CASE REPORT

A case of transient mid-ventricular akinesia (a variant form of Takotsubo cardiomyopathy) followed with I-123-beta-metyl-iodophenyl pentadecanoic acid and I-123-meta-iodobenzyl-guanidine myocardial scintigraphy

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Summary
A 67-year-old woman without history of heart disease was admitted with chest oppression. Her electrocardiogram (ECG) at the time of admission showed ST segment elevation in leads V2—V6. Cardiac ultrasound revealed severe hypokinesis in mid to apical portion of anterior wall. Emergent coronary angiography showed normal coronary arteries. Left ventriculography (LVG) revealed akinesis of mid portion of anterior and inferior wall with hyperkinesis of apex and basal portion of anterior and inferior wall. Cardiac ultrasound examination 3 months later revealed improvement in LV contraction without mid-ventricular akinesia. The LVG performed 6 months later showed no focal asynergy. In I-123-beta-metyl-iodophenyl pentadecanoic acid myocardial scintigraphy the discrepancy of uptake between apical and anterior and inferior wall of mid region (more uptake in apex) was reduced. Using I-123-meta-iodobenzyl-guanidine myocardial scintigraphy in acute phase, decreased uptake in the mid portion of anterior and inferior to lateral wall was seen in early and delayed images and that persisted through 6 months. As these findings resembled those of Takotsubo cardiomyopathy other than affected region, it is possible to say that basically they belong to same entity of disease but they are different in their phenotype.

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Introduction

Takotsubo cardiomyopathy (TTC) is well known among cardiologists for its characteristic left ventricular (LV) contraction pattern of transient apical akinesia and basal hyperkinesia [1,2] and diagnostic guidelines have been proposed [3]. Although the distinct cause is still uncertain, some relation between TTC and a discrepancy of sympathetic innervation in the apical and basal region is suggested [2,4]. Recently other types of reversible cardiomyopathy, which have variant forms or inverted types of Takotsubo contractile pattern have been reported [5–7]. Here we will present a case of transient mid-ventricular akinesia (or a variant form of TTC) followed up with I-123-beta-metyl-iodophenyl pentadecanoic acid (BMIPP) and I-123-meta-iodobenzyl-guanidine (MIBG) myocardial scintigraphy for 6 months.

Case report

A 67-year-old woman without history of heart disease was admitted with vomiting and chest oppression. She was sick for 3 days prior to admission with fever up to 39.5°C and loss in appetite. She had a past medical history of appendectomy when she was 13 years old and had undergone surgery for an ovarian tumor at age 22. In addition, she was injured in a road traffic accident at 63 years of age and an operation of resection of ruptured small intestine and repair of pelvic fracture was done. She was a non-smoker (of cigarettes) and her family history revealed nothing in particular.

Physical examination at the time of admission showed alert consciousness, with body temperature of 35.8°C, blood pressure of 138/78 mmHg, pulse 77 min⁻¹, regular, and there was no rale, no murmur nor extra cardiac sound in chest auscultation and operation scar in her abdomen was visible. There was no other significant finding.

Her electrocardiogram (ECG) at the time of admission is shown in Fig. 1A. It showed sinus rhythm with complete right bundle branch block (CRBBB), which had been previously diagnosed. ST segment elevation was seen in leads V2–V6. Cardiac ultrasound examination revealed severe hypokinesis in the mid to apical portion of the anterior wall with preserved LV global contraction and there was no obvious valvular heart disease. Blood test at the time of admission showed slightly elevated white blood cell (WBC) count of 9490 mm⁻³ (3500—8500 mm⁻³, normal limit in our institute, and so forth), creatine kinase (CK) was elevated slightly but less than twice over normal range to 181 U/l (45—163 U/l) with normal CK-MB of 16 U/l (4—21 U/l) and elevated troponin T level of 0.75 ng/ml (<0.1 ng/ml). Brain natriuretic peptide (BNP) was elevated to 62.8 pg/ml (<18.4 pg/ml) and C-reactive peptide was up to 2.89 mg/dl (<0.45 mg/dl), but there were no other abnormal values including HbA1c and lipids.

According to the findings above, we could not exclude the possibility of acute coronary syndrome. Then emergent coronary angiography (CAG) was performed, which showed normal coronary
Figure 2  Left ventriculography of the acute phase (A: diastole; B: systole) and 6 months later (C: diastole; D: systole). Akinesia seen in mid portion of anterior and inferior wall in acute phase was normalized 6 months later.

arteries. Left ventriculography (LVG) revealed akinesia of mid portion of anterior and inferior wall with hyperkinesis of the apex and basal portions of anterior and inferior wall (Fig. 2A and B). We did not treat her with any drugs, but let her stay in hospital solely for observational purposes.

During observation, she did not complain of any further chest oppression and cardiac enzymes did not increase from the time of admission. WBC counts and CRP returned to normal range spontaneously. The ECG on second day in hospital is shown in Fig. 1B. ST segment elevation disappeared, but inverted T waves were seen in leads II, III, aVF and the entire chest leads. ECG was taken periodically but no significant findings other than CRBBB were seen 3 months later (Fig. 1C) and 6 months later (Fig. 1D). Cardiac ultrasound examination that was performed 3 months after admission revealed improvement in LV contraction without mid-ventricular akinesia. Repeated cardiac catheterization was performed 6 months after onset. CAG showed normal coronary arteries again and LVG looked just as normal without focal asynergy (Fig. 2C and D).

