



Expansion and Atrophy – aortic wall development after EVAR with an endoleak type II

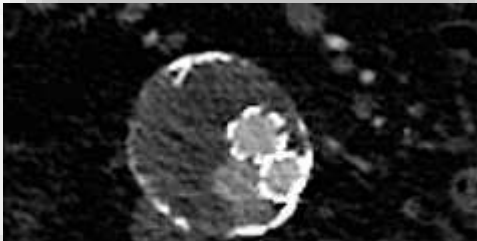
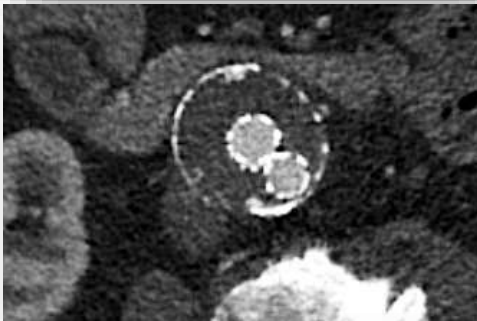
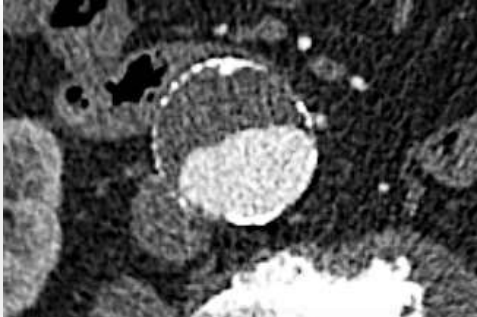
A Busch

Department of Vascular and Endovascular Surgery,
Klinikum rechts der Isar, Munich

**The structural atrophy of the aneurysm wall in secondary
expanding aortic aneurysms with endoleak type II**

Anna-Leonie Menges, MD,^a Albert Busch, MD,^{a,b} Benedikt Reutersberg, MD,^a Matthias Trenner, MD,^a
Philip Kath,^a Ekaterina Chernogubova, PhD,^a Lars Maegdefessel, MD, PhD,^{a,b} Hans-Henning Eckstein, MD,^a
and Alexander Zimmermann, MD,^a Munich, Germany; and Stockholm, Sweden

Introduction



- AAA prevalence age dependent 2-11%
- EVAR in 80% of elective and 60% of acute repairs
- specific complications:
 - type I and III endoleaks (EL) ▶ immediate repair
 - type II EL in 20-32% of EVARs ▶ watch and wait
 - ca. 30% spontaneous resolution
 - ca. 30% persist w/o sac growth
 - ca. 30% persist w/o sac growth ▶ repair >5mm growth
 - low rupture rate reported

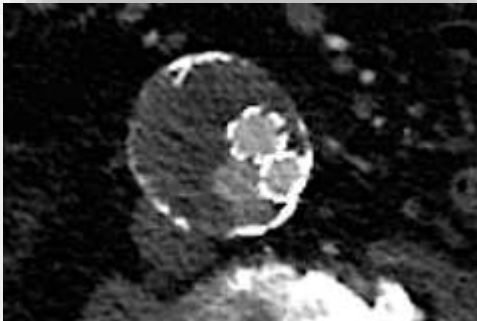
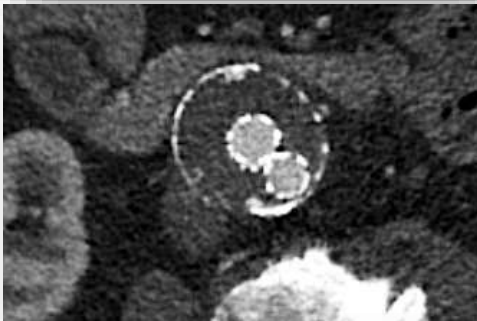
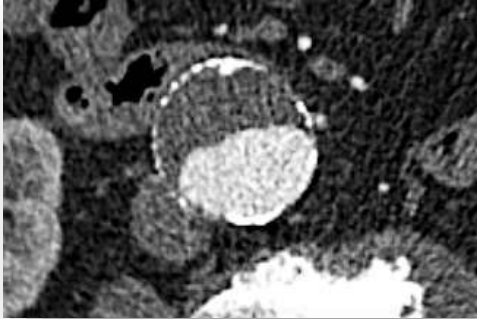
REVIEW

