 doi: 10.2176/jns-nmc.2024-0004

*NMC Case Report Journal 11, 145-150, 2024*

# *A Case of Pontine Infarction due to Subclavian Steal Phenomenon Enhanced by an Arteriovenous Shunt for Hemodialysis*

Motohide TAKAHARA,[1] Tomoaki MURAKAMI,[1] Shingo TOYOTA,[1] Shuki OKUHARA,[1] Kazuhiro TOUHARA,[1] Yuhei HOSHIKUMA,[1] Takamune ACHIHA,[1] Shuhei YAMADA,[1] Maki KOBAYASHI,[1] and Haruhiko KISHIMA[1]

[1]*Department of Neurosurgery, Kansai Rosai Hospital, Amagasaki, Hyogo, Japan*

## Abstract

**We present a case of pontine infarction caused by subclavian steal phenomenon (SSP) due to subclavian artery stenosis (SAS) and an arteriovenous shunt in the forearm in a 74-year-old man with hemodialysis and stenting for SAS with improvement of SSP. He developed dysarthria during dialysis. He was admitted to our hospital and diagnosed with a pontine infarction. As the basilar artery appeared to be occluded on magnetic resonance angiography, an emergency diagnostic angiography was performed. Aortagram showed severe stenosis of the left subclavian artery. Right vertebral artery (VA) angiogram revealed retrograde arterial blood flow from the right VA to the left VA via the VA union, which suggested SSP. In addition, the steal was augmented by an ipsilateral hemodialysis arteriovenous shunt. Percutaneous subclavian artery stenting was performed 12 days later, and there was no recurrence of symptoms in the follow-up period. To our knowledge, this study is the first to report a patient with SSP who developed a pontine infarction due to SAS and an arteriovenous shunt during hemodialysis and who underwent subclavian artery stenting and had a good outcome.**

Keywords: subclavian steal phenomenon, stent treatment, subclavian artery stenosis, brainstem infarction, hemodialysis

## Introduction

In subclavian steal syndrome (SSS), retrograde blood flow is observed in the vertebral artery (VA) (mainly due to stenosis of the subclavian artery), which can result in symptoms such as vertebrobasilar artery circulatory insufficiency, cerebral infarction, and ipsilateral arm lethargy and numbness.[1)] The subclavian steal phenomenon (SSP) is rarely symptomatic and is often detected incidentally.[2)] Asymptomatic SSP during dialysis is rare (2.6%),[3)] as are symptomatic cases,[4)] with only five cases reported so far. Among these cases, there are no reports of acute cerebral infarction. We herein describe a case of SSP augmented by hemodialysis arteriovenous shunt resulting in a pontine infarction, which was successfully treated with stenting for subclavian artery stenosis (SAS).

## Case Report

A 74-year-old man was admitted to our hospital with dysrhythmia, right hemiparesis, and a National Institutes of Health Stroke Scale score of 6 during dialysis. One week earlier, he presented with transient ischemic attack symptoms with dysrhythmia during hemodialysis. His medical history included chronic renal failure due to nephritis, requirement for hemodialysis, critical limb ischemia, dyslipidemia, and angina pectoris. He had a hemodialysis arteriovenous shunt in his left forearm. He was on dual antiplatelet therapy (clopidogrel and aspirin) for angina pectoris.

Diffusion-weighted magnetic resonance imaging showed an acute brainstem infarction in the left pons. Magnetic resonance angiography (MRA) revealed occlusion of the basilar artery (BA) (Fig. 1A, B). Cerebral angiography was then performed, and his right vertebral artery angiogram showed retrograde blood flow through the VA union to the

