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Commentary: “Aneurysm Sac Pressure after EVAR”

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EVAR aims at excluding the aneurysm sac from blood flow and systemic pressure. It is surprising the relatively little knowledge gathered on intra-aneurysm sac pressure after EVAR, especially taking into account that EVAR has been introduced 16 years ago. The review done by Hinnen and coworkers on this important subject is therefore greatly appreciated.

It is widely accepted that reduced intra-sac pressure after EVAR may decrease wall tension and thereby make the aneurysm shrink. However, as the review article by Hinnen and coworkers points out, experimental studies have shown that intra-aneurysm pressure is influenced by multiple factors such as the mechanical properties of the aneurysm wall, the type of graft and the aneurysm size. The measurement of intra-aneurysm sac pressure after EVAR has to address two main issues: the access to the AAA and the use of a pressure sensor that does not loose its reliability within thrombus. Mean pressure index (MPI) and pulse pressure have been the most commonly used parameters in the evaluation of intra-aneurysm pressure. Successful EVAR, defined by shrinkage of the AAA in the absence of endoleak, has been repeatedly associated with low MPI and pulse pressure. However, there is also data to support that the reduction of MPI does not occur immediately after EVAR. It may take at least 1 month for the pressure to drop after stent-graft insertion. Furthermore, aneurysm expansion without endoleaks (endotension or type V endoleaks) is usually associated with elevated intra-sac pressure. Our own studies suggest that the intra-sac pressure may predict future AAA expansion in AAAs that do not shrink after EVAR in the absence of endoleaks.
Endoleaks seem to be associated with varying levels of pressurization of the aneurysm, especially in late follow-up. However, pressure measurements in the presence of endoleaks are conditioned by the non-uniformity of pressure throughout the AAA, with consistently higher pressure in the endoleak channel than in the AAA thrombus (Dias et al, in press, J Vasc Surg 2007). Moreover, the direction of the pressure sensor in relation to the source of pressure can also influence the measurement, even in validated methods. Nevertheless, at least in type II endoleaks the association between low intra-sac pressure and shrinking AAAs seems to be kept and successful embolization of the endoleak leads to a pressure reduction (Dias et al, in press, J Vasc Surg 2007).

Some remarks may need to be added about the interpretation of intra-aneurysm sac pressure measurements. A hygroma can lead to aneurysm expansion and the pressure is not necessarily high. A single spot measurement relies on uniform pressure transmission throughout the aneurysm-sac thrombus. However, the non-uniformity of thrombus structure has been suggested to influence the transmission of pressure. A pressure measurement reflects the intra-aneurysm sac pressure at that single moment and, although it is able to predict future diameter evolution, it does not exclude the rare event of sudden sac pressurization. Non-invasive and repeated assessment of the level of pressurization by wireless pressure sensors has been reported. However, more research is required before these methods can be applied in clinical practice.

In conclusion, we agree with Hinnen and coworkers that intra-aneurysm sac pressure measurements will have a place in the future follow-up after EVAR. It seems that they
will not replace follow-up by imaging. Pressure measurements will preferably be combined with imaging for the optimization of the future follow-up after EVAR.

References:


