Positron Emission Tomography (PET)

# Coronary, aortic and carotid artery inflammation by 18F-fluorodeoxyglucose positron emission tomography in acute and chronic coronary artery disease

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**Background:** 18F-fluorodeoxyglucose (18F-FDG) positron emission tomography (PET) can detect arterial inflammation in individuals with atherosclerosis, but the associations among different vascular territories for 18F-FDG uptake are not known.

**Purpose:** We explored any possible correlation between arterial inflammation quantified by 18F-FDG PET in the aorta, carotid arteries, and coronary arteries in patients presenting with acute coronary syndrome (ACS), or chronic coronary artery disease (CAD).

**Methods:** Prospectively, we performed hybrid computed tomography angiography and 18F-FDG PET in 43 patients (26 ACS and 17 chronic CAD) at 6.6 ± 5.7 days following invasive coronary angiography. 18F-FDG PET was performed 90 minutes after injection of 302.2 ± 28.4 MBq 18F-FDG. Arterial 18F-FDG uptake was measured in the thoracic aorta, carotid arteries, and coronary arteries, and expressed as the target-to-background ratio (TBR; the ratio between arterial maximal standardized uptake value normalized to blood pool mean standardized uptake value) in the whole artery, and in the most diseased segment (MDS).

**Results:** Mean age was 64.9 ± 9.1 years, 90.7% males. The whole artery 18F-FDG uptake was higher in the aorta than in the carotid arteries (median TBR 2.23, interquartile range [0.36] vs. 1.88 [0.42], $p < 0.001$); whereas uptake in the coronary arteries was lower than in the aorta or carotid arteries (1.13 [0.23], $p < 0.001$ both). Similarly, 18F-FDG uptake in the aortic MDS was higher than in the carotid MDS (2.75 [0.62] vs. 2.25 [0.63], $p < 0.001$); whereas 18F-FDG uptake in the coronary MDS was the lowest (1.40 [0.33], $p < 0.001$ both). These findings were consistent in both ACS and chronic CAD patients. The whole artery 18F-FDG uptake of the aorta and carotid arteries correlated in patients with ACS ($r = 0.58$, $p = 0.002$), but not in patients with chronic CAD ($r = 0.21$, $p = 0.3$). There was no correlation between the whole artery 18F-FDG uptake in the coronary arteries and either the aorta or carotid arteries in the whole cohort ($r=-0.16$, $p = 0.2$, $r = 0.01$, $p = 0.9$, respectively), in patients with ACS ($r = 0.06$, $p = 0.7$, $r=-0.01$, $p = 0.9$, respectively), or in those with chronic CAD ($r=-0.4$, $p = 0.1$, $r=-0.09$, $p = 0.7$, respectively).

**Conclusions:** In patients with ACS or chronic CAD, large arteries had higher 18F-FDG uptake than the coronary arteries. The intensity of 18F-FDG uptake in the coronary arteries did not correlate with that in the carotid arteries or the aorta, indicating that disease activity differs between large arteries and coronary arteries.

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