ScientificScholar®
Knowledge is power
Publisher of Scientific Journals

www.surgicalneurologyint.com

Surgical Neurology International

Editor-in-Chief: Nancy E. Epstein, MD, Professor of Clinical Neurosurgery, School of Medicine, State U. of NY at Stony Brook.

SNI: Neurovascular

Editor
Kazuhiro Hongo, MD.
Shinshu University, Matsumoto, Japan

SNI
Open Access

Case Report

Mechanical thrombectomy for cerebral embolism due to cardiac papillary fibroelastoma: A case report

Kentaro Izumi[1,2], Youhei Takeuchi[1,2], Naoya Iwabuchi[1,2], Masahiro Yoshida[1,2], Kuniyasu Niizuma[3,4,5], Hidenori Endo[3]

[1]Department of Neurosurgery, Osaki Citizen Hospital, Osaki, [2]Preemptive Medicine in the Community of the North Miyagi, Tohoku University Graduate School of Medicine, [3]Department of Neurosurgery, Tohoku University Graduate School of Medicine, [4]Department of Translational Neuroscience, Tohoku University Graduate School of Medicine, [5]Department of Neurosurgical Engineering, Graduate School of Biomedical Engineering, Tohoku University, Sendai, Miyagi, Japan.

E-mail: Kentaro Izumi - kentaro.izumi.t8@gmail.com; Youhei Takeuchi - you.takeuchi@olive.plala.or.jp; Naoya Iwabuchi - d06sm013@yahoo.co.jp; *Masahiro Yoshida - myoship1964@gmail.com; Kuniyasu Niizuma - kniizuma@tohoku.ac.jp; Hidenori Endo - h-endo@tohoku.ac.jp

***Corresponding author:**
Masahiro Yoshida,
Department of Neurosurgery,
Osaki Citizen Hospital, Osaki,
Japan.

myoship1964@gmail.com

Received: 21 January 2025
Accepted: 19 March 2025
Published: 18 April 2025

DOI
10.25259/SNI_68_2025

Quick Response Code:

ABSTRACT

Background: Papillary fibroelastoma (PFE) and myxoma are relatively common types of benign cardiac tumors. PFE and myxoma can be associated with fatal embolic events. However, PFE is not widely recognized within the field of cerebrovascular diseases.

Case Description: A 54-year-old male presented with a sudden onset of left hemiparesis. Three-dimensional computed tomography (CT) angiography revealed incomplete occlusion of the right middle cerebral artery. Thrombolytic therapy with recombinant tissue-type plasminogen activator was performed, followed by mechanical thrombectomy. Reperfusion was achieved within 199 minutes, resulting in thrombolysis in cerebral infarction grade 2b. The retrieved emboli appeared as a white gelatinous substance, which was diagnosed as PFE by histopathological examination. Transesophageal echocardiography and cardiac CT identified a 6-mm mobile mass in the left atrium. PFE in the left atrium was considered to be the source of the embolism and tumor resection was performed on day 18. Histopathological findings of the resected tumor were identical to those of the emboli. The patient was transferred to a rehabilitation facility on day 36, with a modified Rankin Scale score of 2.

Conclusion: PFE and myxoma share many clinical features, but PFE tends to be smaller, so detection is more challenging and has likely resulted in under-recognition. PFE and myxoma can be associated with fatal embolic events. Resection is recommended for left-sided, mobile, symptomatic tumors larger than 10 mm. The differential diagnosis of embolus retrieved through mechanical thrombectomy should consider both myxoma and PFE and persistent efforts should be made to detect the embolic origin.

Keywords: Cardiac myxoma, Cardiac papillary fibroelastoma, Cerebral tumor embolism, Mechanical thrombectomy

INTRODUCTION

Papillary fibroelastoma (PFE) and myxoma are relatively common types of benign cardiac tumors.[12,13,15] Both PFE and myxoma can cause severe left-sided embolic events, including ischemic stroke and sudden death. However, PFE is more challenging to detect as the tumor is smaller than myxoma.[23,34] In addition, PFE has not been widely recognized within the field of cerebrovascular diseases because cardiac tumors are rare[12] and infrequently encountered, so they may have been previously overlooked. We present a case of PFE that manifested as cerebral embolism and was treated with mechanical thrombectomy.