Received January 16, 2024; Accepted March 15, 2024

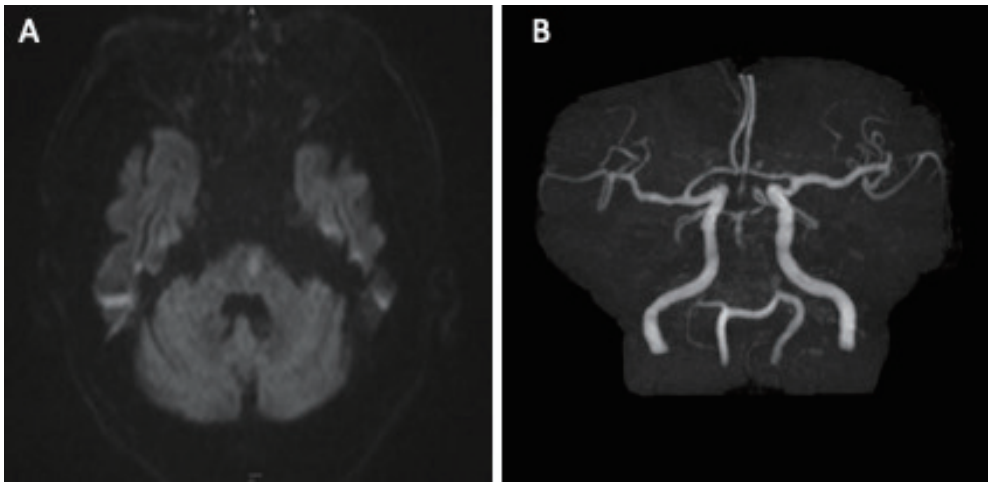


**Fig. 1 (A) Diffusion-weighted magnetic resonance imaging revealed left pontine infarction. (B) Magnetic resonance angiography (MRA) showed a suspected occlusion of the basilar artery (BA).**

left VA. In addition, the retrograde blood flow was in a direction through the left subclavian artery (SA) and was sucked into the left dialysis shunt (Fig. 2A). A small amount of blood ascended from the right VA to the BA, which seemed to indicate a BA occlusion (Fig. 2B). Right vertebral artery angiogram with compression of the left brachial artery revealed increased antegrade blood flow to the BA and decreased retrograde blood flow to the left VA (Fig. 2C). Furthermore, left internal carotid angiogram showed blood flow to the BA via the posterior communicating artery (Fig. 2D). Computed tomography angiography (CTA) showed SAS accompanied by calcification (Fig. 3A). Moreover, carotid echocardiography showed reversal flow of the left VA. The left arm brachial artery-basilic vein arteriovenous shunt blood flow rate in this patient was 914 mL/min. We considered that treatment of SAS could ameliorate these conditions, which was performed at a later date.

**Stenting for subclavian artery stenosis**

On the 13th hospital day, a 7-Fr femoral sheath and a 4-Fr brachial sheath were inserted under general anesthesia, and endovascular treatment was initiated. Aortagram showed severe stenosis of the left SA (Fig. 3B). The stenosis rate, the length of the lesion, and the diameter of the proximal and distal normal SA was 80.2%, 16.04 mm, and 14.60 mm and 12.89 mm, respectively. A 0.035-in. stiff wire (300 cm; Terumo, Tokyo, Japan) was guided from the left brachial artery to the ascending aortic arch via the affected left SA, and a 6-Fr gooseneck snare catheter (Amplatz Gooseneck 6-Fr 25 mm; Medtronic, Minneapolis, MN, USA) from the femoral artery to the ascending aorta. A 0.035-in. stiff wire was caught by gooseneck snare catheter (Medtronic) and was pulled out of the femoral artery sheath. A balloon-expandable stent (Omnilink Elite 10.0 mm/29 mm; Abbott, Chicago, IL, USA) was derived from the femoral artery and positioned at a sufficient margin from the left VA to cover most of the stenotic lesion. The balloon was expanded and the stent was deployed (Fig. 3C, D). MRA and diffusion-weighted imaging on postoperative day 1 showed improved antegrade blood flow in the left VA and BA and no new infarcts (Fig. 4A, B). Carotid echocardiography showed antegrade blood flow in the left VA. Postprocedural therapy using argatroban was initiated to prevent in-stent thrombus formation. The patient was transferred to a rehabilitation hospital with no procedural complications (modified Rankin Scale score of 3) on the 36th day. After treatment, SSP no longer occurred, including during dialysis.

## Discussion

To our knowledge, this study is the first to report SA stenting in a patient with a pontine infarction due to enhanced SSP related to severe SAS and a subcutaneous arteriovenous shunt medically created for hemodialysis. The patient had a prior transient ischemic attack (TIA) during hemodialysis and later developed a cerebral infarction. We hypothesized that SAS and an arteriovenous shunt for hemodialysis enhanced the blood steal phenomenon and hemodynamic mechanisms caused a perforating branch infarction in the pontine during hemodialysis.

