discussion without further evidence detracts from our principal finding that in our large cohort of patients the conservative management of patients with isolated type II endoleak has not resulted in an increased aneurysm-related mortality, or the development of high-pressure endoleaks. We have also demonstrated that whilst angiography may be more discriminative for the diagnosis of occult type 1 or 3 endoleaks, its use in patients with type II endoleak without significant sac expansion may carry more risk than benefit. Importantly, in our paper we have demonstrated that over half of all type II endoleaks spontaneously resolve without intervention.

We agree that a gold standard strategy for managing patients with type II endoleak remains to be established. It is unlikely that a randomised controlled trial comparing different treatment strategies will be possible so the setting up of a multicentre type II endoleak registry to formally compare different approaches is imperative.

REFERENCE


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Available online 4 November 2014

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http://dx.doi.org/10.1016/j.ejvs.2014.10.004
DOI of original article: http://dx.doi.org/10.1016/j.ejvs.2014.09.014

Re: “Spontaneous Delayed Sealing in Selected Patients With a Primary Type Ia Endoleak After Endovascular Aneurysm Repair: Does Correcting the Picture Save the Life?”

I read the paper by Gonçalves et al. with interest.1 The authors state that, in selected patients, a conservative approach for primary type Ia endoleak may be justified. All but one of the 15 primary type Ia endoleaks sealed spontaneously within 5 months. The disappearance of type Ia endoleaks resulted from improved graft wall apposition due to neck remodelling or thrombosis of the non-apposed neck segment.

If the barrier between the aneurysm sac and the systemic arterial circulation consists of thrombus only, systemic pressure can be transmitted through a clot, and it is known that mural thrombus on the surface of the aneurysm sac does not prevent rupture.2 For this reason, a thrombotic barrier on the neck segment may eliminate type Ia endoleak but may not prevent rupture. In the presented study, although ruptures were not detected in the 14 patients, sac growth occurred in four (28.5%).1 In these patients known causes of sac growth were not detected by CT. There are limited data about the long-term results of the other methods of induction of thrombosis on the non-apposed neck segment, such as fibrin glue injection or coil embolization. Feng et al.3 documented results of patients treated by fibrin glue injection: one aneurysm related death and four aneurysm sac growths were detected in 48 cases during 45 months follow up.

Until the long-term results are clearer, we should avoid a conservative or similar approaches to induce thrombus formation for treatment of type I endoleak. Correcting the picture may not save the life.

REFERENCES


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Available online 28 November 2014

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http://dx.doi.org/10.1016/j.ejvs.2014.09.015
DOI of original article: http://dx.doi.org/10.1016/j.ejvs.2014.10.011