Expansion and Atrophy –

aortic wall development after EVAR with an endoleak type II

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I do not have any potential conflict of interest
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)
  - Type II EL in 20-32% of EVARs
    - Ca. 30% spontaneous resolution
    - Ca. 30% persist w/o sac growth
    - Ca. 30% persist w/o sac growth
    - Immediate repair
    - Watch and wait
    - Repair >5mm growth
    - Low rupture rate reported
    - Late type I EL rate unknown

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
- Western Blot
- RT-PCR
- Angiogenesis
- Inflammation
- Wall composition
- Cell proliferation
- Apoptosis

10 patients → OR → structural analysis

Graph showing aortic diameter over time after EVAR (month)
Results

- control aorta
- AAA
- sec expanding AAA
Results

control aorta

AAA

sec expanding AAA
Results

control aorta

AAA

sec expanding AAA

expression fold change vs control aorta

IL6  IL10  IFNy  *
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, little cellularity and enzymatic activity
- widely inert aneurysm sac
- emphasizes the role of pressure and stress on the aneurysm wall
- rupture might be less frequent than secondary/late type I/III endoleaks

**EVAR follow-up should focus on aneurysm enlargement, changes in annual growth, luminal thrombus, stent migration and especially the proximal and distal sealing zones**
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