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Editorial

Seeing is believing – Imaging of a plaque in the renal artery

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Recently, it has become clear that inter-organ communication by way of the autonomic nervous system is necessary to maintain the highly organized orchestra of metabolic and cardiovascular systems [1].

One such case is the renal sympathetic nerves, which have been identified as a major contributor to the complex pathophysiology of hypertension. Patients with essential hypertension have increased efferent sympathetic drive to the kidneys and increased rate of sympathetic-nerve firing modulated by afferent signaling from renal sensory nerves [2–4]. Thus, the renal nerve ablation (RNA) technique using a radiofrequency (RF) catheter has been developed to cure severe, drug-resistant, hypertension. This procedure resulted in a 42% decrease in whole-body norepinephrine spillover and a substantial reduction in blood pressure and sustained normalization of sympathetic-nerve firing rates with a reduction in left ventricular mass during the follow-up period [5–7].

Despite initial studies showing the safety and efficacy of this procedure, recent clinical studies suggested the possibility of vascular impairments after catheter-based intervention. Templin et al. evaluated morphological features before and after RNA by optical coherence tomography (OCT) [8]. OCT has been established as an intracoronary imaging tool for visualizing stent apposition and healing, plaque characterization, thrombus formation, and dissection with a high spatial resolution of 10–15 μm. Thanks to its high resolution, OCT is able to detect local vascular injuries that are angiographically undetectable. They observed diffuse renal artery constriction and local tissue damage at the ablation site with edema and thrombus formation after RNA. Similar findings were also obtained in a porcine model of RNA, and acute loss of endothelialization resulted in thrombus formation [9]. These findings suggest that antiplatelet therapy is definitely required during RNA.

Angioscopy is an endoscopic technique that allows direct visualization of the internal surface of a vessel and provides information regarding plaque morphology and the thrombus in detail in patients with a high resolution (10–50 μm). Ulcerlation, fissures, and tears on plaques can be observed and clearly demarcated from the adjacent vessel wall. There have been many reports about angioscopy of coronary arteries. A normal coronary artery appears as glistening white, whereas atherosclerotic plaque can be categorized as yellow-white. The intensity of the yellow color is also an indicator of fibrous cap thickness, with high yellow intensity associated with thin, fibrous caps overlaying a lipid core. The yellow intensity of the plaque can be classified semiquantitatively [10–12].

In their report, Komatsu et al. analyzed atherosclerosis in a renal artery by several modalities [13]. Computed tomography, renal angiography, and intravascular ultrasound also revealed the existence of a lipid-rich component and fibrous to calcified components in the stenotic region. They observed the plaque by angioscopy after percutaneous transluminal renal angioscopy and found one grade 3 yellow plaque at the proximal end of the stent and grade 1 and 2 plaques in the middle and distal portions of the artery. They discussed the potential implications of angioscopy to evaluate vascular injuries after RNA.

There are several limitations to angioscopy. First, it observes only the surface of the arterial lumen. Second, it is impossible to evaluate angioscopic findings fully quantitatively. Thus, methods of image quantification, such as quantitative colorimetric analysis, have been developed to overcome the limitations inherent in angioscopic images. However, angioscopic analysis has the advantage to detect the thrombus more sensitively than other imaging modalities and it may offer the opportunity for observations by angioscopy after RNA.

It may be necessary to reduce radiofrequency energy levels during RNA to reduce vascular injury. Currently, we cannot select ablation sites during RNA. However, if we can select and ablate only at the healthy vascular lumen without the plaque, it may be possible to reduce the radiofrequency energy. Multi-modalities for vascular imaging including angioscopy may be used for this purpose in the future.