Editor's Choice — Systematic Review and Meta-Analysis of the Outcome of Treatment for Type II Endoleak Following Endovascular Aneurysm Repair

Klaas H.J. Ultee ^{a,f}, Stefan Büttner ^{a,f}, Roy Huurman ^a, Frederico Bastos Gonçalves ^{a,b}, Sanne E. Hoeks ^c, Wichor M. Bramer ^d, Marc L. Schermerhorn ^e, Hence J.M. Verhagen ^{a,*}

Conclusion: There is little evidence supporting the efficacy of secondary intervention for type II endoleaks after EVAR. Although generally safe, the lack of evidence supporting the efficacy of type II endoleak treatment leads to difficulty in assessing its merits.

Introduction

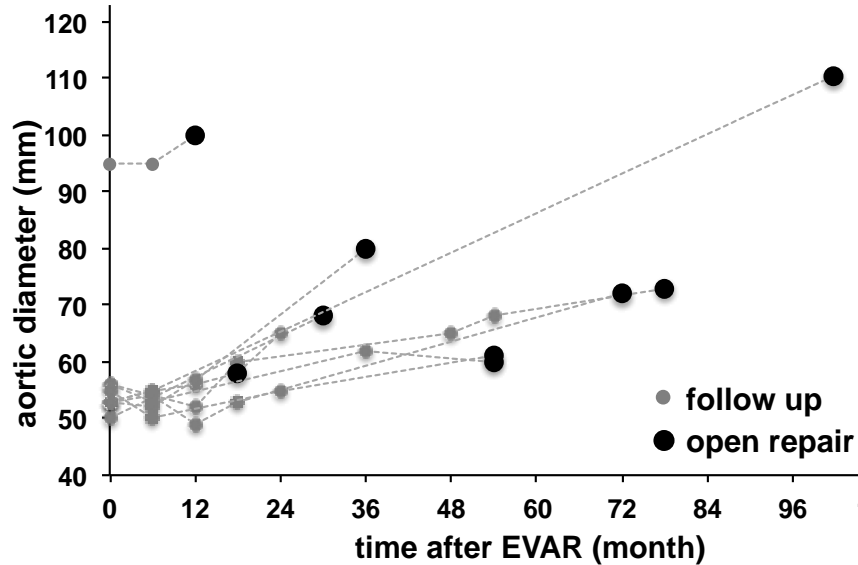


- AAA prevalence age dependent 2-11%
- EVAR in 80% of elective and 60% of acute repairs
- specific complications:
 - type I and III endoleaks (EL) ▶ immediate repair
 - type II EL in 20-32% of EVARs ▶ watch and wait
 - ca. 30% spontaneous resolution
 - ca. 30% persist w/o sac growth
 - ca. 30% persist w/o sac growth ▶ repair >5mm growth
 - low rupture rate reported

central research question

What happens in the secondary expanding aneurysm sac wall due to endoleak type II compared to normal aorta and AAA?

Study Design



11 control aortae

42 AAA samples

- IHC
- WesternBlot
- RT-PCR
- Angiogenesis
- Inflammation
- wall composition
- cell proliferation
- apoptosis

