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Usefulness of Ultrasonography in Carotid Arteries and Combined Positron Emission Tomography/Computed Tomography for Diagnosis of Takayasu Arteritis With Unusual Presentation as Acute Myocardial Infarction: A Case Report

Kotaro MIYAGAWA, MD
Jun SHIRAISHI, MD
Michitaka NASU, MD*1
Sayuki TORII, MD
Masayasu ARIHARA, MD*1
Masayuki HYOGO, MD
Takakazu YAGI, MD
Takatomo SHIMA, MD
Takashi OKADA, MD
Yoshio KOHNO, MD
Hiroaki MATSUBARA, MD*2

Abstract

This unusual case of Takayasu arteritis presenting as acute myocardial infarction could be defined by ultrasonography and 18-fluorodeoxyglucose positron emission tomography (^{18}F-FDG PET) coregistered with computed tomography (CT). A 55-year-old male was admitted to our hospital with continuous chest pain and left-side neck pain. After primary percutaneous coronary intervention, elevation of inflammatory markers persisted and dull pain in the left side of the neck continued. Ultrasonography revealed characteristic wall thickening of the left common carotid artery and subsequent ^{18}F-FDG PET with CT depicted positive uptake in the left common carotid artery and the vessel wall of the ascending aorta, confirming the diagnosis of Takayasu arteritis. Three months after angioplasty, follow-up cardiac catheterization was performed. Coronary angiography showed no restenosis. During the catheterization, angiography confirmed the mild stenosis in the long segment of the left common carotid artery and the left subclavian artery as well as the focal narrowing and the dilation of the abdominal aorta. This case shows that ultrasonography in the cervical region and combined ^{18}F-FDG PET with CT may be useful in the diagnosis and evaluation of Takayasu arteritis. In addition, we should pay attention to underlying disease even in middle-aged or older male patients with acute myocardial infarction.

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京都第一赤十字病院 循環器科，*1救急部：〒605−0981 京都市東山区本町15−749；*2京都府立医科大学大学院医学研究科循環器病態制御学，京都

Departments of Cardiology and *1Emergency Medicine, Kyoto First Red Cross Hospital, Kyoto; *2Department of Cardiovascular Medicine, Kyoto Prefectural University School of Medicine, Kyoto

Address for correspondence: SHIRAISHI J, MD, Department of Cardiology, Kyoto First Red Cross Hospital, Honmachi 15−749, Higashiyama-ku, Kyoto 605−0981; E-mail: sjun@msj.biglobe.ne.jp

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Key Words
■ **Myocardial infarction, pathophysiology** ■ **Ultrasonic diagnosis**
■ **Computed tomography**

INTRODUCTION

Takayasu arteritis (TA) is a chronic vasculitis of unknown etiology, predominantly involving the aorta and its primary branches that frequently lead to stenotic, occlusive, and aneurysmal change.[1,2] TA usually affects young females and its prevalence in females is approximately 10 times that in males.[3,4] Although conventional angiography has been the mainstay of diagnostic image analysis for TA, the diagnosis of early TA is very difficult to make in "the prestenotic stage" by this method. In contrast, recent reports have demonstrated that, in addition to enhanced computed tomography (CT) and magnetic resonance imaging, ultrasonography as well as 18-fluorodeoxyglucose positron emission tomography (^{18}F-FDG PET) are useful in the diagnosis and the assessment of early TA.[5-10] We describe an unusual case of TA in a middle-aged male presenting with acute myocardial infarction, which could be diagnosed noninvasively by ultrasonography and ^{18}F-FDG PET coregistered with CT.

CASE REPORT

A 55-year-old male was admitted to our hospital with continuous chest pain for 2 hr. He had no obvious past history of cardiovascular disease, but had felt a slight dull pain in the left side of the neck for 3 months. His coronary risk factors included smoking and hyperlipidemia. Routine blood test results were normal, except for C-reactive protein (CRP) of 1.9 mg/d*l* (normal range 0.0−0.3 mg/d*l*). Electrocardiography showed abnormal ST-segment elevation in the Ⅱ, Ⅲ, aVF leads, and emergency echocardiography revealed decreased wall motion in the inferoposterior wall of the left ventricle. Emergency coronary angiography indicated total occlusion in the mid segment of the right coronary artery (**Fig. 1−A**) and a diffuse lesion in the left anterior descending coronary artery with severe stenosis in the diagonal branch (**Fig. 1−B**). The left circumflex coronary artery was hypoplastic.