SSS is a configuration of signs and symptoms resulting from retrograde blood flow in the VA due to stenosis or occlusion of the proximal SA.[5-8] It is predominantly caused by atherosclerosis, although other factors such as vasculitis, thoracic outlet syndrome, postsurgical stenosis, or congenital abnormalities may be involved.[1] When the stenosis is at least moderate (>50%), over 90% of patients show retrograde blood flow in the VA.[9] However, not all are symptomatic, and tools like duplex ultrasonography and transcranial Doppler studies may incidentally detect this rever-

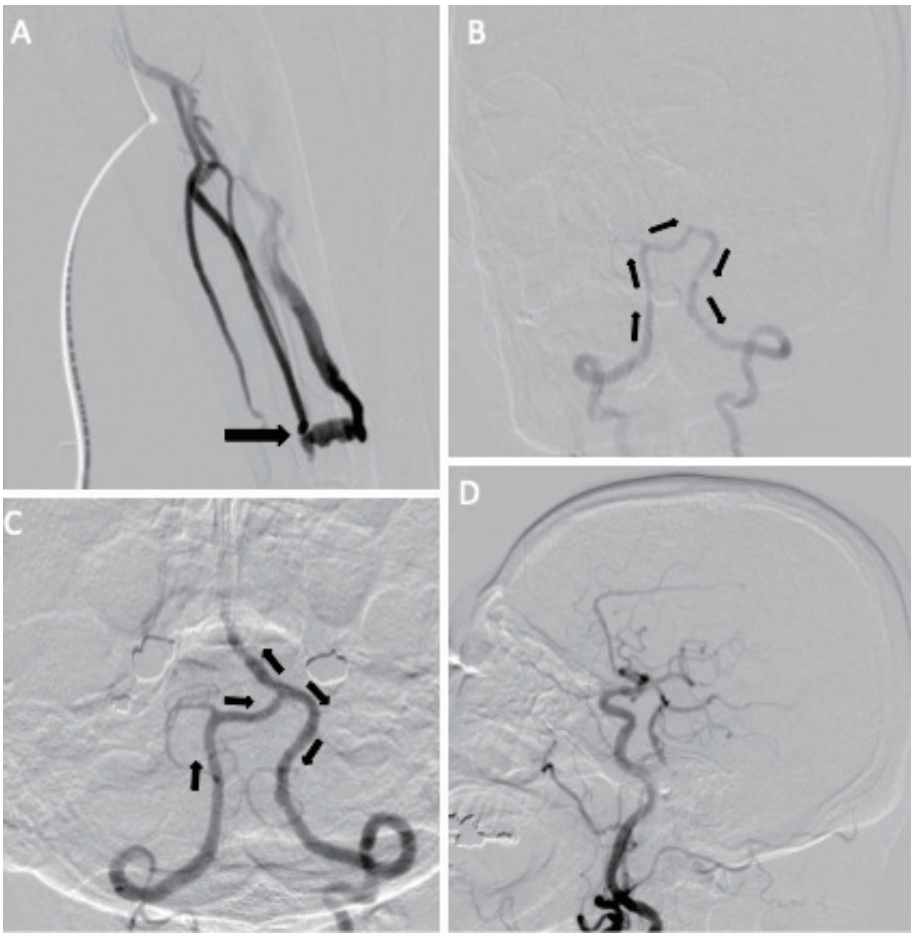


**Fig. 2 (A) The retrograde blood flow was in a direction through the left SA and was sucked into the dialysis shunt (*arrow*). (B) Right vertebral angiography showed retrograde blood flow to the left vertebral artery (VA) (*arrows* indicate direction of blood flow). (C) Right vertebral angiography with left arm compression showed increased antegrade blood flow to the BA (*arrows* indicate direction of blood flow). (D) Lateral view of the left internal carotid angiography showed flow from the anterior circulation to the BA via the posterior communicating artery.**

sal.[10] If the blood supply from the collateral circulation is sufficient to respond to increased demand, most patients remain asymptomatic.[10] Only a few of the patients become symptomatic, which includes exercise-induced arm pain or fatigue.[11] Unilateral flow reversal can cause TIA, while bilateral flow reversal can induce non-lateralizing cerebral ischemia.[8] Furthermore, hemodialysis patients with a subcutaneous arteriovenous shunt medically created for blood access may exhibit a similar steal phenomenon.[10]

A study reviewing 2192 cases of stroke showed that SSP was diagnosed in 2.2% of cases, while cerebral infarctions in the posterior circulation were considered related to the subclavian steal in 20% of these cases and approximately 0.4% of all cases.[3] Other reports show that SSP was diagnosed in 0.6%-6.4% of cerebral infarction cases.[12-15] The present case was due to a combination of severe SAS and a high-flow arteriovenous shunt during hemodialysis.

