Prevention of Radial Artery Occlusion After Transradial Catheterization

Factors influencing radial artery occlusion and different methods for its prevention.

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Coronary interventions utilizing the transradial approach (TRA) are increasingly popular worldwide.[1] At the expense of a slightly more technically demanding procedure, which necessitates operator experience, TRA can lead to reductions in bleeding, vascular complications, and patient discomfort. TRA potentially improves prognosis as compared with the transfemoral approach (TFA) due to a lower risk of vascular complications and bleeding, which have an important impact on morbidity, mortality, and ultimately, on cost of care.[2-5]

BACKGROUND

Despite its many benefits, access site–related complications may occur with TRA, although their effect is usually less detrimental as compared to those associated with TFA. The most common complication is radial artery occlusion (RAO), with a reported incidence rate ranging from < 1% to > 30%.[6-8] This variability in reported incidence rates is due to several reasons, especially to the techniques used during and after the procedure. RAO is usually clinically silent due to a dual blood supply to the hand and often goes unnoticed. Therefore, the reported incidence may be accurate only if it is systematically assessed. It has been reported that > 50% of operators simply do not check radial artery patency before discharge.[1,8] Other factors, such as timing and method of assessment of RAO, become relevant when the possibility of RAO is not overlooked. The pathogenesis of RAO is believed to be the result of vascular damage and endothelial injury at the puncture site, which causes a prothrombotic milieu that consequently leads to thrombus formation, neointimal proliferation, and finally occlusion. Although the occlusion itself usually occurs shortly after the procedure, patency rates increase later as a result of spontaneous recanalization of the artery within 1 to 3 months; thus, the timing of assessment will affect the rates of RAO.[9] Doppler ultrasound is considered to be the gold standard for assessing postprocedural radial artery patency. The reverse Barbeau test is a simpler, more accessible method in which waveforms are assessed by pulse oximetry during manual compression of the ulnar artery to evaluate radial artery patency. The accuracy of these two tests has been shown to be quite similar.[7] It is important to acknowledge that occlusion may be present even if the radial pulse is palpable after the procedure; this is due to a developed network of collaterals and retrograde filling of the radial artery.[10]

Although RAO is usually clinically quiescent, it rarely may result in ischemic symptoms of the hand or digits.[11] In the RIVAL trial, symptomatic RAO requiring medical attention occurred in only 0.2% of patients.[12] RAO precludes the use of the radial artery for any future cardiac catheterizations or other procedures, such as hemodialysis shunt anastomosis or as a graft during coronary artery bypass graft surgery. Thus, the patency of the radial artery is of importance.

INFLUENCING FACTORS

A few factors influencing RAO occurrence have been recognized and are subsequently described.

Catheter or Sheath-to-Artery Ratio

The detrimental combination of bigger catheter and sheath sizes with small-caliber arteries is well recognized as a contributing factor to vascular damage and the creation of a prothrombotic environment.[13] Using a 5-F system as opposed to a 6-F system is associated with a marked reduction in the incidence of RAO.[14,15] One study demonstrated that the incidence of RAO was 13.7% when using a 5-F system compared with 30.5% when using a 6-F system ($P < .001$).[14] Whereas a 6-F sheath has an inner diameter of only 1.98 mm, its outer diameter varies between the different manufacturers but may reach 2.8 mm (which is 8.4 F). Newer slender sheaths with thin walls have been designed, resulting in smaller outer diameters (2.46 mm) (Glidesheath Slender, Terumo Interventional Systems). The sheath size used is important because it has been demonstrated that 20% of men and 40% of women had a radial artery inner diameter that was smaller than or equal to the outer diameter of a standard 6-F sheath.[16] Accordingly, it is not surprising that the proportion of postprocedural severe radial artery blood flow reduction was significantly higher when the outer sheath diameter was larger than the inner artery diameter.[14] Although the radial artery has some elasticity, maintaining a sheath-to-artery ratio < 1 is an essential factor in preventing RAO. Therefore, it is recommended that smaller-size sheaths be used whenever possible.[9] A routine use of ultrasound for assessment of radial artery size and anatomy may prove to be useful, although current evidence to support this is lacking.[17,18]

Anticoagulation

Because thrombogenicity is the cornerstone of RAO development, administration of anticoagulants during the procedure is common and has been shown to reduce the incidence of RAO.[11,13] The administration may be done directly into the radial artery itself or intravenously.[6,19] A dose-dependent effect of unfractionated heparin had been demonstrated.[6,20] A recent meta-analysis has found that a higher heparin dose is associated with reduced RAO rates as compared to a lower dose.[8] However, other recent studies have not demonstrated this benefit.[21,22] All studies to date are relatively small. In fact, there are few data regarding the use of heparin in contemporary practice, and in the setting of achieving patent hemostasis, heparin may be used only as a provisional strategy.[23]