CASE REPORT

Patient information

A 54-year-old male delivery worker employed by a transportation company experienced a sudden onset of left hemiparesis while at work. He presented to our emergency department 101 min after symptom onset.

Clinical findings

On arrival, he exhibited severe left hemiparesis, dysarthria, and left hemispatial neglect, with a National Institutes of Health Stroke Scale score of 11. He had no prior medical history. Noncontrast computed tomography (CT) revealed early ischemic changes in the right frontal operculum, temporal operculum, and insular cortex, with an Alberta Stroke Program Early CT Score of 7 [Figure 1a]. The hyperdense vessel sign was absent in the major cerebral arteries. Three-dimensional CT angiography demonstrated poor visualization of the right middle cerebral artery (MCA) without evidence of cervical carotid stenosis [Figure 1b]. Concurrent contrast-enhanced CT of the chest revealed no clearly visualized intracardiac thrombi or cardiac tumors [Figure 1c], and electrocardiography (ECG) showed sinus rhythm.

Diagnosis assessment

The etiology of the embolism could not be determined, but embolic occlusion of the right MCA was suspected.

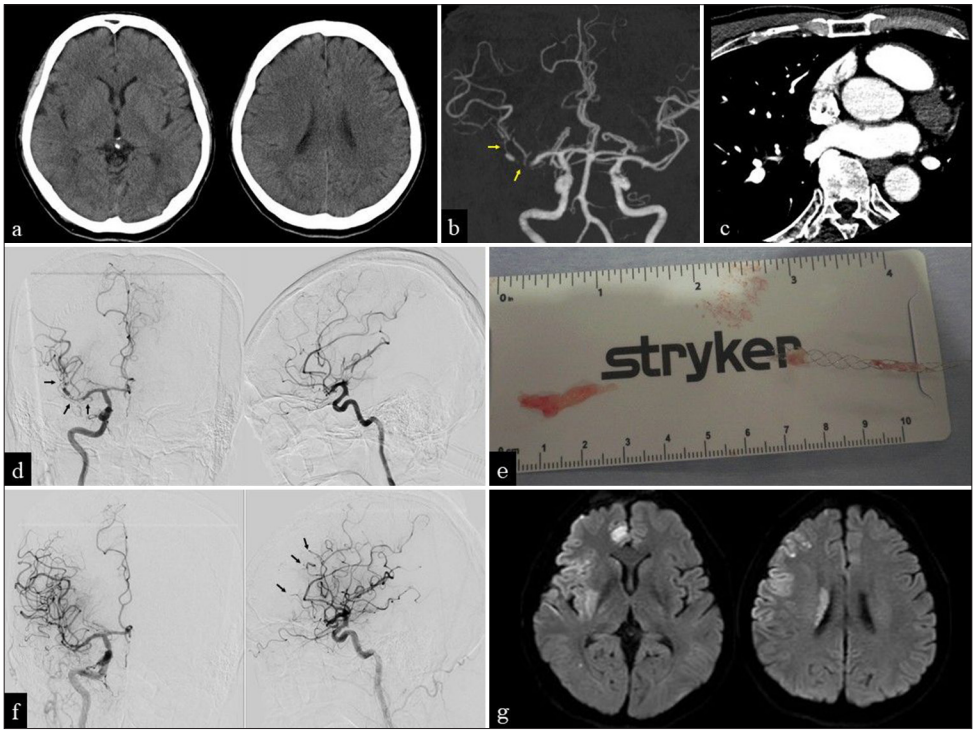

Figure 1: (a-d) Prethrombectomy images. Noncontrast computed tomography (CT) reveals early ischemic changes in the right frontal operculum, temporal operculum, and insular cortex (a). Three-dimensional CT angiography shows contrast defects at two sites: the right distal M1 segment and the distal M2 segment of the lower trunk, indicated by arrows in (b). Contrast-enhanced CT of the chest reveals no clearly visualized intracardiac thrombi or cardiac tumors (c). Cerebral angiography (left: anteroposterior view; right: lateral view) shows poor contrast at several sites: The distal M1 and the distal M2 segments of the lower trunk of the right middle cerebral artery (MCA), and the right anterior temporal artery, indicated by arrows in (d). (e) Two emboli were retrieved with a stent retriever, corresponding to the contrast defects of MCA. One of them has broken away from the tip of the stent retriever. They appear white and gelatinous. (f and g) Postthrombectomy images. Cerebral angiography (left: anteroposterior view; right: lateral view) shows distal embolization in the frontopolar artery, the posterior parietal artery, and angular artery (indicated by arrows), with reperfusion achieving thrombolysis in cerebral infarction grade 2b (f). Diffusion-weighted magnetic resonance imaging shows high-signal intensity areas in the right frontal operculum, temporal operculum, insular cortex, medial frontal gyrus, cingulate gyrus, putamen, and corona radiata (g).