The SSS caused by an arteriovenous shunt during hemodialysis is quite rare. A previous report found that a high-flow arteriovenous shunt in hemodialysis >2000 mL/min caused symptomatic vertebrobasilar artery insufficiency.[5] In the present case, the arteriovenous shunt flow rate was 914 mL/min, which may have had less effect on SSP than that in other reports. Nevertheless, based on our findings that flow from the retrograde left VA was sucked into the arteriovenous shunt and that compression of the left brachial artery reduced blood flow into the retrograde left VA and resulted in the antegrade blood flow of the BA (Fig. 2 C), we considered that the arteriovenous shunt contributed to worsening SSP.

Surgical treatment options include treatment of the SAS or reduction of the arteriovenous shunt flow rate in the hemodialysis shunt. Treatment of atherosclerotic occlusive lesions affecting the proximal SA includes various techniques such as bypass surgery, percutaneous transluminal angioplasty (PTA), stenting, endarterectomy, and transposition.[2] A retrospective medical study involving 51 cases of SA bypass surgery showed a mortality rate of 0% over a

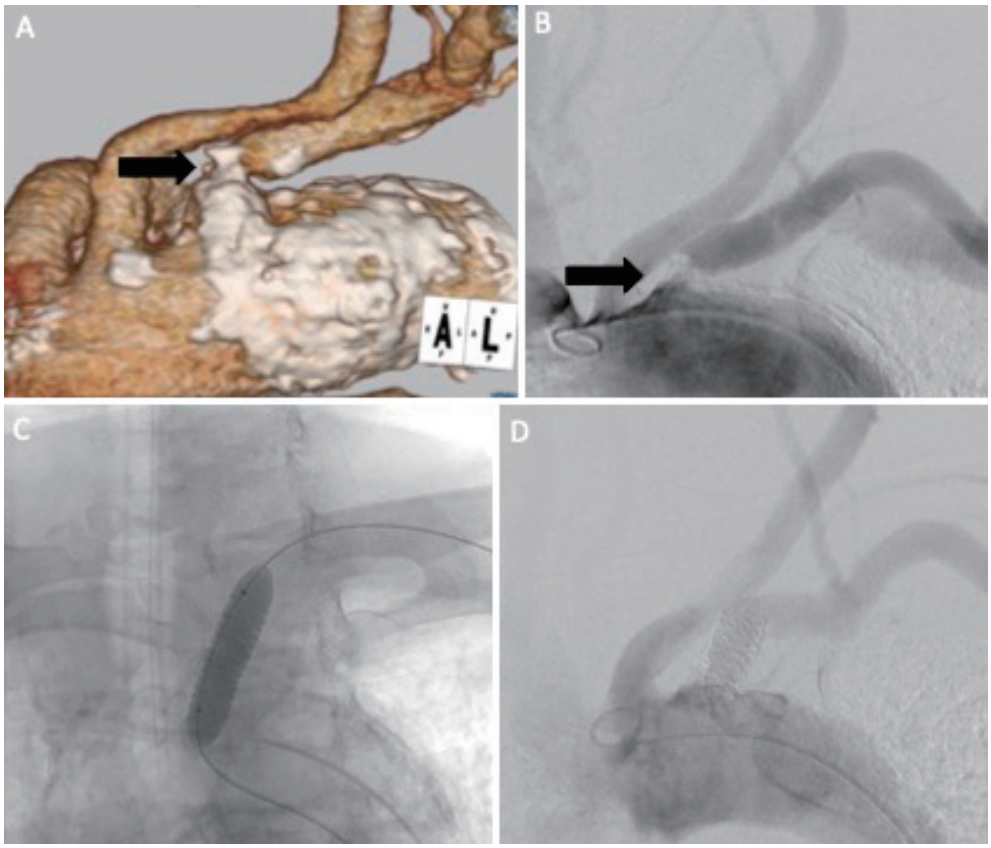


**Fig. 3 (A) Computed tomography angiography showed heavy stenosis at the beginning of the subclavian artery (*arrow*). (B) Aortography showed stenosis at the beginning of the subclavian artery (*arrow*). (C) The expandable stent balloon was dilated at the site of stenosis. (D) After stent deployment, aortography showed antegrade blood flow to the left VA.**