Vasodilators

Another potential factor that may contribute to endothelial injury includes radial artery spasm, which results in friction between the radial artery and devices used during TRA. Intra-arterial nitroglycerin administration before sheath removal was associated with reduced incidence of RAO in a randomized trial of 1,706 patients,[24] supporting the hypothesis that radial artery spasm may contribute to the mechanism of RAO. According to a systematic review, the use of verapamil or verapamil in combination with nitroglycerin is effective in preventing radial artery spasm.[25]

Compression Time

The preferred duration of compression after removal of the radial artery sheath has been investigated. Whereas in the past, prolonged compression as long as 6 hours had been the norm in many centers, shorter-duration compression (ie, 2 hours) was associated with a reduction in RAO without an increase in bleeding, allowing for earlier patient discharge and less discomfort.[5,26] Other nonran-

TABLE 1. STEPS IN THE PATENT HEMOSTASIS PROCESS AFTER TRANSRADIAL PROCEDURES

Step 1 Withdraw the arterial sheath 2–3 cm.
Step 2 Apply the hemostatic compression device 2–3 mm proximal to the skin entry site (at the point of arterial entry), tighten or inflate it, then remove the sheath.
Step 3 Decrease the pressure of the hemostatic compression device to the point of mild pulsatile bleeding at the skin entry site. After two to three cycles of pulsatile bleeding, retighten the hemostatic compression device gradually to eliminate this pulsatile bleeding.
Step 4 Evaluate radial artery patency by using the reverse Barbeau test: ▪ Place the plethysmographic sensor on the index finger of the involved upper extremity with the observation of pulsatile waveforms. ▪ Compress the ulnar artery at the level of the wrist, and observe the behavior of the waveform. ▪ Absence of plethysmographic waveform is indicative of interruption of radial artery flow. ▪ If interruption occurs, the hemostatic compression pressure should be lowered to the point where the plethysmographic waveform returns. This is evidence of antegrade radial artery flow.

Modified with permission from Rao SV, Tremmel JA, Gilchrist IC, et al. Best practices for transradial angiography and intervention: a consensus statement from the Society for Cardiovascular Angiography and Intervention's Transradial Working Group. Catheter Cardiovasc Interv. 2014;83:228-236.

domized trials have tried to show that even shorter compression durations were achievable.[27,28] In the PRACTICAL trial, we were able to achieve hemostasis with a very low RAO rate of 2.8% immediately after clamp removal and < 1% after a week, with a 60-minute compression time. An ultra-short compression duration of only 20 minutes led to a higher rate of bleeding without further reduction in the incidence of RAO.[29] It should be noted that many other studies evaluated RAO in patients undergoing only diagnostic angiography or using smaller introducer sheaths. Our study was performed in patients undergoing diagnostic cardiac catheterization or percutaneous coronary intervention, with either a 5- or 6-F sheath, while administering a standardized dose of heparin. Therefore, in all-comers, a compression duration of 60 minutes seems to be one of the most powerful methods to reduce RAO.

Bleeding or hematoma developing during clamp release, in combination with ultra-short compression in the setting of anticoagulation use, resulted in a need to retighten the clamp, which in turn is associated with higher incidence of RAO.

Patent Hemostasis

Hemostasis can easily be achieved by compression of the radial artery; however, if compression leads to complete cessation of blood flow through the artery, thrombus may form and eventually lead to RAO. The concept of patent hemostasis is applying the amount of pressure required to prevent bleeding from the puncture site while maintaining antegrade blood flow in the radial artery. Patent hemostasis can be achieved in a few steps (Table 1 and Figure 1).[30]

In some patients, it is not possible to achieve patent hemostasis because the release of the clamp to allow antegrade flow may cause bleeding. Achieving patent hemostasis requires dedication and attention to detail, and it is likely that many operators do not perform it accurately.

