Case Report

Detection of yellow plaque by near-infrared spectroscopy – Comparison with coronary angioscopy in a case of no-flow phenomenon during coronary intervention

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ABSTRACT

Yellow plaques detected by coronary angioscopy have been regarded as vulnerable plaques and associated with distal embolization or slow/no-flow phenomenon during coronary intervention. This is the first report that compared the findings of angioscopy and near-infrared spectroscopy (NIRS) in a patient who suffered no-flow phenomenon during coronary intervention.

A 41-year-old male patient with silent myocardial ischemia received coronary intervention. Coronary angiogram revealed diffuse stenosis in the distal right coronary artery. Target lesion was examined by NIRS and angioscopy. NIRS can detect lipid core plaque, which is presented as an yellow area in contrast to the normal red area. Target segment was filled with lipid core plaques. On the other hand, angioscopy revealed a ruptured yellow plaque with a thrombus in the target segment. The distribution of yellow plaques detected by angioscopy appeared well corresponded to the yellow areas detected by NIRS. After the insertion of filter-type distal protection device, balloon pre-dilatation and stent implantation were performed. Then, no-flow phenomenon occurred. Coronary flow was finally recovered in the protected vessel but was still disturbed in the non-protected vessel. The filter was filled with much plaque debris.

The correlation between the yellow area detected by NIRS and the yellow plaques detected by angioscopy appeared very well.

Learning objective: The correlation between the yellow area detected by near-infrared spectroscopy (NIRS) and the yellow plaques detected by angioscopy appeared very well. As well as yellow plaque detected by angioscopy, lipid core plaque as shown in yellow area by NIRS may also be associated with future event of acute coronary syndrome and distal embolization during coronary intervention.

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Introduction

Yellow plaques detected by coronary angioscopy have been regarded as vulnerable plaques [1,2] and have been associated with future events of acute coronary syndrome [3] and distal embolization or slow/no-flow phenomenon during coronary intervention [4,5]. However, the angioscopic examination is not always easy in daily practice. We would like to present a case in which no-flow phenomenon occurred during coronary intervention of the target lesion that had yellow plaque by angioscopy and lipid core plaque by near-infrared spectroscopy (NIRS) to compare the distribution of plaques by those examinations. This is the first report that compared the findings of angioscopy and NIRS.
Case report

A 41-year-old male patient with silent myocardial ischemia received coronary intervention. He had been treated for diabetes mellitus and hypercholesterolemia and was treated by insulin and taking pitavastatin (4 mg/day), metformin (500 mg/day), aspirin (100 mg/day), and clopidogrel (75 mg/day). The serum level of low-density lipoprotein cholesterol was 135 mg/dL; high-density lipoprotein cholesterol was 36 mg/dL; hemoglobin A1c was 9.1%; and C-reactive protein was 0.07 mg/dL. Coronary angiogram revealed diffuse stenosis in the distal right coronary artery (Fig. 1).

The target lesion was examined by a new device that combined NIRS with intravascular ultrasound (IVUS) (TVC system, InfraReDx, Boston, MA, USA) and angioscopy. NIRS can detect lipid core plaque, which is presented as an yellow area in contrast to the normal area shown in red. The result of NIRS is presented as a red/yellow circle around the IVUS image (Fig. 2A) or as a chemogram (Fig. 2B). The target segment was filled with a large amount of lipid core plaque. The amount can be measured by lipid core burden index (LCBI), and the maximum LCBI of a 4-mm segment (maxLCBI_{4\text{mm}}) in this case was 1000. On the other hand, angioscopy revealed the presence of a ruptured yellow plaque with a thrombus in the target segment (Fig. 3). The distribution of yellow plaques detected by angioscopy appeared well corresponded to the yellow areas with NIRS: two large plaques (#2 and #4 in Figs. 2 and 3) were separated by relatively normal segment (#3 in Figs. 2 and 3) in both examinations. More precisely, the comparison of the images between angioscopy and NIRS is shown in Fig. 4. Longitudinally, two large yellow plaques were separated by a short normal segment in both images. However, the presence of the thrombus could not be judged by NIRS as it does not have the algorithm to detect the thrombus. Because the circumferential image was not acquired by angioscopy, circumferential distribution of the yellow plaque could not be compared between angioscopy and NIRS.

