LETTERS TO THE EDITOR

Regarding “Surveillance of small aortic aneurysms does not alter anatomic suitability for endovascular repair”

In the article by Yau et al., the authors recently analyzed changes in anatomical suitability for endovascular repair (EVAR) occurring over time in small aortic aneurysms. The report focuses on a largely debated issue. Two randomized trials (one American and one European) are ongoing to find an answer as to whether or not early endografting can have a better outcome in avoiding adverse events and loss of suitability in patients with aneurysms less than 5.5 cm.

Yau et al retrospectively reviewed the records of 54 patients at a medium follow-up of 24 months and concluded that minor changes occurred and did not affect EVAR suitability. Unfortunately, only a small number of patients (54) were included with a short follow-up (median 24 months, interquartile range 15 to 36 months); reduced patients survival according to Kaplan Meyer estimates (78% at 30 months and 61% at 50 months) further decreased the study population and the resulting number of observations recorded. Furthermore, it should be noted that 25% of patients dropped out during the follow-up due to abdominal aortic aneurysm (AAA) repair performed at another institution. Nevertheless, changes in aortic neck were detected in a substantial percentage of cases. Aortic neck is the key point to assess EVAR suitability and to ensure long-term success of endografting. In the present study, the authors showed that median neck length decreased nearly 30% (from 26.5 to 20 mm; P = .001) median neck diameter increased (from 23 to 24 mm; P = .02), and growth in median AAA diameter from 44.5 to 48.9 mm was observed. All these relevant modifications seem to contradict the conclusions by the authors that the observed changes did not decrease the suitability for EVAR. Considering a larger study group and a longer follow-up, if this trend is maintained, we may expect more substantial modifications and a potential increased risk of adverse events.

This study further confirms that growth rate and morphological modifications in small AAA may be difficult to predict, compromising a safe repair at a later stage. The substantial uncertainty whether surveillance is better than EVAR in small aneurysms still remains. Significant answers could be obtained by the results of the ongoing randomized trials.

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Reply

We appreciate Dr Cao’s discerning comments and questions. Although our study consisted of only 54 participants, it is likely the largest cohort of its kind to longitudinally observe the natural morphologic history of small aneurysm growth in the same patients over time. Most other studies compare different populations, different aneurysm sizes, at different time points. As previously mentioned, studying small aneurysm growth will always be handicapped by attrition from surgery, rupture and death. More than half of small aneurysm (45 to 49 mm) in the Aneurysm Detection and Management (ADAM) study required surgery during a 2-year period. No study can escape this reality.

Despite the study population size and 24-month median follow-up period, this study fittingly examines the critical period of small aneurysm growth between 4.5 and 5.0 cm. Our cohort accurately recapitulates “real world” surveillance of small aneurysm patients. Even with 54 patients, we were able to comfortably demonstrate statistically significant morphologic changes during small aneurysm growth. However, in spite of these changes, we were able to also show no statistically significant differences in endovascular aortic aneurysm repair (EVAR) suitability during surveillance.

Although it may seem like an apparent contradiction that observed adverse morphologic changes (shorter and larger aortic necks) would not lead to decreases in EVAR suitability, closer examination of the data can explain how these two conclusions can coexist. The majority of patients who remained EVAR candidates throughout the study (45 patients, 84%) continued to have long (>15 mm) and suitably narrow (<26 mm) infrarenal aortic necks. Only 16% had borderline aortic neck lengths (10 to 15 mm) and only 18% had borderline aortic neck diameters (26 to 28 mm). Therefore, only a minority of patients with small aneurysms are “at risk” to lose their EVAR suitability during the surveillance period. This observation suggests that an aneurysm’s suitability for EVAR is determined early in the morphologic life of the aneurysm—likely before the maximum aortic diameter reaches 4 cm. If an aneurysm is not suitable for EVAR when surgical repair is warranted, it is likely that the aneurysm was unsuitable for EVAR during most of its natural history. Surveillance likely will exclude very few patients.