In the PROPHET trial, 436 consecutive patients were randomized to conventional compression or patent hemostasis. Radial artery patency was assessed using the reverse Barbeau test. Patent hemostasis was associated with a reduced incidence of RAO of 5% versus 12% in the conventional arm ($P < .05$) when assessed at 24 hours and 1.8% versus 7% ($P < .05$) at 30 days.[8] The PHARAOH trial compared the safety and efficacy of patent hemostasis compared with standard a priori heparin administration after radial sheath introduction. In the patent hemostasis group, heparin was given only if patent hemostasis was not achieved. The incidence of RAO was similar in the two groups at early (24 hours; 7.5% vs 7%; $P = .84$) and late (30 days; 4.5% vs 5%; $P = .83$) evaluations.[22] These results emphasize the importance of maintaining radial artery patency during hemostasis but perhaps suggest that there is no need for heparin if patent hemostasis is achieved. A new approach for maintaining radial artery patency is compression of the ulnar artery after sheath removal with an aim of inducing hyperperfusion of the radial artery. These methods have been evaluated in the PROPHET II trial; in 3,000 randomized patients, prophylactic ulnar compression reduced the incidence of RAO at 30 days from 3% to 0.9%, without increasing hand ischemia.[31]

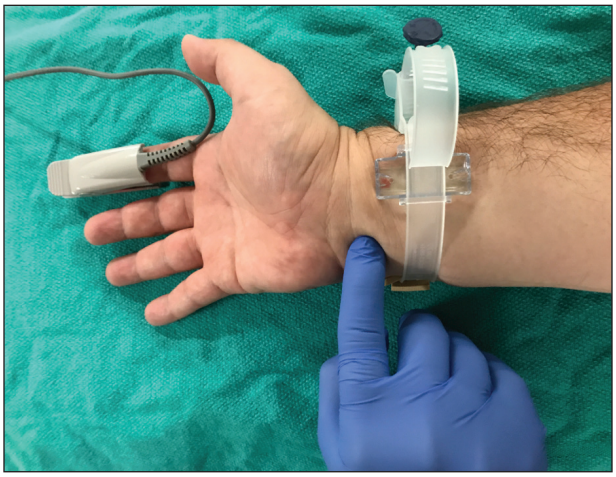

Figure 1. Reverse Barbeau test for assessment of patent hemostasis.

CONCLUSION

The most evident strength of TRA is the ability to lower the rates of clinically significant bleeding due to a more controlled and better visualized hemostasis. Although not very common, significant bleeding may occur and can cause large hematomas or, rarely, compartment syndrome.[2,10] A delicate balance exists between the different influencing factors. Anticoagulants, patent hemostasis, and reduced compression duration may reduce the incidence of RAO but may cause bleeding, which in turn may lead to increased rates of RAO. To achieve the best possible results, the treating physician must assess where each patient is located along the continuum of thrombogenesis and bleeding and integrate different methods for optimal patient care. ■

1. Bertrand OF, Rao SV, Pancholy S, et al. Transradial approach for coronary angiography and interventions: results of the first international transradial practice survey. JACC Cardiovasc Interv. 2010;3:1022-1031.
2. Bradley SM, Rao SV, Curtis JP, et al. Change in hospital-level use of transradial percutaneous coronary intervention and periprocedural outcomes: insights from the national cardiovascular data registry. Circ Cardiovasc Qual Outcomes. 2014;7:550-559.
3. Jolly SS, Mehta SR. Coronary intervention: radial artery access comes of age. Lancet. 2015;385:2437-2439.
4. Valgimigli M, Gagnor A, Calabro P, et al. Radial versus femoral access in patients with acute coronary syndromes undergoing invasive management: a randomised multicentre trial. Lancet. 2015;385:2465-2476.
5. Bertrand OF, De Larochelliere R, Rodes-Cabau J, et al. A randomized study comparing same-day home discharge and abciximab bolus only to overnight hospitalization and abciximab bolus and infusion after transradial coronary stent implantation. Circulation. 2006;114:2636-2643.
6. Pancholy SB. Strategies to prevent radial artery occlusion after transradial PCI. Curr Cardiol Rep. 2014;16:505.