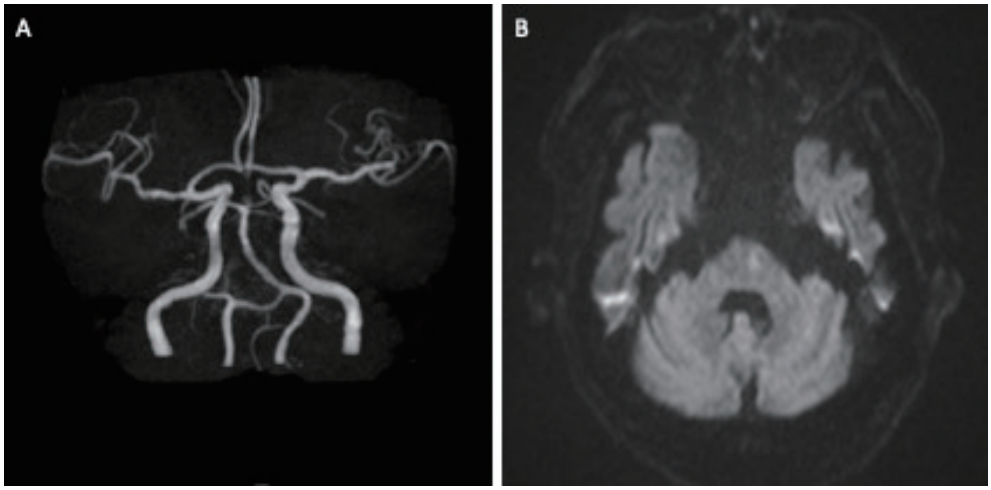


**Fig. 4 (A) (B) MRA and DWI on postoperative day 1 showed improved blood flow in the left VA and BA and no new infarcts.**

period of 8 years, with four cases exhibiting recurrence.[16] In addition, from a cohort of 53 cases, follow-up at 24.5 months indicated a mortality rate of 0% and a positive outcome characterized by a 100% patency rate.[17] Moreover, a retrospective study of 110 cases of endovascular treatment showed a patency rate at 3 years of 93% for subclavian stenosis and 65% for obstruction. Furthermore, a report of 61 cases showed 98% patency at 1 year and 82% at 5 years.[18] Endovascular treatment options include PTA and stenting. Percutaneous PTA and stenting are associated with higher long-term patency and less restenosis and occlusion.[19,20]

Considering invasiveness, we selected endovascular treatment. Furthermore, SAS was the primary factor of SSP in our case, while the effect of the shunt on hemodialysis was the secondary one. Thus, endovascular stenting was the preferred treatment.

## Conclusion

We report a case of SA stenting in a patient with pontine infarction caused by enhanced SSP related to severe SAS and a subcutaneous arteriovenous shunt medically created for hemodialysis.

## Acknowledgments

We thank Edanz (https://jp.edanz.com/ac) for editing a draft of this manuscript.

## Abbreviations

BA: basilar artery
CTA: computed tomography angiography
MRA: magnetic resonance angiography
PTA: percutaneous transluminal angioplasty
SA: subclavian artery
SAS: subclavian artery stenosis
SSP: subclavian steal phenomenon
SSS: subclavian steal syndrome
TIA: transient ischemic attack
VA: vertebral artery

## Informed Consent

Written informed consent for publication of the patient's information and images was obtained from the patient.

## Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

## Authors' Contributions

Drs. Toyota, Murakami, and Takahara contributed to the conception and design of the study. Drs. Toyota, Murakami, Touhara, Hoshikuma, Okuhara, Achiha, Yamada, Kobayashi and Takahara performed the surgery. Drs. Kishima, Toyota, and Murakami wrote and revised the manuscript.

## Conflicts of Interest Disclosure

The authors declare no conflict of interest.