Therapeutic intervention

Recombinant tissue-type plasminogen activator was administered intravenously at a dose of 0.6 mg/kg, followed by mechanical thrombectomy. Cerebral angiography identified two sites with contrast-filling defects in the distal M1 and the distal M2 segments of the lower trunk of the right MCA. The right anterior temporal artery originating from the M1 segment was also poorly visualized [Figure 1d]. Antegrade flow in the lower trunk was preserved, but delayed perfusion indicated partial occlusion. A combined thrombectomy was performed using a stent retriever and aspiration catheter. A Traxcess 14 microguidewire (Terumo, Tokyo, Japan) was used coaxially with a Trevo Trak 21 microcatheter (Stryker, Kalamazoo, MI, USA) to access the distal M2 segment of the lower trunk. A Trevo NXT ProVue 4 × 41 mm stent retriever (Stryker) was deployed from the distal M2 segment of the lower trunk to the distal M1 segment. An AXS Catalyst 6 aspiration catheter (Stryker) was advanced to the stent to compress the emboli between the devices. The stent retriever and aspiration catheter were retrieved following the standard procedure. With the guiding catheter's balloon inflated to occlude the internal carotid artery, both devices were slowly withdrawn together into the guiding catheter. Consequently, two emboli corresponding to the MCA filling defects were removed by the stent retriever [Figure 1e]. During the procedure, distal embolization occurred in the frontopolar artery, which is a cortical branch of the right anterior cerebral artery, as well as in the posterior parietal artery and the angular artery, both of which are branches of the right MCA. The distribution of these distal emboli suggested that the embolus fragmented during retrieval and migrated to distal vessels, including previously unaffected territory. These distal emboli were unrecoverable, so the procedure was concluded. Recanalization was achieved with thrombolysis in cerebral infarction grade 2b, 199 min after symptom onset [Figure 1f]. Postprocedure CT revealed no hemorrhagic complications. Diffusion-weighted magnetic resonance imaging on the following day showed high signal intensity in the right frontal operculum, temporal operculum, insular cortex, medial frontal gyrus, cingulate gyrus, putamen, and corona radiata [Figure 1g]. After treatment, the patient's symptoms gradually improved; however, mild hemiparesis in the left upper limb remained. This residual neurological deficit was suspected to be attributable to an infarction in the putamen or corona radiata, likely caused by distal embolization.

Follow-up: Posttreatment evaluation of the embolic source was conducted together with rehabilitation. The retrieved emboli appeared as a white gelatinous substance atypical of conventional thrombi [Figure 1e]. The gross findings initially suggested the possibility of a cardiac myxoma; however, the actual diagnosis differed from this initial assumption. Histopathological examination of the emboli revealed papillary structures [Figure 2a]. Each papilla contains a poorly vascularized, eosinophilic myxomatous stroma, which contains collagen fibers. A single layer of endothelial cells lines the surface of the papilla. The presence of myxoma cells characteristic of myxomas was not observed [Figure 2a and b]. In conclusion, the emboli were diagnosed as PFE, although the elastic fiber characteristics were not evident. Transthoracic echocardiography did not reveal intracardiac lesions. In contrast to the prethrombectomy chest CT, which did not clearly visualize the cardiac lesion, ECG-synchronized CT identified a 6-mm mass in the left atrium [Figure 3a], and transesophageal echocardiography confirmed it as a mobile lesion [Figure 3b]. Based on these findings, the left atrial PFE was considered to be the embolic source. On hospital day 18, the patient underwent open thoracotomy for tumor resection. A gelatinous mass was identified on the superior wall of the left atrium [Figure 3c] and excised together with the surrounding tissue. Histopathological examination of the resected mass revealed findings identical to those of the emboli [Figure 4a and b]. The postoperative course was uneventful. The patient exhibited residual neurological deficits, including fine motor coordination impairments and sensory impairments in the left upper limb, but was ambulatory and regained independence in daily activities within the hospital. On hospital day 36, the patient was transferred to a rehabilitation facility with a modified Rankin Scale score of 2.