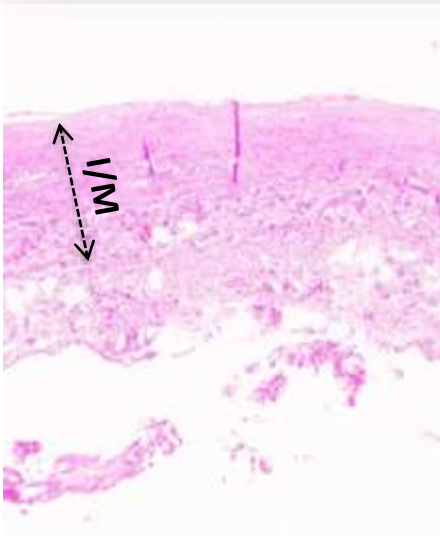
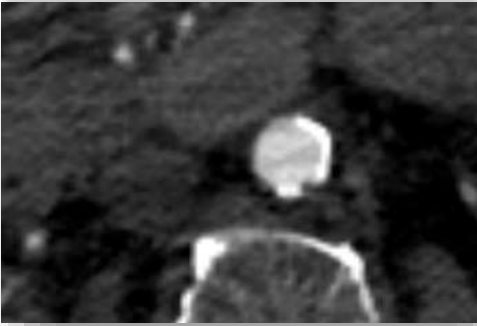
10 patients

OR

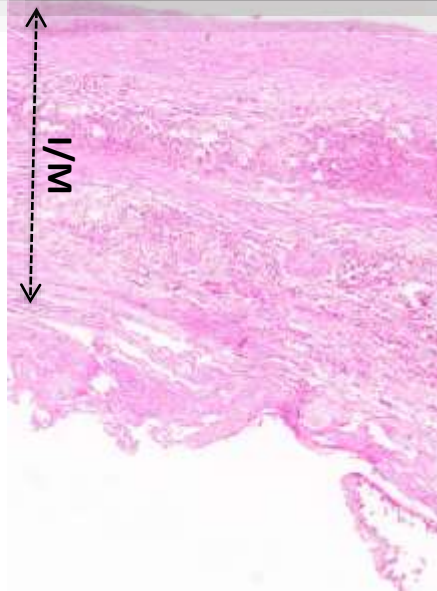
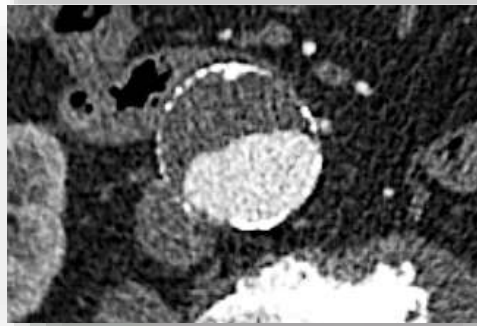
structural analysis

Results

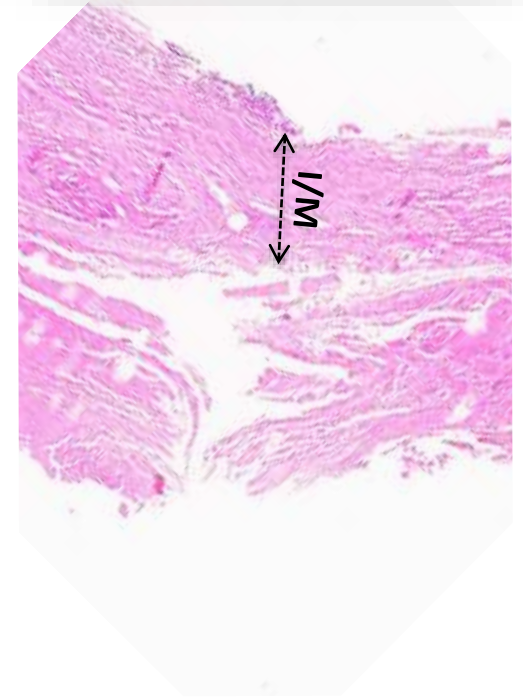
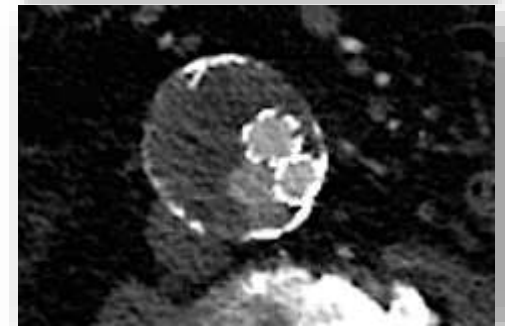
control aorta



