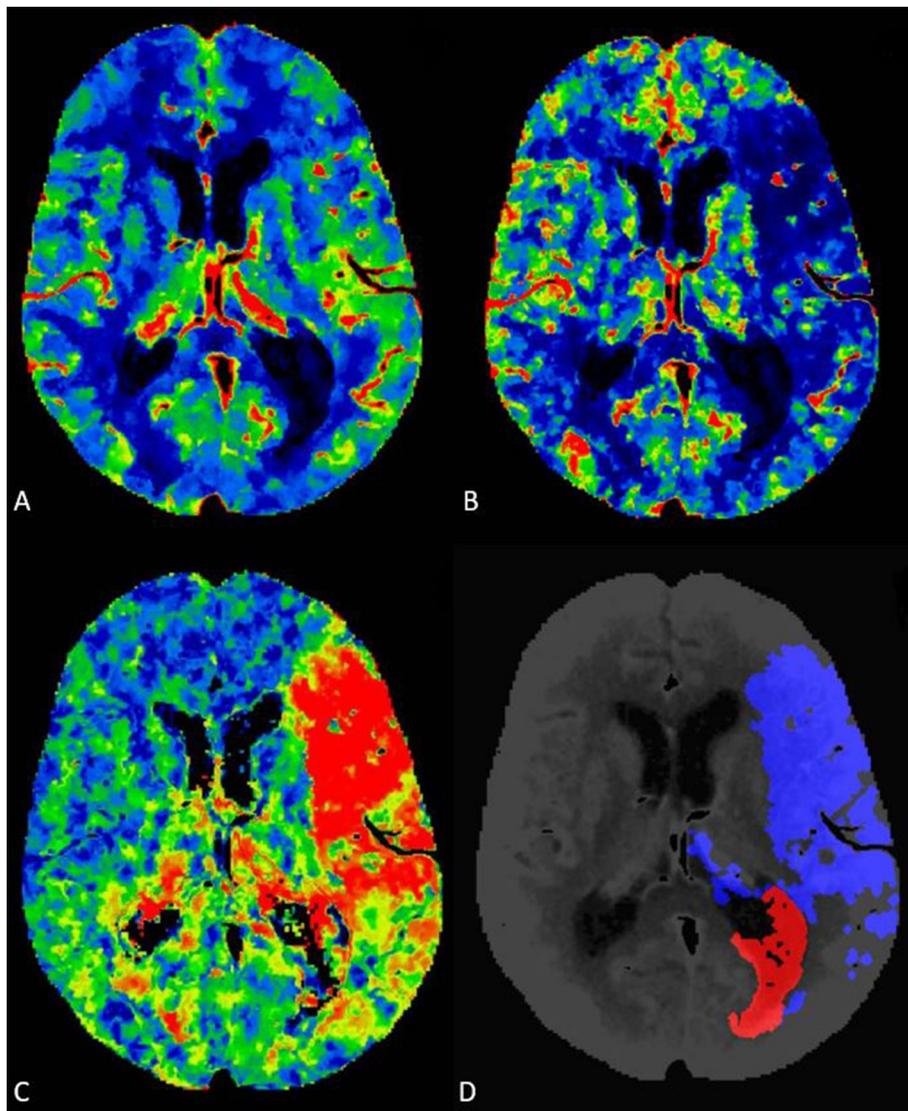


Fig. 2. CT perfusion showing (a) comparable blood volume bilaterally, (b) decreased blood flow in the left MCA distribution, (c) increased mean transit time in the left MCA distribution, and (d) a tissue classification scan. All of these findings suggest significant penumbra in the left MCA distribution.