The course of BMIPP and MIBG myocardial scintigraphy is presented in Fig. 3 (device used: scintigraphy camera and data analysis; Starcom 4000 XR/T, General Electric, Milwaukee, WI, USA). We have defined the scope of the apex as one-third of the inside part, mid as next one-third region, and base as outer one-third of bull's eye image. During acute phase, decreased uptake in mid portion of anterior and inferior to lateral wall was seen in BMIPP myocardial scintigraphy and it was persistent through 3 and 6 months later. As for MIBG myocardial scintigraphy in acute phase, decreased uptake in the mid portion of anterior and inferior to lateral wall was seen in early image and delayed (4h after early imaging) image similar to BMIPP myocardial scintigraphy. Washout map showed increased washout in mid to basal portion of inferior and lateral wall. Although images did not fully match, the tendency of the images of early and delayed MIBG resembled those images taken 3 and 6 months later. Washout map showed increased washout in mid portion of anterolateral wall in images taken 3 and 6 months later, unlike those taken during the acute phase. Heart/mediastinum ratio (H/M) in MIBG showed normal value in all measurements (acute phase 2.87, 3.03; 3 months later 2.57, 2.51; 6 months later 2.59, 2.92, early and delayed, respectively).

Discussion

There is no unified name for this disorder so far. Some say "inverted TTC" for this condition [7]. But even in the same "inverted TTC", detailed
Figure 3  Serial colored bull’s eye images of BMIPP and MIBG myocardial scintigraphy in the acute phase (A), 3 months later (B), and 6 months later (C). Below each image, except for MIBG washout, the respective single photon emission computed tomography (SPECT) images are shown (short axis, long axis horizontal and long axis vertical, from left to right). MIBG imagings showed persistent decreased uptake in anterior and inferior wall of mid portion compared with BMIPP. Washout map showed increased washout in mid portion of anterolateral wall 3 and 6 months later.

LV wall motion abnormalities were different among reporters. For example, one says LV wall motion was akinesis in the basal region, normal in the mid-ventricular region, and hyperkinesis in the apex, while the other says akinesis in the mid-ventricular, and hyperkinetic in the basal and apical region. So far the definition of "inverted TTC" is not established.

We named "transient mid-ventricular akinesis" for our case. Usually TTC consists of transient apical to mid akinesia and basal hyperkinesis. If you diagnose this disorder as "inverted TTC", the basal
region must be transient akinesia, but in this case, the basal region was not akinesia and showed normal contraction in acute phase. One report termed as "transient mid-ventricular dyskinesia" for a case that has almost the same contractile pattern with our case [5]. However, we hesitate to use the word "dyskinesia" to express our case's middle region's contraction pattern because mid region did not go outward in the systolic phase. In this regard, we considered that "transient mid-ventricular akinesia" is the most suitable for this condition and that is why we used it for the title of this report.

There were differences in LV wall motion between cardiac ultrasound and LVG. From her cardiac position or some other reason, LV inferior wall was very difficult to see using ultrasound and we might have overlooked the asynergy in the inferior wall. The time course between ultrasound and LVG is almost within an hour. It is unnatural to think the difference was due to period. We speculate the difference is due to method.

BMIPP myocardial scintigraphy is useful to determine cardiac metabolism by means of fatty acid and MIBG myocardial scintigraphy is useful to assess adrenergic nervous activity [8]. Using these nuclides and 201-Tl, an article noted the suggestion that primary cause of TTC is related to a disturbance of cardiac sympathetic innervation [2]. In addition, the author published a subsequent report that speculated after 6 months' follow-up of TTC with BMIPP and MIBG myocardial scintigraphy that the discrepancy of sympathetic innervation between the apical and basal regions was the cause of characteristic transient LV akinesia [4]. The other article says TTC might represent a stunned myocardium caused by a disturbance of the coronary microcirculation using 99mTc-tetrofosmin, BMIPP, MIBG, and 99mTc-pyrophosphate myocardial scintigraphy [9]. As for cause of atypical TTC, it may be differentiated from entities with similar clinical presentations such as myocarditis and myocardial infarction by using cardiovascular magnetic resonance imaging [10]. To our knowledge, no reports are available about findings of BMIPP or MIBG myocardial scintigraphy of the variant form of TTC and this time we applied the same methodology to the prior report of the author's to clarify the mechanism of transient mid-ventricular akinesia.

Observing our results in Fig. 3, the discrepancy of uptake between apical and anterior and inferior wall of mid region was reduced during the course of BMIPP scintigraphy aside from some other subtle findings. So if we consider the main findings of the acute phase BMIPP scintigraphy is the discrepancy of uptake between anterior and inferior wall of mid region and apex, it is reasonable to acknowledge that abnormal finding was discovered during the course.

With MIBG myocardial scintigraphy, on the other hand, decreased uptake in the mid portion of anterior and inferior to lateral wall was seen in early and delayed images in acute phase and that persisted through 6 months. These results suggested the abnormal findings of MIBG persisted for 6 months even though the LV contraction was recovered and discrepancy of MIBG uptake was seen between affected and unaffected regions.

Though there are differences in the affected region, the outline of those findings resembles the findings of TTC that the author reported before [4]. The discrepancy of sympathetic innervation between the mid portion of anterior and inferior wall and other regions was suspected. We cannot ascertain the details by this report but we may speculate that transient mid-ventricular akinesia or variant forms of TTC have resembled basic pathophysiology with TTC but they affect different regions. In other words, it is possible to say that basically they belong to the same entity of disease but they are only different in their phenotypes through the findings that we obtained from this case.

References