DISCUSSION

Cardiogenic or atherosclerotic thrombi cause most cases of cerebral embolism.[11,21] However, the underlying cause is rarely nonthrombotic emboli. Nonthrombotic emboli may consist of fat, air, calcified fragments, bacterial clumps, and tumor tissue.[11,21] Cerebral tumor embolism is a rare pathological condition, accounting for approximately 0.5% of all ischemic strokes, in which the tumor tissue acts as an embolus, leading to obstruction of the cerebral arteries.[9] The most common cause of cerebral tumor embolism is cardiac

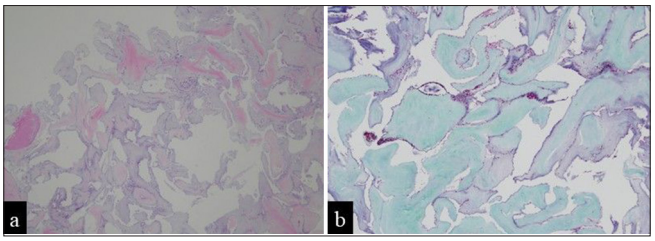

Figure 2: Histopathological examination of the emboli (a: Hematoxylin and Eosin staining, 2x, b: Elastica and Masson staining, 4x). (a) The lesion has a papillary structure. Each papilla contains a poorly vascularized, eosinophilic myxomatous stroma. A single layer of endothelial cells lines the surface. Myxoma cells were not observed. (b) Collagen fibers are observed. The elastic fibers are not evident.

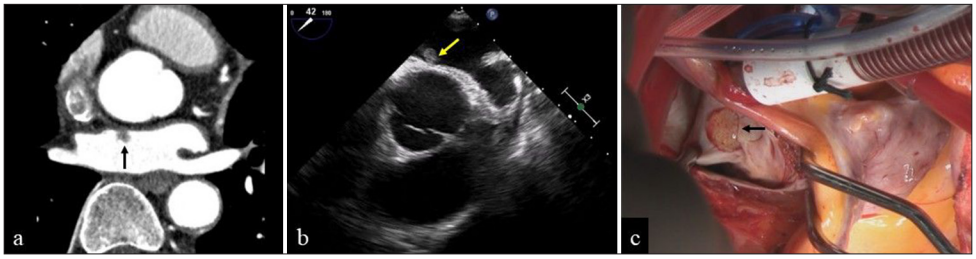

Figure 3: (a) Electrocardiography-synchronized computed tomography shows a 6-mm mass within the left atrium (indicated by an arrow). (b) Transesophageal echocardiography shows mobility of the mass in the left atrium (indicated by an arrow). (c) Intraoperative findings during open thoracotomy for tumor resection show a gelatinous mass in the superior wall of the left atrium (indicated by an arrow).

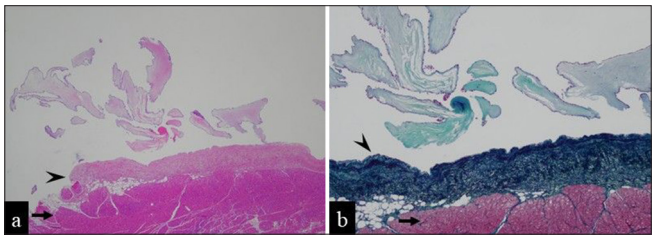

Figure 4: Histopathological examination of the resected tumor (a: Hematoxylin and Eosin staining, 2x, b: Elastica and Masson staining 4x). The lesion was excised along with the myocardium (indicated by arrows) and the endocardium (indicated by arrowheads). The histological features of the tumor are consistent with those of the emboli.