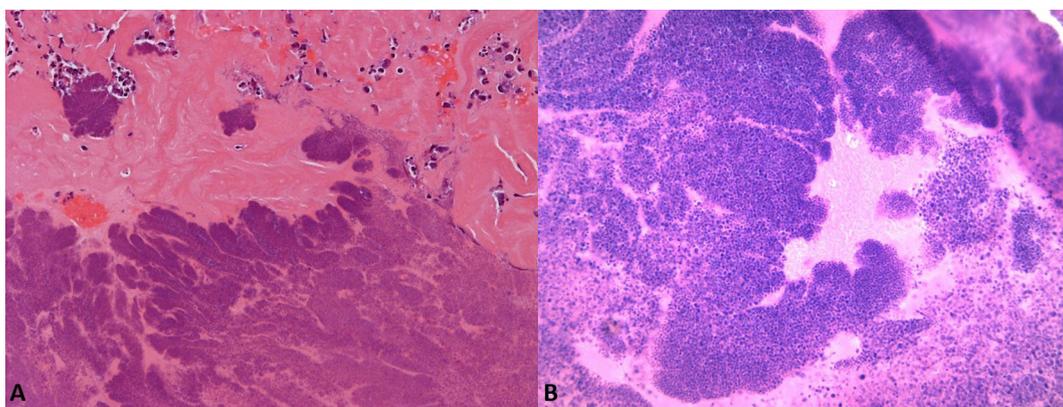


Fig. 3. Hematoxylin and eosin (H&E) stained slide demonstrating (A, 20 \times) embolus composed of large collections of purple bacteria along the bottom half with blood components (fibrin, red blood cells, and inflammatory cells) above, and (B, 100 \times) the collections of bacteria at higher magnification. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 1
Literature review of cases of septic emboli treated by thrombectomy.

Author	Age, gender	Presentation	NIHSS	Location	Intervention	Post-intervention Imaging	Clinical outcome	NIHSS	Valvular lesions present?
Bain et al. (2011)	24, F	Hemiparesis, speech difficulty	18	Left carotid terminus	IA tPA - > thrombectomy with Merci, stent with balloon angioplasty	TICI 3, no hemorrhage	Mild expressive aphasia	2	Not tested
Bhaskar et al. (2017)	36, M	Hemiparesis	16	M1	IV tPA - > thrombectomy (aspiration)	TICI 3, no hemorrhage	Mild hemiparesis	2	No
Bolognese et al. (2018)	42, M	Hemihypoesthesia, hand paresis, aphasia	3	M2	Thrombectomy - > IA tPA	TICI 2b, no hemorrhage	No deficits	0	Yes
Dababneh et al. (2012)	67, F	Hemiparesis, facial droop, dysarthria	NA	M1	Thrombectomy (Penumbra)	TICI 2/3, no hemorrhage	Gradual improvement	NA	Yes
Hernandez-Fernandez et al. (2017)	57, M	N/A	13	Large vessel occlusion	Thrombectomy (stent + aspiration)	TICI 2b	N/A	2	Yes
	66, M	N/A	15	Large vessel occlusion	Thrombectomy (stent + aspiration)	TICI 2b	N/A	8	No
Kan et al. (2012)	78, F	Hemiparesis, aphasia	16	M2	Thrombectomy (solitaire)	TICI 3, no hemorrhage	Improved hemiparesis	12	Yes
Kang et al. (2013)	39, F	Hemiparesis	18	M1	IA tPA, IA tirofiban - > Thrombectomy (solitaire)	TICI 2b, no hemorrhage	Mild hemiparesis, mild aphasia	3	Yes
Kim et al. (2014)	40, F	Hemiparesis, aphasic	15	M1	Thrombectomy (forced arterial suction thrombectomy)	TICI 3, no hemorrhage	Mild language deficits	3	Yes
Ladner et al. (2015)	40s, M	Hand paresis, facial droop, dysarthria	3	M1	Thrombectomy (Penumbra)	TICI 3, no hemorrhage	No neurologic deficits	0	Yes
Nishino et al. (2017)	72, M	Hemiparesis, aphasia	NA	M2	Thrombectomy (Codman & Shurtleff)	Reocclusion, no hemorrhage	Vasospasm, death	NA	Yes
Scharf et al. (2017)	NA	Hemiparesis, hemineglect, gaze preference, dysarthria	12	M1	Thrombectomy (Penumbra)	TICI 3, no hemorrhage	Mild facial palsy	1	Yes
	NA	NA	18	M2	Thrombectomy	TICI 3, no hemorrhage	NA	5	Yes
	NA	NA	19	M1	IV tPA - > Thrombectomy (aspiration)	TICI 2a, no hemorrhage	NA	17	Yes
Sukumaran et al. (2012)	33, M		14	M1	Thrombectomy	TICI 2b, no hemorrhage	Mild dysphasia	0	Yes
Sveinsson et al. (2016)	33, M	Hemiparesis, aphasia	14	M1	Thrombectomy (solitaire)	TICI 2b/3, no hemorrhage	Mild dysphasia	1	Yes
	67, M	Hemiparesis	13	M1	Thrombectomy (solitaire)	TICI 2b/3, asymptomatic hemorrhage	No neurologic deficits	0	Yes
Toeg et al. (2014)	39, F	Hemiplegia, aphasia	15	M2	Thrombectomy (solitaire)	TICI 2b/3, no hemorrhage	NA	4	Yes
	73, M	Hemiplegia, facial droop, dysarthria	20	Carotid terminus	IA tPA - > thrombectomy (stent retriever and aspiration catheter)	TICI 3, no hemorrhage	No neurologic deficits	0	Not tested

Table 2
Literature review of cases of septic emboli treated solely by tPA.