Primary percutaneous coronary intervention was performed for the mid right coronary artery occlusion. Using a 6F JR4 with side hole guiding catheter (mach-1, Boston Scientific) and a guide wire (Runthrough NS 0.014 inch, TERUMO), the occlusion was passed through without difficulties. After thrombectomy using an aspiration catheter (Export, Medtronic), Thrombolysis in Myocardial Infarction (TIMI) 2 flow was observed (**Fig. 1−C**). Then, after dilation using a balloon catheter (Maverick 2, 3.5/20 mm, Boston Scientific), a bare metal stent (Driver, 4.0/30 mm, Medtronic) was implanted (**Fig. 1−D**). Finally, TIMI 3 flow was obtained without residual stenosis (**Fig. 1−D**). He began daily treatment with 100 mg aspirin, 200 mg ticlopidine, 5 mg enalapril, 10 mg carvedilol, and 10 mg pravastatin.

After the primary percutaneous coronary intervention, his clinical course was uneventful except for the continuous dull pain in the left side of the neck. His plasma lipid profile showed that low-density lipoprotein cholesterol level was 221 mg/d*l*, high-density lipoprotein cholesterol level was 34 mg/d*l*, and triglyceride level was 225 mg/d*l*. Unexpectedly, the CRP gradually increased up to 18.2 mg/d*l* and the erythrocyte sedimentation rate (ESR) was 108 mm in the first hour. The value of these inflammatory makers did not decrease significantly and other laboratory data, including rheumatoid factor, antinuclear antibody, and antineutrophil cytoplasmic antibody did not show any abnormalities.

Ultrasonography of the cervical region was performed to examine the origin of his neck pain, which revealed diffuse, isoechoic, circumferential wall thickening of the left common carotid artery (**Figs. 2−A, B**). The wall of the right common carotid artery was also slightly thickened with some atherosclerotic change (**Fig. 2−C**). The wall thickening of both carotid arteries spared the external and internal carotid arteries, findings considered consistent with TA. In addition, ^{18}F-FDG PET coregistered with CT was employed to identify the inflammation of the vasculature directly. Whole-body ^{18}F-FDG PET suggested FDG uptake in the bilateral common carotid arteries as well as ascending aorta, arch, and descending aorta (**Fig. 3**). ^{18}F-FDG PET coregistered with CT showed elevated accumulations in the left common carotid artery, the vessel wall of the ascending aorta, and the lymph nodes (**Fig. 4**). TA was highly suspected