Our study also does not support the contention that the observed morphologic changes will necessarily result in adverse long-term EVAR outcomes. There is no data that EVAR with longer aortic necks (26.5 mm at the beginning of the study) necessarily results in better long-term outcomes than EVAR with adequately long but shorter necks (20.0 mm at the end of the study). Surveillance does not change the fact that the majority of patients suitable for EVAR at the end of surveillance will continue to have comfortably long and small aortic necks that should not compromise long-term outcomes with EVAR. Moreover, further analysis of our data determining EVAR suitability with the potential use of endografts up to 36 mm in diameter, which only became recently available in the United States, revealed that EVAR sui-
ability rates were almost the same during the study period (80% vs 76%; McNemar, \( P = .5 \)).

Finally, as level 1 evidence, it has clearly demonstrated that the treatment for small abdominal aortic aneurysms (AAA) is unnecessary and given that EVAR suitability does not change during surveillance, as demonstrated in our study, we could not agree more with Dr Palamara, who in a previous letter to the editor of this journal appropriately expressed that randomized clinical trials involving EVAR for small AAAs are “neither morally nor scientifically justified”.

Please note a correction in original manuscript. The third sentence in the last paragraph of the discussion should read: “If an aneurysm is not suitable for EVAR when surgical repair is warranted, it is likely that the aneurysm was unsuitable for EVAR during most of its natural history.”

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I do congratulate you for such an important report, which would have been a lot better if its embryologic, pathophysiologic, and hemodynamic backgrounds were included as one of the CVMs.

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I enjoyed your Case Report of Popliteal Artery Aneurysm on Klippel-Trénaunay (KTS) patient, and also, your reply to the Letter to the Editor from H. Komai. However, there seems to be unnecessary confusion on the pathogenesis of arterial aneurysm/arterial dilatation on both reports, which represents “truncular” form of arterial malformation (AM), although you referred to it briefly in your report. AM is one of various congenital vascular malformations (CVMs) and its “truncular lesion” is the result of the developmental arrest in the “latter” stage of embryogenesis. It often remains along the line of aplasia/hypoplasia/hyperplasia. Therefore, it does not possess mesenchymal cell characteristics of evolutionary power to grow; only embryonic tissue remnant from the “earlier” stage of embryogenesis has, which we call “extratruncular” lesion.

Your observation is typical truncular AM lesion, as you have already shown in the histology. This truncular lesion lacks sufficient lamellae of smooth muscle cells within the media as its characteristic. Depending upon the severity, location, and “prenatal” hemo-arterio-dynamics, this lesion will progress to an aneurysmal condition or stay as “ectatic” condition, which is not uncommon. Based on this fundamental defect in the arterial wall structure, it would become more susceptible to pathological change (eg, atherosclerosis) such as Dr Komai’s case, as you properly speculated in your reply, and the pathogenesis of both cases of the report is embryonic tissue defect at the latter stage of embryogenesis resulting in abnormal arterial wall. Therefore, understanding of embryologic background is warranted for the proper management of this “truncular” form of arterial malformation.

I also noticed that the “old” name-based nosology/term such as Klippel Trénaunay Syndrome added more confusion; this old term failed to fulfill its mandate as a proper classification for various congenital vascular malformations (CVMs). We try to discourage its further use, at least among the professionals who adopted the Hamburg Classification.

Any vascular malformation can be involved to KTS but in the traditional KTS, it generally consists of venous malformation (VM) and lymphatic malformation (LM) in addition to capillary malformation (CM) known as “port wine stain”. This “combined form” of CVM is generally represented by “hemolymphatic” malformation (HLM) in the new classification. Occasionally, HLM has additional CVM known as AV malformation (AVM), which condition has been called by a different name: Parkes Weber Syndrome.

Now, if you should have such AM in addition to all other CVMs, what is the use for such name-based nosology for the practical management after all?

Reply

In his letter to the editor, Professor Lee mentioned that our observation could correspond with a typical truncular arterial malformation situation, which lacked sufficient lamellae of smooth muscle cells within the media and would progress to an aneurysmal condition or stay as ectatic condition, to no small extent, depending on the severity, location, and postnatal hemo-arterio-dynam-