EDITORS’ INTRODUCTION

Trans-Atlantic Debate: Nonoperative versus Surgical Management of Small (less than 3 cm), Asymptomatic Popliteal Artery Aneurysms

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Popliteal artery aneurysms represent a common pathology that vascular surgeons are often confronted with. However, several issues remain incompletely understood, including indications for intervention and optimal methods of treatment. In the following paper, our discussants debate the appropriate management of small popliteal artery aneurysms. Further complicating this discussion is the unclear relationship between popliteal artery aneurysm diameter and subsequent complications. Whereas with abdominal aortic aneurysms diameter is linked to rupture risk, it is less clear with popliteal artery aneurysms where complications are more likely to include thrombosis, embolization and compression whether aneurysm diameter is accurately predictive. Perhaps other anatomic features should be included in our management algorithms? Regardless, our debaters will try to convince us whether small popliteal artery aneurysms warrant repair or not.

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Part One: For the Motion.
Asymptomatic Popliteal Artery Aneurysms (less than 3 cm) Should be Treated Conservatively

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Popliteal aneurysm (PAA) management has been confounded by paradox and controversy. Until the start of the 20th century the principle of management was to induce thrombosis within the aneurysm either by compression or ligation. 1 Subsequently the aim of treatment was to prevent thrombosis from happening! This is the paradox. Controversial aspects of their treatment include the use of intra-arterial thrombolysis for thrombosed PAA,2 3 4 which operation to carry out, what approach to use and whether an endovascular repair is appropriate. 5 6 However, the greatest controversy is probably when to operate on an asymptomatic PAA.

Demographics and Natural History

PAAs account for more than 80% of all peripheral aneurysms, having a prevalence of approximately 1% in men aged 65–80 years. 7 They are mostly atherosclerotic in origin: other rarer causes include infection, trauma, familial or those associated with Marfan’s and Behcet’s
being asymptomatic at the time of diagnosis. Asymptomatic PAAs become symptomatic at a rate of approximately 14% per year. Symptoms include pain or discomfort behind the knee, intermittent claudication from thrombosis, repeated micro-emboli or combined stenotic arterial disease and leg swelling, with or without deep venous thrombosis secondary to compression of the popliteal vein. Rupture is extremely rare. Acute limb ischaemia secondary to thrombosis is the most serious complication with a reported amputation rate of 14%. PAAs treated electively have superior outcomes in terms of limb loss, graft patency and patient mortality than those presenting as an emergency. Following thrombosis, after the initial high amputation rate, five-year graft patency is 65% (range 50–80%) and five-year limb salvage rate 95% (90–97%). On the other hand, five-year patency following elective repair is 80% (range 70–94%) and five-year limb salvage 98.4% (95–100%).

Protagonists of early elective repair will quote series showing negligible morbidity and mortality with elective repair. However these series are not likely to be representative of most surgeons’ experience. There is risk to both life and limb following an elective repair with a 30-day mortality up to 1% and 30-day limb loss up to 2% having been reported. It is impossible to know how many limbs are ultimately lost when a synthetic graft occludes with subsequent loss of run off. Of 110,000 procedures recorded in the Swedish Vascular Registry, there were 717 primary operation for PAA. Limb loss at one year following repair of 219 asymptomatic PAAs was 1.8% (four limbs). Residual symptoms in the previously asymptomatic limb also occur in up to 2% of cases. Limb swelling following repair can be particularly troublesome.

The key question is when is the risk of morbidity and mortality associated with surgical intervention of asymptomatic PAAs less than the risk posed by surveillance alone and the potential to develop acute limb ischaemia? Size, distortion, presence of thrombus and state of run off have all been suggested as means of identifying a high risk PAA. But what is the evidence to support these suggestions?

Identification of the High Risk Aneurysm

Size

Unlike abdominal aortic aneurysms, PAA diameter does not seem to be an adequate predictor for development of complications. Whilst it is generally agreed that operating on PAAs of less than 2 cm diameter confers no benefit, controversy surrounds those in the 2–3 cm category. Although size does seem to relate to symptoms, there is little evidence to support 2 cm as the surgical cut off point. Most published series regarding PAA size are small; one multicentre study of 200 aneurysms found that asymptomatic PAAs were on average 2 cm in diameter and that those with limb-threatening ischaemia were 3 cm in diameter. Other series have shown that symptomatic aneurysms are generally larger than asymptomatic; however the finding of a consistent, statistically significant difference in size between the two groups is not well supported. In a series from Poland, describing 86 aneurysms, there was no difference in aneurysm diameter between those with non limb-threatening symptoms and those with limb-threatening symptoms and diameter did not influence limb loss. In our series of 116 aneurysms a cut off of 2 cm diameter did not discriminate well between asymptomatic and thrombosed PAAs. The sensitivity, specificity, positive predictive and negative values being 73%, 39%, 54% and 59% respectively. We also found that none of 17 PAAs 2–3 cm in diameter without significant distortion, thrombosed during a median 17 month follow-up.