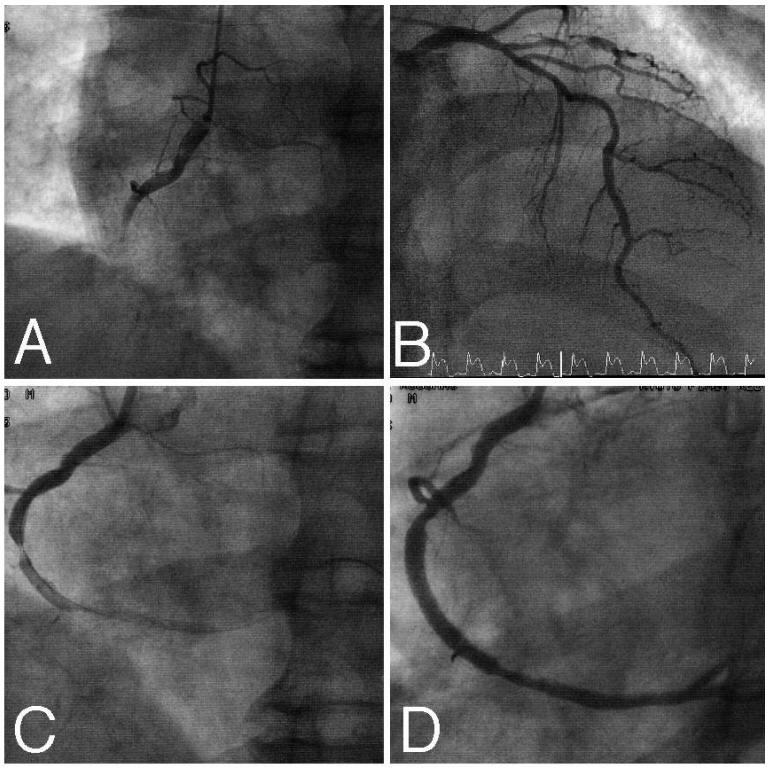

Fig. 1 Coronary angiograms
A: Left anterior view of right coronary angiography before primary percutaneous coronary intervention showing a total occlusion in the mid segment.
B: Cranial view of left coronary angiography showing a diffuse lesion in the left anterior descending coronary artery with severe stenosis in the diagonal branch.
C: Right coronary angiography immediately after thrombectomy showing residual stenosis.
D: Right coronary angiography immediately after coronary stenting showing the optimal result.

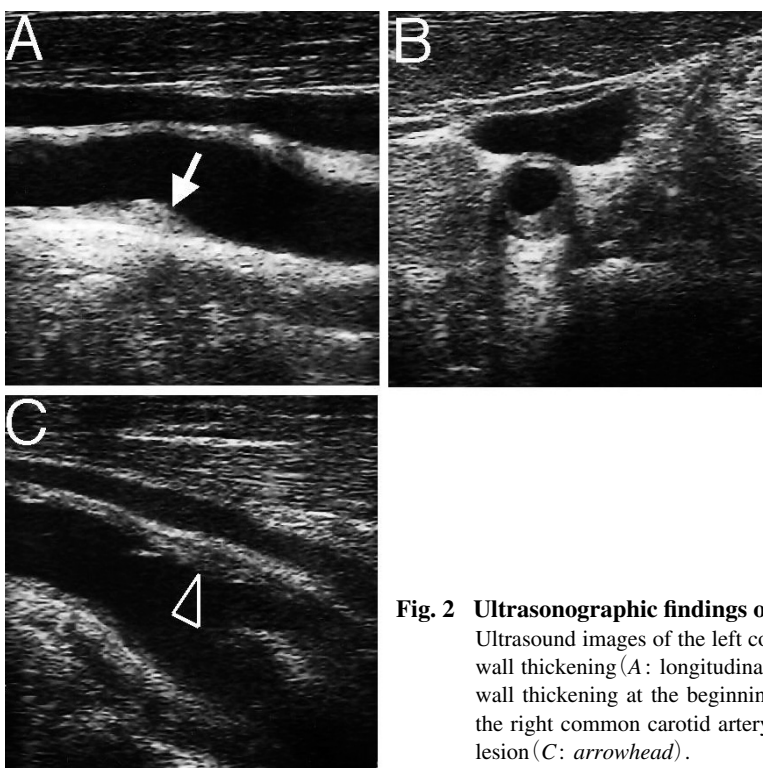

Fig. 2 Ultrasonographic findings of the common carotid arteries
Ultrasound images of the left common carotid artery showing diffuse, circumferential wall thickening (*A*: longitudinal view; *B*: transverse view), and the distal end of the wall thickening at the beginning of the carotid sinus (*arrow*). Ultrasound images of the right common carotid artery showing a slight wall thickening with atheromatous lesion (*C*: *arrowhead*).