tumors, such as myxoma, which are responsible for about 50% of cases of cerebral tumor embolism.[9,33] The second most common source is pulmonary tumors, which can lead to cerebral embolism through invasion of the pulmonary veins or the left heart system.[24,33] These epidemiological findings have been confirmed since the increased use of thrombectomy procedures. Historically, the diagnosis of cerebral tumor embolism, excluding autopsy cases, lacked histopathological confirmation of the identity between the embolus and its source. The clinical diagnosis of tumor embolism was made by screening for potential embolic sources and considering the tumor as the most likely cause. Recently, the widespread adoption of thrombectomy has enabled the retrieval of embolus, thus allowing for histopathological confirmation. A more definitive diagnosis is now possible by demonstrating the histopathological identity between the retrieved embolus and the suspected tumor tissue. Consequently, the number of cases of cerebral tumor embolism caused by various tumors has increased.[2-4,10,17,22,24,26,32] This development emphasizes the dual role of thrombectomy as both a therapeutic intervention and a diagnostic tool for identifying rare etiologies of ischemic stroke.[2-4,10,17,22,24,26,32]

PFE is a benign cardiac tumor first described by Cheitlin *et al.* in 1975.[5] PFE and cardiac myxoma are commonly associated with cerebral tumor embolism.[14] PFE is a rare cardiac tumor but is the third most common after myxoma and lipoma, with a reported incidence of approximately 0.002% in autopsy studies.[1] However, the detection rate of PFE has risen with advances in echocardiographic imaging, increased utilization of cardiac ultrasonography, and growing awareness of the condition. PFE has possibly surpassed myxoma as the most common benign cardiac tumor.[31] PFE is generally considered benign, but its true neoplastic nature remains uncertain. The etiology of PFE is not well understood, and its macroscopic, microscopic, and molecular heterogeneity has led to hypotheses that these lesions may represent tumors, hamartomas, organized thrombi, or abnormal endocardial responses to trauma.[18] Histopathologically, PFE has a characteristic papillary structure. Each papilla consists of a poorly vascularized, eosinophilic myxomatous stroma with dense cores of elastic and collagen fibers. The surface of each papilla is lined by a single layer of endothelial cells that transition into the endocardium at the lesion base. Macroscopically, PFE typically appears as gelatinous, sea anemone-like structures with a lobulated morphology and is frequently pedunculated, usually with a short stalk forming the attachment to the endocardial surface.[8,18,35]

Cardiac tumors, exemplified by myxomas, are widely recognized as causes of cerebral embolism. However, the increasing detection rates of cardiac tumors indicate that PFE is also a significant cause of cerebral tumor embolism. A study reviewing 37 cases of cerebral tumor embolism treated with thrombectomy reported that 17 cases (45%) were of cardiac origin, including 11 myxomas (29.7%) and 6 PFEs (16.2%).[33] The similarities and differences between PFE and myxoma are summarized in Table 1. Approximately 80% of PFEs originate from the valvular apparatus or annulus, predominantly in the heart valves of the left side. Among these, 44% arise from the aortic valve, followed by 35% from the mitral valve. In contrast, myxoma typically originates from within the cavities, with 75% occurring in the left atrium and 15–20% in the right atrium. PFE tends to be smaller than myxoma,[15] with an average size of 9 mm[30] compared to 50–60 mm for myxoma.[20] Consequently, PFE is asymptomatic in about 60% of cases.[23] Both PFE and

Table 1: Comparison of the characteristics of PFE and myxoma.

	PFE	Myxoma
Common sites	Cardiac valve, especially the mitral valve and aortic valve[15]	Cardiac cavity, especially the left atrium[25]
Average size	9 mm[30]	50–60 mm[20]
Macroscopic findings	Pedunculated and sea-anemone-like structures with lobulated morphology[8,18,35]	Pedunculated[25]
Pathological findings	Papillary structure lined by a single layer of endothelial cells[8,18,35] Myxomatous stroma with deposits of acid mucopolysaccharides[8,18,35] Dense cores of elastic and collagen fibers[8,18,35] Poorly vascularized tissue	Myxoma cells appear stellate or fusiform[7] Myxomatous stroma with deposits of acid mucopolysaccharides[7,25] Mesenchymal tissue with abundant vascularity[7]
Pathophysiology	Asymptomatic (60%), TIA/CI (19%), angina pectoris (8%), MI (4%), heart failure (4%), sudden death (3%)[23]	Heart failure/sudden death (67%), systemic inflammatory response due to IL-6 (34%), TIA/CI (20%), other systemic embolism (9%)[25]
Treatment	Surgical intervention: symptomatic, mobile, left-sided, or ≥10 mm lesions[16] Anticoagulation therapy: other lesions[12]	Surgical intervention[25]