If surveillance is to be started, how often should duplex scans be carried out? There is little information on the rates of growth of PAA. We found that of 24 PAAs, followed with serial duplex scans, the rate of expansion increased with aneurysm size. On average PAA of 2–3 cm diameter grew at an average rate of 3 mm/year. Hypertension was associated with a more rapid growth. A recent study of 125 PAAs did not find hypertension to be associated with a more rapid growth rate. PAAs in patients with previously treated contralateral popliteal artery ectasia and those with extra-PAs had more rapid expansion. On the other hand PAAs in patients with diabetes had a slower than average growth rate.

In our study the upper 95% confidence interval for expansion of PAA of less than 2 cm in diameter was 3 mm. If 2 cm is taken as the cut off point then PAA less than 17 mm can be scanned yearly rising to 6 month intervals when the PAA reaches 17 mm. Similarly, if it is decided to operate at a diameter of 3 cm, then yearly scans are required up to a diameter of 2.4 cm, increasing to 6 monthly scans for the larger PAA. However rather than recommending surveillance intervals based on diameter alone, intervals should ideally be customised to the individual patient after consideration of risk factors for growth and risk factors for thrombosis. More frequent surveillance intervals should be considered for PAA deemed to be at high risk.

Distortion

As the popliteal artery dilates it also lengthens. The upper and lower ends of the artery are relatively fixed and so the artery becomes distorted. As the distortion increases, the PAA is more likely to become symptomatic. We measured the most proximal angle of distortion in the antero-posterior plane on angiograms and found a median distortion of 60° in patients with acutely thrombosed PAAs; that distortion was greater in symptomatic compared with asymptomatic PAAs and with thrombosed compared with non thrombosed PAAs. There was a direct correlation between diameter and degree of distortion. Size alone did not differentiate the two
groups. Distortion of the aneurysm appears to be a more sensitive predictor of thrombosis than size alone.

**Size and distortion**

Combination of factors may be a better way of identifying high risk aneurysms. We found that the combination of size and distortion provided the best sensitivity, specificity and positive and negative predictive values comparing asymptomatic with thrombosed PAAs, these being 90%, 89%, 83% and 94% respectively.  

**Presence of thrombus**

In common with aneurysms at other sites, laminated thrombus develops within PAs. It has been postulated that thrombus is at greater risk of disintegration and embolisation compared with aneurysms elsewhere because the popliteal artery is continually subjected to flexion and extension. It is often stated that thrombus within a PAA is an indication for elective operation. However in the Joint Vascular Research Group study of 200 PAAs, thrombus was present in 70% on ultrasound scanning. This was not confirmed by the Townsville study of 125 PAAs, where only four aneurysms (median diameter of 15.5 mm) had thrombus at the start of surveillance (none of these had operative intervention or complications) and only five developed thrombus during surveillance (median diameter of 25 mm). It is interesting to note that only nine patients in this study reached diameters of greater than 2 cm. The Swedish study quoted earlier reported that 96.4% of asymptomatic aneurysms smaller than 2 cm had more than 2 mm of thrombus in the wall of the aneurysm. PAAs with thrombus are generally larger than those without. It is reasonable to assume that as the aneurysm enlarges thrombus will develop. However, there is no evidence to support the theory that presence of thrombus indicates a high risk aneurysm.

**Run off**

Large emboli usually produce an obvious clinical picture. In many patients micro-embolisation of the peripheral circulation occurs silently. Compromise of the run off can impact adversely on the outcome from bypass surgery. Once embolisation occurs the aneurysm cannot be regarded as being asymptomatic and therefore surgical repair should be considered. Poor run off secondary to embolisation has been suggested as being an indication for early repair. Evidence supporting this is sparse. One retrospective study demonstrated a greater risk of complications developing in those PAA associated with no distal pulses compared with those having distal pulses.

**Other factors**

Statins have been shown to be associated with less likelihood of severe ischaemia developing, whereas antiplatelet medication and lipid levels were no different in patients with or without thrombosed PAAs.