AAA



sec expanding AAA

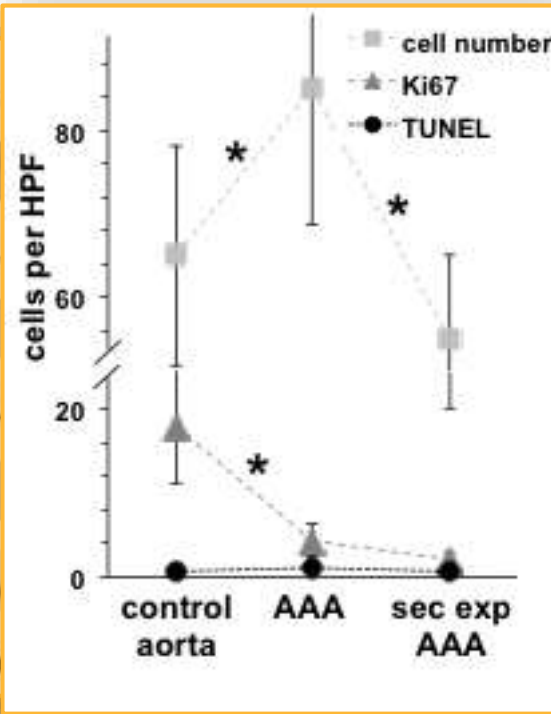
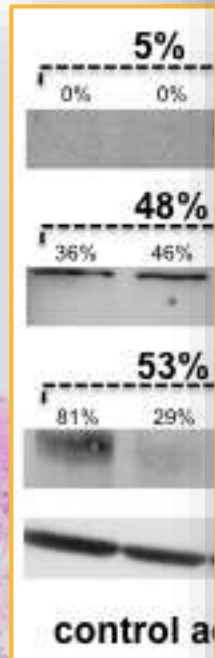
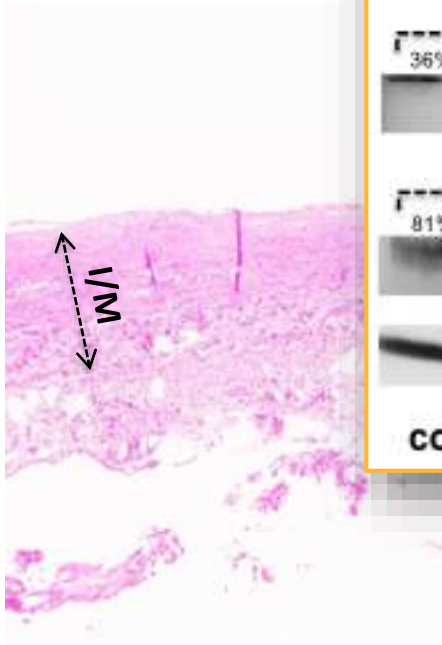