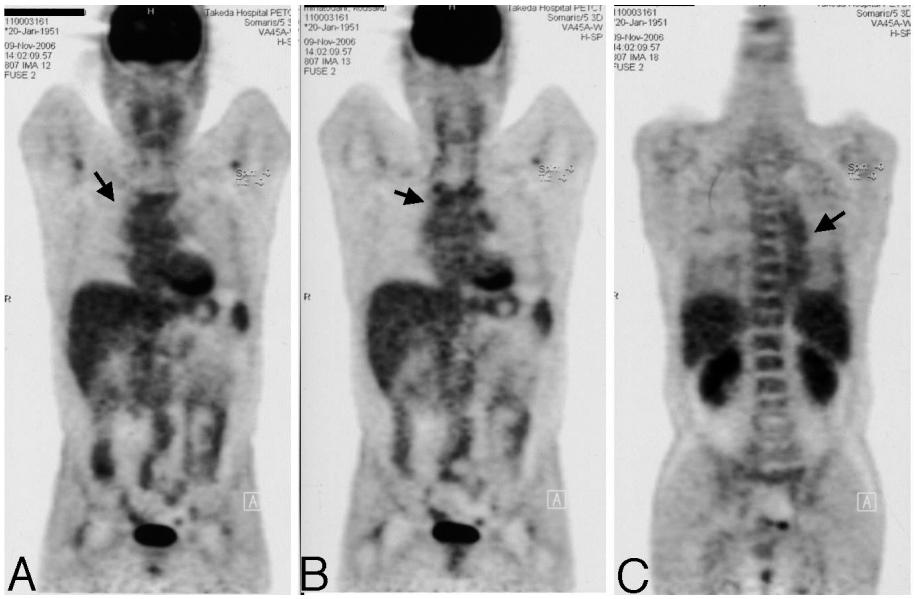

Fig. 3 Whole-body 18-fluorodeoxyglucose (18F-FDG) positron emission tomography (PET)
A−*C*: Coronal slices from anterior to posterior showing suspected FDG uptakes in the bilateral common carotid arteries as well as the ascending aorta, arch, and descending aorta (*arrows*).

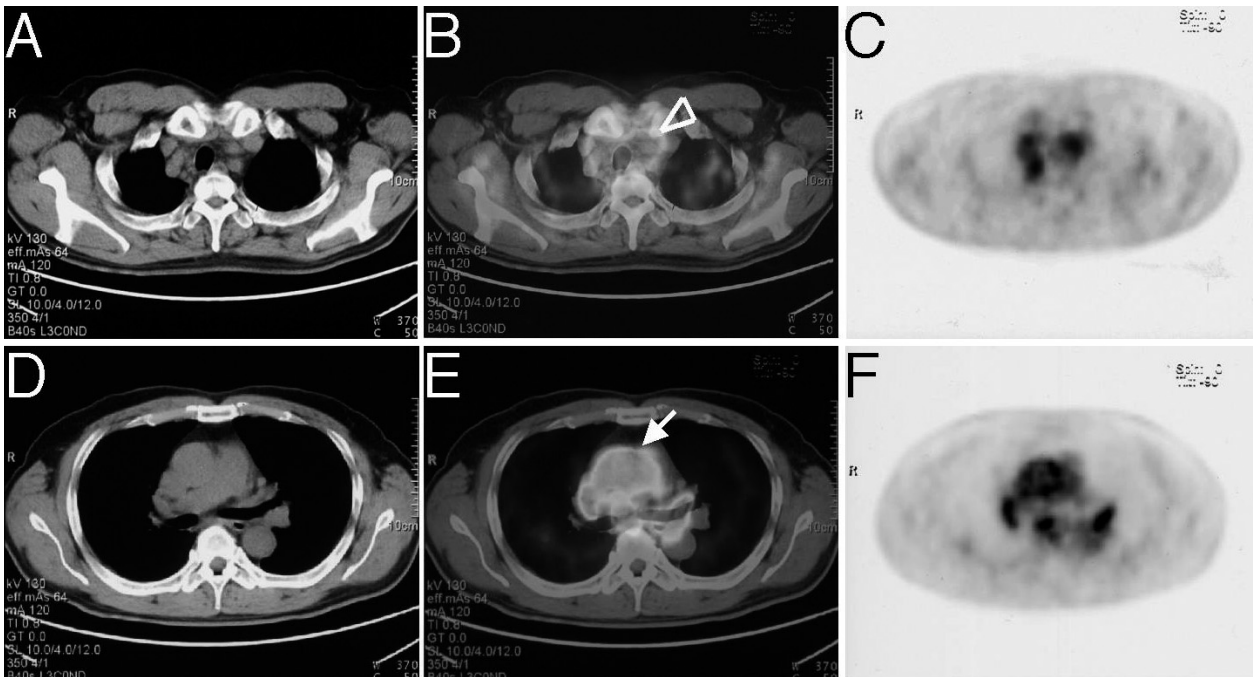

Fig. 4 18F-FDG PET coregistered with computed tomography
A, *D*: Computed tomography images.
B, *E*: Coregistered 18F-FDG PET with computed tomography images. 18F-FDG accumulations were observed in the left common carotid artery (*arrowhead*), the vessel wall of the ascending aorta (*arrow*), and the lymph nodes.
C, *F*: 18F-FDG PET images of same sections of *A* and *D*.
Abbreviations as in Fig. 3.