**Conclusion**

All patients with PAAs should have "Best Medical Treatment" as recommended to anyone with cardiovascular disease. They should be assessed for the presence of other aneurysms. Management of asymptomatic popliteal artery aneurysms remains controversial. It is clear that no single criterion is sufficiently robust to identify reliably high risk PAAs. Using a cut off greater than 2 cm diameter alone as an indicator for elective repair will subject an unacceptably large number of patient to unnecessary morbidity and mortality. However, combining risk factors may be more useful. Size and distortion appear to be a more reliable means of identifying high risk aneurysms than size alone.

Asymptomatic PAA measuring less than 3 cm in diameter should be treated conservatively……providing that there is no significant distortion of the aneurysm.
Part Two: Against the Motion. Asymptomatic Popliteal Artery Aneurysms (less than 3 cm) Should be Repaired  

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In our prior review of 34 popliteal artery aneurysms (PAA),1 we suggested that small PAA’s were associated with higher incidence of thrombosis, clinical symptoms, and distal occlusive disease. This was based upon the liberal use of duplex scanning to make the diagnosis.2,3 We concluded that small PAA (<2.0 cm) may not be as benign as previously suggested. Symptomatic PAA were significantly smaller than asymptomatic aneurysms (p < 0.03). The majority of the small aneurysms (64%) in this series were partially thrombosed and this incidence was not significantly different from that of larger aneurysms (70%). Complete thrombosis of the PAA did not correlate with aneurysm size. When comparing runoff scores, these smaller symptomatic PAA had poorer outflow vessels. These data suggest that size alone does not correlate with the presenting symptoms of PAA. The presenting symptoms or lack thereof did not correlate with the prevalence of diabetes, location of the PAA, or degree of superficial femoral artery disease. Despite their small size, these aneurysms can be lined with thrombus that can embolize to the infrapopliteal arteries. We suggested that small popliteal aneurysms in patients with low operative risk, acceptable runoff and adequate venous conduit should be considered for repair. This is especially true if duplex imaging demonstrates mural thrombus. While admittedly there is no prospective evidence that mural thrombus is an indication for repair, we suggest that these data indicate that mural thrombus may be the main source of symptoms and limb loss. This is especially true as the morbidity and mortality of repair has decreased with the advent of minimally invasive techniques.

In Dr. Galland’s 2005 paper,4 he stated that his data examining 116 PAA found that “size was not significantly different between the two groups” (asymptomatic and symptomatic). Therefore, it would be difficult to justify the topic assigned to Dr. Galland of “Asymptomatic popliteal artery aneurysms <3 cm should be treated conservatively” based upon his data. However, if PAA were >3 cm and if distortion of the proximal artery >45° was found, then a positive correlation was found with the presenting symptoms. While we cannot compare whether distortion was observed in our patients with PAA with mural wall thrombus or whether mural wall thrombus was noted in the patients with distortion in the series of Dr. Galland, it is possible there may be some overlap between the subsets. These data have the same limitation as our dataset as they correlate the presenting symptoms with the anatomy of the lower extremity arteries in a retrospective fashion. Ideally, we should obtain data that prospectively help guide the management of patients with PAA. Interestingly, there have been no reports in the ensuing years from other centers confirming or denying these findings.

These comparisons highlight the need for modern series with longitudinal prospective data to explore issues of natural history or PAA. Many of the prior data weighed the risks of open bypass as compared to endovascular repair which in some centers has been the first line therapy.5 On the other hand, Dr. Galland’s group have suggested that endovascular repair of PAA is associated with a longer procedure, higher thirty day reintervention rates and similar long term primary patency rates.6 Five of the 37 endovascular repairs in this series were performed with Wallgrafts which may make the dataset somewhat outdated. This data are also in contrast to ours suggesting endovascular repair is a shorter procedure and its patency rates rival that of open repair.7,8 The marked reduction in morbidity of endovascular PAA has assured its place in our armamentarium in the treatment of PAA and suggests that prior attempts of comparing the risks versus benefits of open surgical repair of PAA need to be modified in the present modern day era.9,10 Based upon these data, we suggest the need for other centers to examine their data, especially focusing on prospective longitudinal data to help answer some of these unresolved questions. While many questions about the management of PAA do remain, we maintain that some patients with PAA less that 3 cm especially with mural thrombus should be repaired either with open or endovascular methods.

References

3 Kallakuri S, Ascher E, Hingorani A, Marks N, Shiferson A, Tran V, et al. Effect of duplex arteriography in the management of