PFE: Papillary fibroelastoma, TIA: Transient ischemic attack, CI: Cerebral infarction, MI: Myocardial infarction, IL-6: Interleukin-6

myxoma are classified as benign tumors but are associated with severe embolic complications, including ischemic stroke and coronary ischemia.[23] Embolic events are often caused by fragmentation of the tumor itself or by fibrin thrombi forming on the tumor surface.[23] Myxoma carries the risk of fatal embolic events, including sudden death, thus requiring urgent surgical excision as the standard treatment.[25] PFE carries similar embolic risks, but their management is less clear due to the frequent detection of asymptomatic cases.[16] Surgical intervention is generally recommended for symptomatic, mobile, left-sided, or ≥10 mm lesions.[16] Careful observation may be considered for asymptomatic cases or if cardiac surgery is high risk.[16] Anticoagulation therapy has been advocated to prevent embolism from microthrombi, but the evidence supporting this approach remains limited.[16] Myxoma carries the risk of recurrence, so the standard surgical approach is wide excision, including the surrounding tissues.[6,25] In contrast, the postoperative recurrence rate of PFE is reported to be 0–1.6%, so the indications for extended resection remain unclear.[19,30,31]

An important point to note in this case is distal embolization. Although distal embolization is common as a technical complication of thrombectomy, the distal embolization in this case may be attributed to the nature of the embolus. Distal embolization typically occurs due to thrombus fragmentation and embolic migration during retrieval.[28] This was also observed in our case. Thrombectomy was performed using a combined technique with a stent retriever and an aspiration catheter, which resulted in distal embolization. This may be related to the embolus being composed of tumor tissue. Tumor emboli are soft and fragile.[3,24,26] Therefore, retrieving with a stent retriever is more likely to fragment the soft embolus, increasing the risk of distal embolization.[24,26] In contrast, retrieving with an aspiration catheter is less likely to fragment the embolus and may also shorten the procedure time, potentially making it more suitable for retrieving tumor emboli.[24,26] In our case, preprocedural evaluation indicated a low likelihood of tumor embolism. However, if tumor embolism had been considered highly likely based on the preprocedural assessment, a more careful selection of the thrombectomy technique would have been warranted.

In the present case, the macroscopic findings of the retrieved emboli raised the suspicion of a cardiac tumor. Subsequent imaging identified a cardiac lesion, which was surgically resected as the embolic source. Pathological examination confirmed the histopathological identity between the retrieved emboli and the resected tumor tissue, establishing the diagnosis of cerebral tumor embolism caused by cardiac PFE. Only one previous similar case of PFE manifesting as cerebral tumor embolism identified the embolic source by imaging, and surgical resection was performed.[14] In contrast, scattered reports have suggested PFE as the embolic source based on pathological findings, but the embolic source could not be identified by imaging, so clinical observation was continued without intervention.[27,29,32] PFE may have been underdiagnosed in the past because of its small size and limited recognition within the field of cerebrovascular diseases. In the present case, the lesion was not detected by both contrast-enhanced CT of the chest and transthoracic echocardiography; however, it was successfully identified using ECG-synchronized CT and transesophageal

echocardiography. PFE, like myxoma, has the potential to cause fatal embolic events despite their small size. Therefore, persistent efforts to detect these lesions are essential.

CONCLUSION

The present case of PFE manifesting as cerebral embolism was treated with mechanical thrombectomy. The differential diagnosis based on the retrieved embolus should consider not only myxoma but also PFE. PFE is smaller and more challenging to detect compared to myxoma but carries a similar risk of fatal embolic events. Therefore, persistent efforts to detect PFE are essential.