Author	Age, gender	Presentation	NIHSS	Location	Intervention	Post-intervention imaging	Clinical outcome	NIHSS	Valvular lesions present?
Bhuvu et al. (2009)	46, M	Hemiparesis and facial droop	15	M1	IV tPA	SAH, ICH	Death	NA	No
	65, F	Hemiparesis and speech difficulty	21	M1	IV tPA	SAH, ICH	Gradual improvement	NA	Yes
	61, M	Hemiparesis, global aphasia	17	M1	IV tPA	SAH, ICH	Gradual improvement	NA	Yes
Brownlee et al. (2014)	27, F	Hemiparesis, dysphasia	15	M2/3	IV tPA	No hemorrhage	Hand clumsiness	0	Yes
Junna et al. (2007)	56, M	Hemiparesis, dysphasia, homonymous hemianopia	15	M2	IV tPA	No hemorrhage	Mild hemiparesis, mild dysphasia	4	Yes
Ong et al. (2013)	68, M	Hemiparesis, facial droop, dysphasia	12	M2	IV tPA	SAH, ICH	Slight aphasia	1	Yes
Siccoli et al. (2013)	31, F	Hemiplegia, dysarthria, nystagmus	13	Basilar	IA tPA	No hemorrhage	Mild hemiparesis, mild aphasia, nystagmus, gait ataxia	5	Yes
Sontineni et al. (2010)	70, M	Hemiparesis, dysphasia	13	M1	IV tPA	No hemorrhage	Minimal aphasia	5	Yes
Tan et al. (2009)	12, F	Hemiparesis, aphasia	18	Carotid terminus	IA tPA	Complete recanalization, no hemorrhage	Mild hemiparesis, mild aphasia	5	Yes
Tversky et al. (2016)	57, M	Hemiparesis, aphasia	14	M1	IV tPA	IPH	NA	NA	Yes

recanalization (TICI-2b or greater) 89% (17/19) of the time. The embolus was located in the following locations: 2 carotid terminus, 10 M1, 5 M2, and 2 unspecified large vessel occlusions. Paresis was present in all of these cases due to location of the embolus except for the unspecified large vessel occlusions because there were no clinical presentations documented. The average NIHSS improvement for these cases is 10.7 ± 5.2 (mean \pm standard deviation) with one death due to vasospasm. Two patients did not have follow-up NIHSS scores. On follow-up examination 18 patients improved based on clinical outcome or NIHSS and 1 patient passed away. Valvular vegetations were present in 15 cases, absent in 2 cases, and not tested for in 2 cases. Only two of these cases published by Hernandez-Fernandez et al. [21] obtained histopathological analysis of the emboli before diagnosis of infective endocarditis.

Table 2 summarizes 10 patients who received tPA alone [10,16,19,20,23,33–35]. Average age was 49 ± 20 (mean \pm standard deviation) years old. The embolus was located in the following locations: 1 carotid terminus, 5 M1, 2 M2, 1 M2/M3, and 1 basilar. These patients had an average change in NIHSS of 11.0 ± 2.5 (mean \pm standard deviation) with one death due to intracerebral hemorrhage. On follow-up examination, 7 patients improved, 1 patient passed away, and 2 patients did not have follow-up data. There was only one case where vegetations were not seen.

Changes in the NIH Stroke Scores are 10.7 for thrombectomy (with or without tPA) and 11.0 for tPA alone ($p = 0.748$). New intracranial hemorrhage was detected in 50% (5/10) of patients who received IV tPA alone vs 8% (1/13) of patients who received thrombectomy alone ($p = 0.05$). The one case of hemorrhage within the thrombectomy group was asymptomatic. Patients receiving both tPA and thrombectomy had hemorrhage 0% (0/6) of the time. New intracerebral hemorrhage in the setting of septic emboli increases the morbidity and mortality significantly in the long-term follow-up period. It is difficult to know the etiology of the embolus before IV tPA administration so hemorrhage is unavoidable in these settings.

4. Conclusion

Our case presentation and review of the literature describes a clinical scenario in which acute ischemic stroke was caused by septic emboli. Histopathological analysis prompted further workup and treatment in our case and was suggested as beneficial in the literature. Routine histopathologic analysis of the retrieved embolus can assist in diagnostic workup and decrease the time to treatment. Prompt diagnosis expedites antibiotic initiation and has the potential to decrease the complications of infective endocarditis due to delayed treatment.

Conflicts of interest

The authors declare no conflict of interest pertaining to this article. Dr. Bohnstedt is a Consultant, Speaker, Site PI for Penumbra Consultant, Speaker, Site PI for Stryker Neurovascular Site PI and Speaker for Ceranovos Site PI and Speaker for Nico.

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