After the insertion of a filter-type distal protection device (Filtrap, Japan Lifeline, Tokyo, Japan), balloon pre-dilatation was performed and 3.5 mm × 23 mm and 3.0 mm × 38 mm stents (Xience Prime, Abbott Vascular Japan, Tokyo, Japan) were implanted. No-flow phenomenon was detected after balloon pre-dilatation (Fig. 5A) and was still observed after stent implantation (Fig. 5B). Coronary flow was only partially recovered after the filter removal (Fig. 6A), which recovered in the final angiogram (Fig. 6B) after repeated injection of nicorandil in the protected vessel (yellow arrow) but was still disturbed in the non-protected vessel (red arrow). The filter was filled with much plaque debris (Fig. 7A), which included the thrombus, foamy cells, cholesterol crystals, collagen fibers, and calcifications by histological examination (Fig. 7B).

Fig. 1. Coronary angiogram of the right coronary artery. Coronary angiogram [(A) left anterior oblique view and (B) cranial view] revealed diffuse stenosis (red line with arrows) in the distal right coronary artery.

Fig. 2. Findings of near-infrared spectroscopy (NIRS). NIRS can detect lipid core plaque, which is presented as an yellow area in contrast to the normal area shown in red. The result of NIRS is presented as a red/yellow circle around the intravascular ultrasound image (A) or as a chemogram (B). The amount of lipid core plaque can be measured by lipid core burden index (LCBI), and the maximum LCBI of 4-mm segment (maxLCBI_{4\text{mm}}) in this case was 1000. The distribution of yellow plaques detected by angioscopy appeared well correlated with the yellow areas with NIRS: two large plaques (2 and 4) were separated by relatively normal segment (3) in both examinations (see Fig. 3).

Fig. 3. Findings of angioscopy. Angioscopy revealed the presence of a ruptured yellow plaque with a thrombus in the target segment. The distribution of yellow plaques detected by angioscopy appeared well correlated with the yellow areas with near-infrared spectroscopy: two large plaques (2 and 4) were separated by relatively normal segment (3) in both examinations (see Fig. 2).
Fig. 4. Comparison between angioscopy and near-infrared spectroscopy (NIRS). Longitudinally, two large yellow plaques were separated by a very short normal segment both by angioscopy and NIRS. However, the presence of the thrombus could not be judged by NIRS as it does not have the algorithm to detect it. Because the circumferential image was not acquired by angioscopy, further detailed comparison of the images could not be performed.

Fig. 5. No-flow phenomenon after intervention. No-flow phenomenon was detected after balloon pre-dilatation (A) and was still observed after stent implantation (B). POBA, plain old balloon angioplasty.

Fig. 6. Coronary angiogram after the removal of filter. Coronary flow only partially recovered after the filter removal (A), which recovered in the final angiogram (B) after repeated injection of nicorandil in the protected vessel (yellow arrow) but was still disturbed in the non-protected vessel (red arrow).

Fig. 7. The filter device removed from the coronary artery with no-flow phenomenon. (A) The filter was filled with much plaque debris, which caused filter no-flow but protected the vessel from real no-flow phenomenon in the vessel (yellow arrow in Fig. 6). (B) The captured material included the thrombus, foamy cells, cholesterol crystals, collagen fibers, and calcifications on histological examination.

The patient suffered periprocedural myocardial infarction with the peak serum creatine kinase level of 1500 U/L.

Discussion

Although the risk of slow/no-flow phenomenon has not been clarified and the beneficial effect of distal protection has not been established [6], we have previously reported that distal protection is associated with a lower incidence of slow-flow phenomenon and with better cardiac function in patients with ruptured yellow plaque at the culprit lesion of acute myocardial infarction [4]. Our recent report also revealed that the presence of ruptured plaque or yellow plaque is associated with distal embolization of plaque debris both in acute coronary syndrome and in stable coronary heart disease patients [5]. As maxLCBlum ≥ 500 has been associated with the high incidence (50%) of periprocedural myocardial infarction in a previous report [7], the present case had maxLCBlum of 1000 and suffered no-flow phenomenon and periprocedural myocardial infarction.

The correlation between the yellow area detected by NIRS and the yellow plaques detected by angioscopy appeared very well in the present case regarding its longitudinal distribution. However, the circumferential distribution of yellow plaques could not be compared, because circumferential image was not acquired by angioscopy. Further investigations would establish the correlation between the yellow plaques by angioscopy and the yellow area by
NIRS. Although NIRS cannot detect the thrombus or morphological information of the plaques, it can evaluate the plaques quantitatively and may contribute to the investigations of vulnerable plaques and to the risk stratification of patients.

**Conclusion**

The correlation between the yellow area detected by NIRS and the yellow plaques detected by angioscopy appeared very well.

**Conflict of interest**

The authors declare no conflict of interest.

**References**