Ethical approval: Institutional Review Board approval is not required.
Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent.
Financial support and sponsorship: Nil.
Conflicts of interest: There are no conflicts of interest.
Use of artificial intelligence (AI)-assisted technology for manuscript preparation: The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

REFERENCES

1. Arai S, Tanaka D, Sakuma M, Tamamura T, Ishihara R, Sato Y, *et al.* Papillary fibroelastoma of the left ventricular outflow tract; report of a case. Kyobu Geka 2018;71:697-700.
2. Araki S, Maekawa K, Kobayashi K, Sano T, Yabana T, Shibata M, *et al.* Tumor embolism through right-to-left shunt due to venous invasion of esophageal carcinoma. J Stroke Cerebrovasc Dis 2020;29:105352.
3. Baek SH, Park S, Lee NJ, Kang Y, Cho KH. Effective mechanical thrombectomy in a patient with hyperacute ischemic stroke associated with cardiac myxoma. J Stroke Cerebrovasc Dis 2014;23:e417-9.
4. Bhatia S, Ku A, Pu C, Wright DG, Tayal AH. Endovascular mechanical retrieval of a terminal internal carotid artery breast tumor embolus. J Neurosurg 2010;112:572-4.
5. Cheitlin MD, McAllister HA, De Castro CM. Myocardial infarction without atherosclerosis. JAMA 1975;231:951-9.
6. Děrgel M, Gofus J, Smolák P, Stejskal V, Hanke I, Matějka J, *et al.* Surgical treatment of primary cardiac tumors: 20-year single center experience. Kardiochir Torakochir Pol 2022;19:36-40.
7. Di Vito A, Mignogna C, Donato G. The mysterious pathways of cardiac myxomas: A review of histogenesis, pathogenesis and pathology. Histopathology 2015;66:321-32.
8. Fishbein MC, Ferrans VJ, Roberts WC. Endocardial papillary elastofibromas. Histologic, histochemical, and electron microscopical findings. Arch Pathol 1975;99:335-41.
9. Fujiwara S, Fukumoto S, Watanabe M, Kusakabe K, Aso K, Shinohara T, *et al.* A case of left middle cerebral artery occlusion diagnosed as malignant lung tumor embolus by mechanical thrombectomy. Jpn J Stroke 2022;44:59-64.
10. Fujiwara Y, Hayashi K, Shibata Y, Furuta T, Yamasaki T, Yamamoto K, *et al.* Cerebral tumor embolism from thyroid cancer treated by mechanical thrombectomy: Illustrative case. J Neurosurg Case Lessons 2023;5:CASE22293.
11. Gomyo M, Tsuchiya K. Cerebral infarctions due to a special embolus. Clin Imagiol 2022;38:321-9.
12. Gowda RM, Khan IA, Nair CK, Mehta NJ, Vasavada BC, Sacchi TJ. Cardiac papillary fibroelastoma: A comprehensive analysis of 725 cases. Am Heart J 2003;146:404-10.
13. Hoffmeier A, Sindermann JR, Scheld HH, Martens S. Cardiac tumors--diagnosis and surgical treatment. Dtsch Arztebl Int 2014;111:205-11.
14. Itrat A, George P, Khawaja Z, Min D, Donohue M, Wisco D, *et al.* Pathological evidence of cardiac papillary fibroelastoma in a retrieved intracranial embolus. Can J Neurol Sci 2015;42:66-8.
15. Iwata Y, Nozawa Y, Sato S, Sakasai T, Katayama H, Sato M, *et al.* A case of papillary fibroelastoma originating on interventricular septum. Jpn J Med Ultrasound Technol 2016;41:174-81.
16. Kamamura M, Tanaka H, Suzuki H, Suzuki Y, Shiojiri T. A case of recurrent multiple cardioembolic stroke due to papillary fibroelastoma. Jpn J Stroke 2021;43:524-8.
17. Kim CS, Jung HR, Cho KH, Chang HW, Sohn SI, Choi TH, *et al.* Forced-suction thrombectomy of an arterial tumor embolism due to metastatic melanoma. Arch Neurol 2012;69:272-3.
18. Kurup AN, Tazelaar HD, Edwards WD, Burke AP, Virmani R, Klarich KW, *et al.* Iatrogenic cardiac papillary fibroelastoma: A study of 12 cases (1990 to 2000). Hum Pathol 2002;33:1165-9.
19. Maeda T, Sakurada T, Muraki S, Nakashima S, Uchiyama H, Sasaki J, *et al.* Papillary fibroelastoma arising from the left atrial wall: Report of a case. Kyobu Geka 2021;74:449-52.
20. McAllister HA Jr., Hall RJ, Cooley DA. Tumors of the heart and pericardium. Curr Probl Cardiol 1999;24:57-116.
21. Nakajo M, Hasegawa T, Nakano T, Kamimura K, Yoshiura T. Rare arterial embolisms. Jpn J Imaging Diagn 2023;43:1365-70.
22. Nukata R, Ikeda H, Akaike N, Fujiwara T, Yamashita H, Uezato M, *et al.* White embolus-induced basilar artery occlusion due to pulmonary vein invasion of a metastasis of a malignant melanoma. Intern Med 2023;62:2889-93.
23. Ohya Y, Fujimoto S, Kanazawa M, Tagawa N, Osaki M, Kitazono T. A case of cardioembolic stroke due to intracardiac papillary fibroelastoma evaluated by using transesophageal echocardiography. Rinsho Shinkeigaku 2017;57:9-13.
24. Oyama T, Asai T, Miyazawa T, Yokoyama K, Kogure Y, Torii A, *et al.* A case of cerebral tumor embolism from extracardiac lung cancer treated by mechanical thrombectomy. NMC Case Rep J 2020;7:101-5.
25. Pinede L, Duhaut P, Loire R. Clinical presentation of left atrial cardiac myxoma. A series of 112 consecutive cases. Medicine (Baltimore) 2001;80:159-72.
26. Pop R, Mihoc D, Manisor M, Richter JS, Lindner V, Janssen-Langenstein R, *et al.* Republished: Mechanical thrombectomy for repeated cerebral tumor embolism from a thoracic sarcomatoid carcinoma. J Neurointerv Surg 2018;10:e26.
27. Salam KA, Rafeeque M, Hashim H, Mampilly N, Noone ML. Histology of thrombectomy specimen reveals cardiac tumor