## References

1) Ochoa VM, Yeghiazarians Y: Subclavian artery stenosis: a review for the vascular medicine practitioner. *Vasc Med* 16: 29-34, 2011
2) Osiro S, Zurada A, Gielecki J, Shoja MM, Tubbs RS, Loukas M: A review of subclavian steal syndrome with clinical correlation. *Med Sci Monit* 18: RA57-RA63, 2012
3) Kimura K, Yamaguchi T, Yasaka M, Tsuchiya T: [Hemodynamics of the vertebral artery in subclavian steal syndrome and subclavian steal phenomenon]. *Rinsho Shinkeigaku* 31: 970-973, 1991 (Japanese)
4) Agarwal S, Schwartz L, Kwon P, et al.: Subclavian steal syndrome due to dialysis fistula corrected with subclavian artery stenting. *Neurol Clin Pract* 8: e23-e25, 2018
5) Kaneko Y, Yanagawa T, Taru Y, et al.: Subclavian steal syndrome in a hemodialysis patient after percutaneous transluminal angioplasty of arteriovenous access. *J Vasc Access* 19: 404-408, 2018
6) Schenk WG III: Subclavian steal syndrome from high-output brachiocephalic arteriovenous fistula: a previously undescribed complication of dialysis access. *J Vasc Surg* 33: 883-885, 2001
7) Maiodna E, Ambekar S, Johnson JN, Elhammady MS: Dialysis arteriovenous fistula causing subclavian steal syndrome in the absence of subclavian artery stenosis. *Case Rep Vasc Med* 2015: 720684, 2015
8) Reivich M, Holling HE, Roberts B, Toole JF: Reversal of blood flow through the vertebral artery and its effect on cerebral circulation. *N Engl J Med* 265: 878-885, 1961
9) Harper C, Cardullo PA, Weyman AK, Patterson RB: Transcranial Doppler ultrasonography of the basilar artery in patients with retrograde vertebral artery flow. *J Vasc Surg* 48: 859-864, 2008
10) Potter BJ, Pinto DS: Subclavian steal syndrome. *Circulation* 129: 2320-2323, 2014
11) Hennerici M, Klemm C, Rautenberg W: The subclavian steal phenomenon: a common vascular disorder with rare neurologic deficits. *Neurology* 38: 669-673, 1988
12) Tan TY, Schminke U, Lien LM, Tegeler CH: Subclavian steal syndrome: can the blood pressure difference between arms predict the severity of steal? *J Neuroimaging* 12: 131-135, 2002
13) Fields WS, Lemak NA: Joint Study of extracranial arterial occlusion. VII. Subclavian steal—a review of 168 cases. *JAMA* 222: 1139-1143, 1972
14) Lord RS, Adar R, Stein RL: Contribution of the circle of Willis to the subclavian steal syndrome. *Circulation* 40: 871-878, 1969
15) Bornstein NM, Norris JW: Subclavian steal: a harmless haemodynamic phenomenon? *Lancet* 2: 303-305, 1986
16) AbuRahma AF, Robinson PA, Jennings TG: Carotid-subclavian artery bypass grafting with PTFE grafts for symptomatic subclavian artery stenosis or occlusion: 20-year experience. *Vasca J Surg* 32: 411-418, 2000
17) Qi L, Gu Y, Zhang J, et al.: Surgical treatment of subclavian artery occlusion. *Zhongguo Xiu Fu Chong Jian Wai Ke Za Zhi* 24: 1030-1032, 2010
18) Wang KQ, Wang ZG, Yang BZ, et al.: Long-term results of endovascular therapy for proximal subclavian arterial obstructive lesions. *Chin Med J (Engl)* 123: 45-50, 2010
19) Ahmed MA, Parwani D, Mahawar A, Gorantla VR: Subclavian artery calcification: a narrative review. *Cureus* 14: e23312, 2022
20) Chatterjee S, Nerella N, Chakravarty S, Shani J: Angioplasty alone versus angioplasty and stenting for subclavian artery stenosis—a systematic review and meta-analysis. *Am J Ther* 20: 520-523, 2013

Corresponding author: Tomoaki Murakami, MD, PhD
Department of Neurosurgery, Kansai Rosai Hospital, 3-1-69 Inabaso, Amagasaki, Hyogo 660-8511, Japan.
*e-mail*: mmtmtomoaki@gmail.com