Preventing Radial Artery Occlusion

A description of the causes of radial artery occlusion and best practices to avoid this common complication of transradial access.

BY JOHN T. COPPOLA, MD, FACC

Traditionally, in the United States, percutaneous coronary interventions (PCIs) have been performed using a transfemoral approach. The realization that bleeding complications are associated with both short- and long-term morbidity and mortality has led to an increased interest in strategies that reduce the bleeding risk. This has led to the use of smaller sheaths and catheters for interventional procedures, earlier sheath removal, and the use of ultrasound and x-ray imaging to optimize femoral access, along with the use of different anticoagulation regimens.[1]

Multiple randomized studies have shown a consistent reduction in bleeding with transradial access for diagnostic and interventional procedures. A meta-analysis of 23 studies comparing femoral to radial access found a 78% reduction in bleeding with the use of radial access,[2] leading to a shift in practice patterns in the United States. A recent review of the National Cardiovascular Data Registry showed an increase in radial access for intervention from 1.2% in the first quarter of 2007 to 16.1% in the third quarter of 2012.[3] After performing a multivariable adjustment, radial PCI was associated with lower risk of bleeding (adjusted odds ratio, 0.51; 95% confidence interval, 0.49–0.54). These data continue to fuel the rapid increase in transradial procedures.

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INCIDENCE OF RADIAL ARTERY OCCLUSION

Unfortunately, no procedure is without complications, and the most common complication of radial access is asymptomatic radial artery occlusion (RAO). RAO renders the radial artery unusable for repeat procedures or for use as a bypass graft. To date, only two cases of hand ischemia and one case of chronic regional pain syndrome have been reported after radial artery occlusion.[4,5] In the RIVAL trial, only 0.2% of RAOs required medical attention. The incidence of RAO varies from 1% to 10%, with the range usually reported of 2% to 8%.[6-8]

PATHOGENESIS

The etiology of RAO is thrombus, as demonstrated by Patel et al.[9] Thrombus formation is seen with the triad of vascular injury, stasis, and a hypercoagulable state; prevention of RAO requires attention to all three components. In an ultrasound study of patients undergoing radial artery procedures, Wakeyama et al compared images obtained in first-time radial access cases and compared them to patients who had returned for a second procedure via the same radial artery.[10] They demonstrated a reduction in radial artery diameter in the distal vessel close to the access site, but not in the proximal radial artery. The change in diameter was due to a marked increase in intimal thickening, resulting from previous radial access. The increased intimal thickening was thought to be a result of healing after injury to the artery at the time of the first procedure.

A study using optical coherence tomography immediately after completion of a transradial intervention showed intimal tears in 67% of patients and medial dis-

section in 35%.[11] Again, the area of injury was more often close to the sheath insertion site and more common in patients undergoing repeat procedures. This study also showed increased intimal thickening in patients undergoing repeat procedures. Injury at the time of insertion is thought to contribute to the decreased endothelial function seen in the radial artery after a transradial procedure. The use of smaller sheaths to decrease radial artery trauma and pretreatment with statins to improve endothelial function showed a decrease in the incidence of RAO in one study.[12] Several studies suggest that a radial artery-to-sheath ratio of < 1 is a risk for occlusion, making it important to use the smallest sheath possible to perform the procedure.[13,14]

One group attempted to use patient variables to help predict radial size.[15] This study suggested that wrist circumference correlates with radial size, and wrist circumference can be predicted by shoe size. Male sex was a marker of a larger artery, and South Asian ancestry correlated with smaller artery size.

ANTICOAGULATION

In 1996, Spaulding et al clearly showed the benefit of heparin use in the prevention of RAO.[16] In this article, the first 49 cases were performed without heparin, and an RAO incidence of 71% was noted. With the use of 2,000 units of heparin (up to 3,000 units, if patient weight was > 80 kg), they noted a decrease in the RAO rate to 24%. When the dose of heparin was increased to 5,000 units, the RAO rate decreased further to 4.3%. A 2010 international survey on the transradial approach to cardiac catheterization and intervention reported that three-quarters of cardiologists worldwide who perform radial procedures use between 2,000 and 5,000 units of heparin.[17]

Bernat et al were able to decrease the 30-day RAO rate to 0.8% with 5,000 units of heparin as compared to 4.3% when 2,000 units was used.[18] The current Society for Cardiac Angiography and Interventions best practice guidelines suggest a heparin dose of 50 units/kg (up to a 5,000-unit maximum dose) for a radial diagnostic procedure.[19] A 500-patient study compared the administration of intravenous heparin to heparin given via the arterial sheath and found no difference in the incidence of RAO, suggesting that the heparin effect is systemic.[20]