Results

control aorta

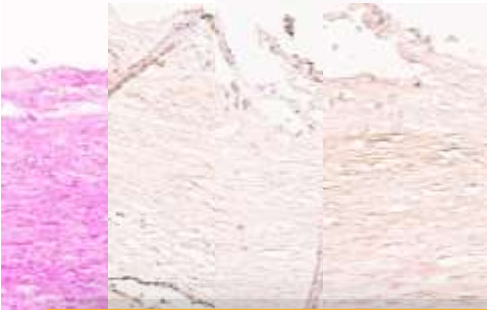
AAA

sec expanding AAA

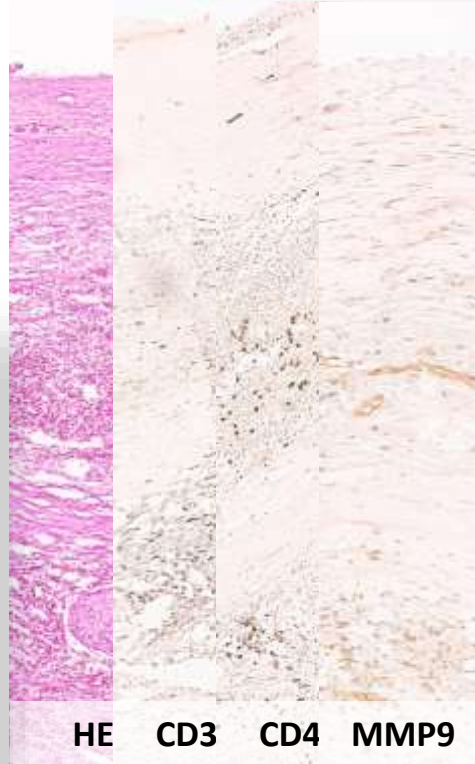


Results

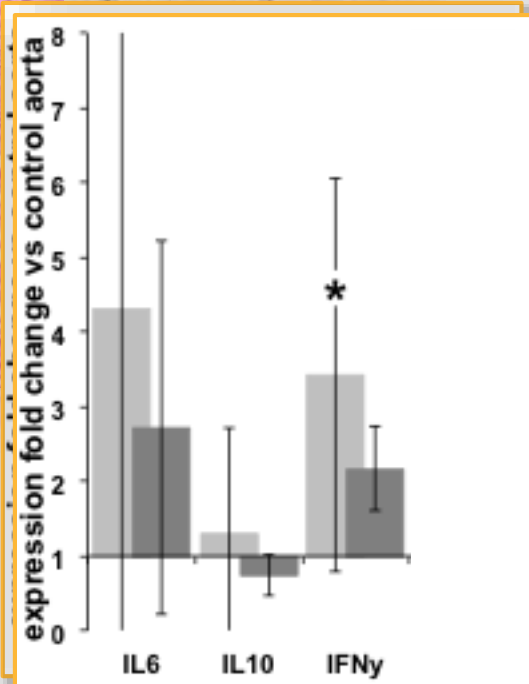
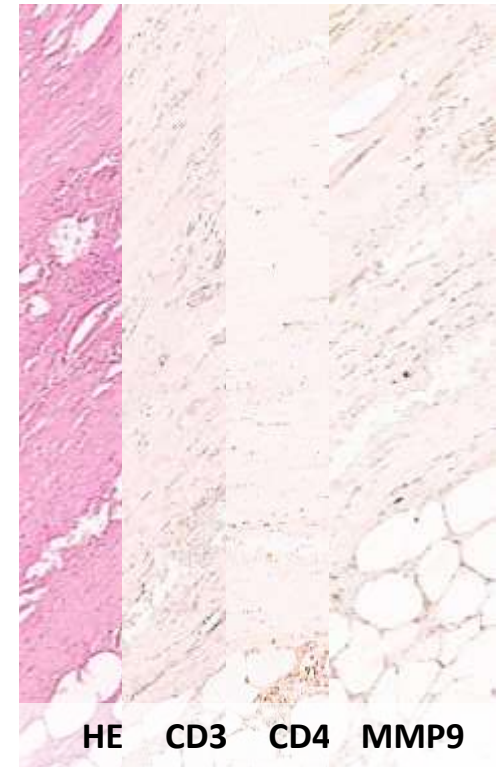
control aorta