References

[1] Izumida Y, Yahagi N, Takeuchi Y, Nishi M, Shikama A, Takarada A, Masuda Y, Kubota M, Matsuzaka T, Nakagawa Y, Iizuka Y, Itaka K, Kataoka K, Shioda S, Niijima A, et al. Glycogen shortage during fasting triggers liver–brain–adipose neurocircuitry to facilitate fat utilization. Nat Commun 2013;4:2316.
[2] Esler M, Jennings G, Lambert G. Noradrenaline release and the pathophysiology of primary human hypertension. Am J Hypertens 1989;2:140S–6S.
[3] Converse Jr RL, Jacobsen TN, Toto RD, Jost CM, Cosentino F, Fouad-Tarazi F, Victor RG. Sympathetic overactivity in patients with chronic renal failure. N Engl J Med 1992;327:1912–8.
[4] Schlaich MP, Lambert E, Kaye DM, Krozowski Z, Campbell DJ, Lambert G, Hastings J, Aggarwal A, Esler MD. Sympathetic augmentation in hypertension: role of nerve firing, norepinephrine reuptake, and Angiotensin neuromodulation. Hypertension 2004;43:169–75.
[5] Krum H, Schlaich M, Whitbourn R, Sobotka PA, Sadowski J, Bartus K, Kapelak B, Walton A, Sievert H, Thambar S, Abraham WT, Esler M. Catheter-based renal

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http://dx.doi.org/10.1016/j.jccase.2013.09.011

sympathetic denervation for resistant hypertension: a multicentre safety and proof-of-principle cohort study. Lancet 2009;373:1275–81.
[6] Esler MD, Krum H, Schlaich M, Schmieder RE, Bohm M, Sobotka PA. Renal sympathetic denervation for treatment of drug-resistant hypertension: one-year results from the Symplicity HTN-2 randomized, controlled trial. Circulation 2012;126:2976–82.
[7] Hering D, Lambert EA, Marusic P, Walton AS, Krum H, Lambert GW, Esler MD, Schlaich MP. Substantial reduction in single sympathetic nerve firing after renal denervation in patients with resistant hypertension. Hypertension 2013;61:457–64.
[8] Templin C, Jaguszewski M, Ghadri JR, Sudano I, Gaehwiler R, Hellermann JP, Schoenenberger-Berzins R, Landmesser U, Erne P, Noll G, Lüscher TF. Vascular lesions induced by renal nerve ablation as assessed by optical coherence tomography: pre- and post-procedural comparison with the Simplicity catheter system and the EnligHTN multi-electrode renal denervation catheter. Eur Heart J 2013;34:2141–8.
[9] Steigerwald K, Titova A, Malle C, Kennerknecht E, Jilek C, Hausleiter J, Nährig JM, Laugwitz KL, Joner M. Morphological assessment of renal arteries after radiofrequency catheter-based sympathetic denervation in a porcine model. J Hypertens 2012;30:2230–9.
[10] Kawasaki M, Takatsu H, Noda T, Sano K, Ito Y, Hayakawa K, Tsuchiya K, Arai M, Nishigaki K, Takemura G, Minatoguchi S, Fujiwara T, Fujiwara H. In vivo quantitative tissue characterization of human coronary arterial plaques by use of integrated backscatter intravascular ultrasound and comparison with angioscopic findings. Circulation 2002;105:2487–92.
[11] Kawano T, Honye J, Takayama T, Yokoyama S, Chiku M, Ando H, Endo M, Ichikawa M, Ishii N, Watanabe Y, Watanabe I, Saito S. Compositional analysis of angioscopic yellow plaques with intravascular ultrasound radiofrequency data. Int J Cardiol 2008;125:74–8.
[12] Takano M, Jang IK, Inami S, Yamamoto M, Murakami D, Okamatsu K, Seimiya K, Ohba T, Mizuno K. In vivo comparison of optical coherence tomography and angioscopy for the evaluation of coronary plaque characteristics. Am J Cardiol 2008;101:471–6.
[13] Komatsu S, Ohara T, Takewa M, Takahashi S, Nomamoto T, Kamata T, Nishiuchi K, Kobayashi Y, Kodama K. Angioscopy in patients with atherosclerotic renal artery stenosis. JC Cases 2013, http://dx.doi.org/10.1016/j.jccase.2013.08.014.

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13 September 2013