embolus in cryptogenic young stroke. J Stroke Cerebrovasc Dis 2018;27:e70-2.
28. Saver JL, Chapot R, Agid R, Hassan A, Jadhav AP, Liebeskind DS, *et al.* Thrombectomy for distal, medium vessel occlusions: A consensus statement on present knowledge and promising directions. Stroke 2020;51:2872-84.
29. Semerano A, Saliou G, Sanvito F, Genchi A, Gullotta GS, Michel P, *et al.* Fishing an anemone in the brain: Embolized cardiac fibroelastoma revealed after stroke thrombectomy. Eur Heart J 2021;42:4094-95.
30. Sun JP, Asher CR, Yang XS, Cheng GG, Scalia GM, Massed AG, *et al.* Clinical and echocardiographic characteristics of papillary fibroelastomas: A retrospective and prospective study in 162 patients. Circulation 2001;103:2687-93.
31. Tamin SS, Maleszewski JJ, Scott CG, Khan SK, Edwards WD, Bruce CJ, *et al.* Prognostic and bioepidemiologic implications of papillary fibroelastomas. J Am Coll Cardiol 2015;65:2420-9.
32. Tejada J, Galiana A, Balboa O, Clavera B, Redondo-Robles L, Alonso N, *et al.* Mechanical endovascular procedure for the treatment of acute ischemic stroke caused by total detachment of a papillary fibroelastoma. J Neurointerv Surg 2014;6:e37.
33. Toruno M, Al-Janabi O, Karaman I, Ghozy S, Senol YC, Kobeissi H, *et al.* Mechanical thrombectomy for the treatment of large vessel occlusion due to cancer-related cerebral embolism: A systematic review. Interv Neuroradiol 2024:15910199241230356.
34. Watanabe T, Maeda T, Inoue S. Papillary fibroelastoma on the tricuspid valve: Report of a case. Kyobu Geka 2016;69:131-3.
35. Zoltowska DM, Sadic E, Becoats K, Ghetiya S, Ali AA, Sattiraju S, *et al.* Cardiac papillary fibroelastoma. J Geriatr Cardiol 2021;18:346-51.

How to cite this article: Izumi K, Takeuchi Y, Iwabuchi N, Yoshida M, Niizuma K, Endo H. Mechanical thrombectomy for cerebral embolism due to cardiac papillary fibroelastoma: A case report. Surg Neurol Int. 2025;16:141. doi: 10.25259/SNI_68_2025