7. Pancholy S, Coppola J, Patel T, et al. Prevention of radial artery occlusion-patent hemostasis evaluation trial (PROPHET study): a randomized comparison of traditional versus patency documented hemostasis after transradial catheterization. Catheter Cardiovasc Interv. 2008;72:335-340.
8. Rashid M, Kwok CS, Pancholy S, et al. Radial artery occlusion after transradial interventions: a systematic review and meta-analysis. J Am Heart Assoc. 2016;5.
9. Avdikos G, Karatasakis A, Tsoumeleas A, et al. Radial artery occlusion after transradial coronary catheterization. Cardiovasc Diagon Ther. 2017;7:305-316.
10. Yoo BS, Yoon J, Ko JY, et al. Anatomical consideration of the radial artery for transradial coronary procedures: arterial diameter, branching anomaly and vessel tortuosity. Int J Cardiol 2005;101:421-427.
11. Rao SV, Bernat I, Bertand OF. Clinical update: remaining challenges and opportunities for improvement in percutaneous transradial coronary procedures. Eur Heart J. 2012;33:2521-2526.
12. Jolly SS, Yusuf S, Cairns J, et al. Radial versus femoral access for coronary angiography and intervention in patients with acute coronary syndromes (RIVAL): a randomised, parallel group, multicentre trial. Lancet. 2011;377:1409-1420.
13. Wagener JF, Rao SV. Radial artery occlusion after transradial approach to cardiac catheterization. Curr Atheroscler Rep. 2015;17:489.
14. Uhlemann M, Mobius-Winkler S, Mende M, et al. The Leipzig prospective vascular ultrasound registry in radial artery catheterization: impact of sheath size on vascular complications. JACC Cardiovasc Interv. 2012;5:36-43.
15. Dahm JB, Vogelgesang D, Hummel A, et al. A randomized trial of 5 vs. 6 French transradial percutaneous coronary interventions. Catheter Cardiovasc Interv. 2002;57:172-176.
16. Saito S, Ikei H, Hosokawa G, Tanaka S. Influence of the ratio between radial artery inner diameter and sheath outer diameter on radial artery flow after transradial coronary intervention. Catheter Cardiovasc Interv. 1999;46:173-178.
17. Chugh SK, Chugh S, Chugh Y, et al. Feasibility and utility of pre-procedure ultrasound imaging of the arm to facilitate transradial coronary diagnostic and interventional procedures (PRIMAFACIE-TRI). Catheter Cardiovasc Interv. 2013;82:64-73.
18. Camuglia AC, Majed M, Preston SD, et al. Ultrasound guidance for vascular access in patients undergoing coronary angiography via the transradial approach. J Invasive Cardiol. 2015;27:163-166.
19. Pancholy SB. Comparison of the effect of intra-arterial versus intravenous heparin on radial artery occlusion after transradial catheterization. Am J Cardiol. 2009;104:1083-1085.
20. Spaulding C, Lefèvre T, Funck F, et al. Left radial approach for coronary angiography: results of a prospective study. Cathet Cardiovasc Diagn. 1996;39:365-370.
21. Degirmencioglu A, Buturak A, Zencirci E, et al. Comparison of effects of low- versus high-dose heparin on access-site complications during transradial coronary angiography: a double-blind randomized study. Cardiology. 2015;131:142-148.
22. Hahalis G, Xathopoulou I, Tsigkas G, et al. A comparison of low versus standard heparin dose for prevention of forearm artery occlusion after 5 French coronary angiography. Int J Cardiol. 2015;187:404-410.
23. Pancholy SB, Bertrand OF, Patel T. Comparison of a priori versus provisional heparin therapy on radial artery occlusion after transradial coronary angiography and patent hemostasis (from the PHARAOH Study). Am J Cardiol. 2012;110:173-176.
24. Dharma S, Kedev S, Patel T, et al. A novel approach to reduce radial artery occlusion after transradial catheterization: postprocedural/prehemostasis intra-arterial nitroglycerin. Catheter Cardiovasc Interv. 2015;85:818-825.
25. Kwok CS, Rashid M, Fraser D, et al. Intra-arterial vasodilators to prevent radial artery spasm: a systematic review and pooled analysis of clinical studies. Cardiovasc Revasc Med. 2015;16:484-490.
26. Pancholy SB, Patel TM. Effect of duration of hemostatic compression on radial artery occlusion after transradial access. Catheter Cardiovasc Interv. 2012;79:78-81.
27. Edris A, Gordin J, Sallam T, et al. Facilitated patent haemostasis after transradial catheterisation to reduce radial artery occlusion. EuroIntervention. 2015;11:765-771.
28. Amin H. Prevention of radial artery occlusion: it's the right thing to do. EuroIntervention. 2015;11:731-733.
29. Lavi S, Cheema A, Yadegari A, et al. Randomized trial of compression duration after transradial cardiac catheterization and intervention. J Am Heart Assoc. 2017;6.
30. Rao SV, Tremmel JA, Gilchrist IC, et al. Best practices for transradial angiography and intervention: a consensus statement from the Society for Cardiovascular Angiography and Intervention's Transradial Working Group. Catheter Cardiovasc Interv. 2014;83:228-236.
31. Pancholy SB, Bernat I, Bertrand OF, et al. Prevention of radial artery occlusion: prophylactic hyperperfusion evaluation trial. JACC Cardiovasc Interv. 2016;9:1992-1999.

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