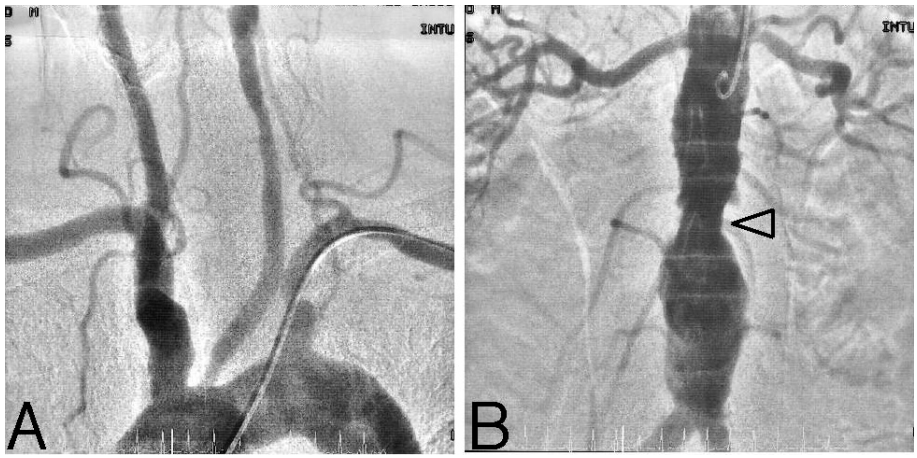

Fig. 5 Angiograms of the branches of the aortic arch (*A*) and the abdominal aorta (*B*)
Angiography of the branches of the aortic arch (*A*) showing slight stenosis in the long segment of the left common carotid artery and moderate stenosis of the left subclavian artery. Angiography of the abdominal aorta (*B*) showing focal narrowing (*arrowhead*) and dilation.

based on the findings from ultrasonography and ^{18}F-FDG PET combined with CT. Without specific treatment, the CRP gradually decreased down to 1.5 mg/d*l* spontaneously, the ESR to 21 mm in the first hour concomitantly, and the neck pain disappeared 2 months after the onset of acute myocardial infarction.

Three months after the angioplasty, follow-up cardiac catheterization was performed. Coronary angiography showed no restenosis. During the catheterization, angiography of the branches from the aortic arch and the abdominal aorta was also performed to confirm the diagnosis and evaluate the distribution of the disease. The slight stenosis in the long segment of the left common carotid artery was confirmed (**Fig. 5−A**). Slight stenosis of the left proximal common carotid artery as well as intermediate stenosis of the left subclavian artery were also found (**Fig. 5−A**). Additionally, focal narrowing and dilation of the abdominal aorta were detected (**Fig. 5−B**). These findings confirmed the diagnosis of TA. He has been carefully observed on an outpatient basis without steroid treatment, because the inflammatory makers have remained slightly above the normal laboratory value (CRP $<$ 1.5 mg/d*l*, ESR $<$ 20 mm in the first hour).

DISCUSSION

TA is an idiopathic granulomatous vasculitis, occurring predominantly in young females, which mainly attacks the aortic trunk, its main branches, and the pulmonary and coronary arteries.[1,2] The prevalence of coronary involvements in TA is about 9% to 10%.[11,12] However, recent studies have indicated that the incidence of coronary lesions in TA is relatively high (approximately 40%)[13,14] and TA is a relatively common underlying disease in young females with acute myocardial infarction.[15] Besides aortic regurgitation, heart failure ascribed to coronary artery disease and hypertension is the major cause of death in patients with TA.[11,13,14,16] Coronary artery lesions in TA can be classified into three types based on the pathological findings:[17] stenosis or occlusion of the coronary ostium and the proximal segments of the coronary arteries; diffuse or focal coronary arteritis (skip lesions); and coronary aneurysm. Most coronary involvements in TA are located at the ostium or the proximal segments of the coronary arteries, and this type frequently affects females.[17] In contrast, most cases of diffuse or focal coronary arteritis occur in middle-aged or older males.[17−19] According to this classification, the present patient might have focal coronary arteritis in the right coronary artery and diffuse coronary arteritis in the left anterior descending coronary artery. On the other hand, recent reports have demonstrated that platelet and coagulation activities are significantly enhanced in patients with TA.[20−22] Not only coronary stenotic lesion but also platelet hyperaggreability and hypercoagulability might have contributed to the pathogenesis of acute myocardial infarction in the present case.