To evaluate the contribution of heparin anticoagulation compared to the method used to achieve hemostasis after sheath removal, a 400-patient study was performed. Two hundred patients received the standard heparin dose of 50 units/kg at the time of sheath insertion; in the other group, no heparin was given until the time of sheath removal, and heparin was only given to those patients in whom patent hemostasis could not be achieved at a dose of 50 units/kg up to a maximum dose of 5,000 units. The incidence of RAO was similar in both groups, suggesting that patent hemostasis is the major parameter in the reduction of RAO.[21]

This study was performed by skilled high-volume operators with average procedure times < 10 minutes. This would suggest that in the setting of a cardiac catheterization and possible intervention, a decision on the use of anticoagulation can be made after completing the diagnostic study. If a diagnostic angiogram is only performed, heparin can be given at completion of the procedure. If an intervention will be performed, bivalirudin can be given for the intervention, or heparin can be given after the diagnostic study. One published study of 400 patients were given 70 units/kg of heparin after completion of the diagnostic study prior to sheath removal, or, if an intervention was to be performed, bivalirudin was given as a bolus of 0.75 mg/kg followed by an infusion of 1.75 mg/kg per hour. The overall RAO rate was not significantly different between the heparin or bivalirudin group when measured between 4 and 8 weeks after the procedure.[22]

A common clinical problem is presented by the patient on oral anticoagulation with warfarin and a therapeutic internalized normalized ratio. A case review of patients with internalized normalized ratios of 2 to 4 who were not given heparin yielded an RAO rate of 18.6%, whereas during a similar time frame, an RAO rate of 9.6% was noted in the group receiving heparin.[23]

STASIS

During compression of the radial artery, if occlusive pressure is applied, stasis will occur in a region of recent vascular injury and inflammation, setting the stage for thrombus formation.[24] Pancholy et al showed that if hemostasis can be achieved while still having flow in the radial artery, RAO rates decreased from 12% to 5% at 24 hours. The following method of patent hemostasis can be achieved in close to 75% of all patients. While applying a compression device to the radial artery, hemostasis is achieved; next, while compression of the ulnar artery is performed, pressure is removed from the device until bleeding or return of a pulse wave is seen. In an attempt to have a better idea of the amount of compression required, Cubero et al used the manufacturer's recommended 15 mL of air in the compression band in the control group, and in the study group, they applied enough pressure in the band to reach the mean arterial pressure.[25] The group that had the pressure-guided inflation showed a remarkable reduction in RAO to 1.1%.

Once hemostasis is achieved, another factor is the length of time that the compression device should remain in place. The outcome of prolonged compres-

sion may be RAO, but when compression is removed too early, repeat bleeding can occur. A comparison of 6 hours of compression to 2 hours showed no difference in bleeding but a more than 50% reduction in RAO, with a decrease from 12% to 5.5%.[26] It is currently our practice to start removing the air from the compression device at 1 hour for diagnostic procedures and 2 hours after an intervention with bivalirudin. Kaolin-filled pads were used in an attempt to achieve rapid hemostasis after radial artery procedures (they have been used by the military for a number of years to stop bleeding after trauma). The pad was applied directly to the puncture site, and adhesive tape was used to hold the pad in place tightly for 15 minutes. The tape was removed and the pad held in place for 2 more hours by a Tegaderm dressing (3M). This treatment was compared to standard gauze and an occlusive adhesive dressing in terms of time to hemostasis and RAO. The initial attempt at removal of the standard gauze at 15 minutes was quickly stopped due to a high rate of repeat bleeding. The standard compression arm was then changed to 2 hours. The incidence of RAO in the standard group was 10%; in the kaolin-assisted group, no patients had RAO at 24 hours.[27]

The prevention of RAO starts at the beginning of the procedure with limited radial punctures, use of the smallest possible sheath, and use of adequate anticoagulation. At the completion of the procedure, the key factor seems to be close attention to achieving patent hemostasis and adequate compression time.

Despite attempting to achieve patent hemostasis, this can only be achieved in 75% of patients. If patent hemostasis cannot be achieved initially, after 15 minutes, a small amount of compression can be released from the access site for another attempt to achieve flow. Some operators may extend upon the ulnar occlusion method described for treatment of RAO by placing a compression device on the ulnar artery to force intraosseous collateral to increase radial flow and maintain patency. Bernat et al showed that if the diagnosis of RAO is made after compression band removal, applying the band to the ulnar artery for 1 hour resulted in recanalization in nearly 70% of patients.[18]

CONCLUSION

Despite the fact that RAO is asymptomatic in the vast majority of patients, it remains a problem if repeat procedures are required and, in the rare patient, can lead to some discomfort. As part of standard postprocedural care, the presence of RAO should be documented. Because best practices should yield RAO rates of 2% to 4%, if higher rates are noted in your current practice, each aspect of the procedure should be re-examined, including the method of access, the size of the sheath, the anticoagulation protocol, and, most importantly, the method of hemostasis after sheath removal. ■

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