AAA

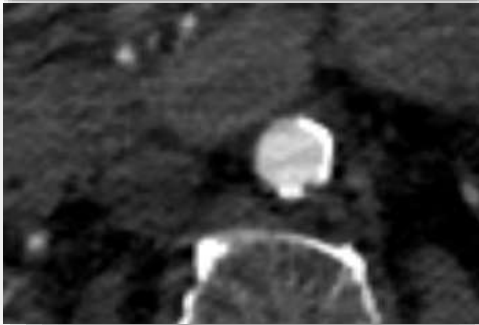


sec expanding AAA

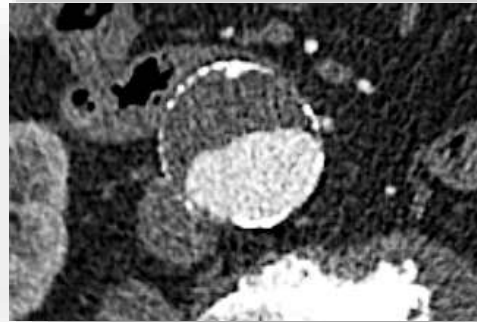


Summary

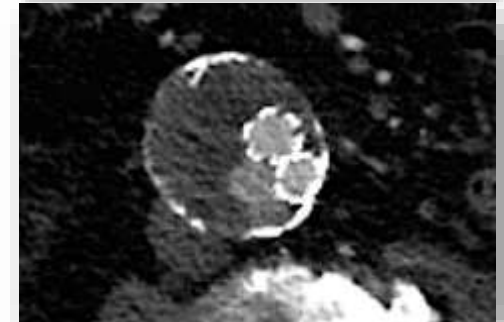
control aorta



AAA



sec expanding AAA



fibrosis

altered hemodynamics

intraluminal thrombus

proteolytic imbalance

angiogenesis

humoral immune answer



?



reduced fibers and cellularity

altered hemodynamics

intraluminal thrombus

proteolysis

no angiogenesis

no inflammatory cells

Conclusion

central research question

What happens in the secondary expanding aneurysm sac wall due to endoleak type II compared to normal aorta and AAA?

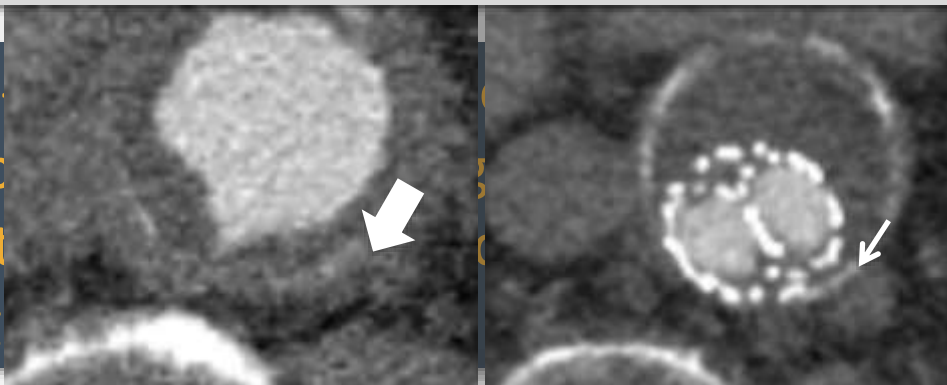
thin and fibrotic wall with little cellularity and enzymatic activity

suggests a widely inert aneurysm sac exposed to the special conditions of EL type II

adds additional evidence to the role of pressure and stress on the aneurysm wall

rupture might be less frequent than secondary/late type I/III endoleak

EVAR follow-up shows sac enlargement, backflow thrombus, stent migration and distal sealing



aneurysm enlargement, luminal thrombus, proximal and distal sealing

Acknowledgement

Klinik und Poliklinik für Vaskuläre und Endovaskuläre Chirurgie, MRI, München

HH Eckstein	L Maegdefessel
J Pelisek	R Hegenloh
A Zimmermann	P Kath
AL Menges	G Biro

Molecular Vascular Medicine Group, CMM, Karolinska Institute, Stockholm, Sweden

L Maegdefessel	Y Li
E Chernoguobova	H Jin
P Erikson	P Jansson
V Paloschi	

Klinik für Allgemein-, Viszeral, Gefäß- und Kinderchirurgie, UKW Würzburg

CT Germer	C Tiurbe
U Lorenz	A Holm
R Kellersmann	C Schmidt
C Bühler	C Otto
M Koospal	B Mühling
M Schneider	