Ultrasonography is a noninvasive, repeatable,

and superior method compared with contrast angiography for the detection of involvements of the common carotid artery, one of the most commonly affected vessels in patients with TA.[23,24] Ultrasonography can be used to estimate both the lumen and the vessel wall morphology, so can detect early TA in "the prestenotic stage".[7,8] The characteristic findings from ultrasonography associated with early TA are long segmental lesions with homogeneous, midechoic, circumferential arterial wall thickening of the common carotid artery.[23] ^{18}F-FDG PET has also been recently reported to be useful in the diagnosis of early TA.[8–10] ^{18}F-FDG PET can directly identify the distribution and the activity of the inflammation in the aorta and its main branches. ^{18}F-FDG PET coregistered with enhanced CT is a more appropriate diagnostic tool than only ^{18}F-FDG PET in cases in which ^{18}F-FDG accumulation is weak.[25] Recent studies have indicated that the inflammation in the atherosclerotic plaque of the aorta and the carotid artery could also be detected by ^{18}F-FDG PET.[26–28] Inflammation is an important risk factor for atherosclerosis[29] and vasculitis might be a serious background for secondary atherosclerosis. Indeed, coexisting atherosclerotic changes were observed in some cases of TA, consistent with our present case.[2,30,31] Therefore, in the diagnosis of TA, ^{18}F-FDG accumulations in PET should be carefully estimated together with clinical symptoms, inflammatory markers, and findings from other imaging techniques.

In the present case, the differential diagnosis included other causes of large vessel vasculitis such as giant cell arteritis, syphilis, tuberculosis, Behçet's disease, and Kawasaki disease, in addition to atherosclerosis. Based on the findings from laboratory data, clinical symptoms, and specific features of carotid ultrasonography and PET/CT, the diagnosis of TA could be made without difficulty. TA has two phases during disease progression; the acute active phase with arterial wall thickening, and the chronic inactive phase with occlusive luminal changes.[5,6] Based on the findings from ultrasonography, ^{18}F-FDG PET combined with CT, and angiography, we cannot rule out the possibility that, in the present case, the recurrent active phase might be superimposed over the chronic inactive phase with occlusive luminal changes. Steroid therapy has been expected to change the course of the disease if started early. However, steroid treatment may encourage the development of atherosclerosis, because it alters lipid metabolism, glucose metabolism, coagulation fibrinolytic system, and blood pressure. In some cases of TA, arteritis and atherosclerosis have been reported to coexist in the vessel wall.[2,30–32] Therefore, steroid treatment should be carefully employed, particularly in TA patients with high risk of atherosclerosis.

In conclusion, this unusual case of TA presenting acute myocardial infarction could be promptly diagnosed by ultrasonography and ^{18}F-FDG PET/CT. We should pay attention to the underlying disease even in middle-aged or older male patients with acute myocardial infarction.

要　約

急性心筋梗塞発症を契機に診断された高安動脈炎の1例: 頸動脈エコーおよび陽電子放射線断層撮影/コンピューター断層撮影の有用性

宮川浩太郎　白石　淳　那須　道高　鳥居さゆ希
有原　正泰　兵庫　匡幸　八木　孝和　島　孝友
岡田　隆　河野　義雄　松原　弘明

症例は55歳，男性．前胸部痛，左側頸部違和感を主訴に救急搬送された．急性心筋梗塞の診断のもと冠動脈造影を施行し，右冠動脈中間部の閉塞に対してステントを留置し再灌流に成功した．入院経過中に炎症反応の上昇が遷延し，左側頸部違和感が持続していたことから頸動脈エコーを施行したところ，左総頸動脈のび漫性，全周性の壁肥厚を認めた．加えて陽電子放射線断層撮影/コンピューター断層撮影(PET/CT)で左総頸動脈および上行大動脈の壁に一致して集積を認めたことから，高安動脈炎と診断した．3ヵ月後の冠動脈造影では再狭窄は認められず，大動脈造影により

左総頸動脈のび漫性狭窄，左鎖骨下動脈の狭窄，腹部大動脈の一部狭小化を確認した．急性心筋梗塞発症を契機に診断されたまれな高安動脈炎の1例を経験した．同疾患の診断に頸動脈エコーおよびPET/CTが有用であった．加えて中年男性の急性心筋梗塞症例においても，基礎疾患の存在について留意する必要性が示唆された．

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