Many Patients (>1/3) With Unstable AAA Sacs And Endotension After EVAR Really Have Positional- Dependent Endoleaks: How To Diagnose And Treat This Problem.

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**Background:**
Follow up of patients undergoing elective EVAR at the Royal Prince Alfred Hospital between May 1992 and November 2011 disclosed that they fell into three distinct groups: those with no endoleak, those with a definite endoleak and a group of 31 patients who had an unstable AAA sac. The well known features of AAA sac instability include an increase in sac diameter, a change in shape of the aneurysm sac and device migration. In the absence of any demonstrable endoleak these changes were given the title endotension.

An increase in ultrasound technology with improved resolution and colour sensitivity capabilities have enabled endografts to be imaged more clearly. Improvements in B-mode resolution and the use of harmonic imaging have also allowed the thrombus within the aneurysm sac to be characterised with greater confidence. The B-mode and colour duplex ultrasound features that are suggestive of aneurysm sac instability include the following:

- Increase in endograft pulsatility, prominent echolucent areas within the sac thrombus, mobile thrombus within the sac and low amplitude atypical colour signals close to the endograft wall.

**Patients:**
We first became aware of the concept of an intermittent endoleak when an 82 year old male patient presented with a ruptured AAA in 2003. He had previously undergone endovascular repair of his aneurysm in 1998. The diameter of his aneurysm had progressively increased from 5cm to 7.6cm before rupture. No endoleak was ever demonstrated despite investigation with contrast computed tomography, duplex ultrasound scanning and directed aortography. The duplex ultrasound scan, however demonstrated that the aneurysm sac had changed shape and that streaks of echolucency were noted within the aneurysm thrombus. The patient survived conversion to open repair of his ruptured aneurysm. When the explanted endograft was filled with blood under pressure, a jet of blood appeared if the endograft was subjected to changes in position. The jet could be controlled by flexion of the endograft. We postulated, retrospectively, that the patient’s endotension resulted from an endoleak which could only be demonstrated intermittently due to positional change.

Subsequently we have confirmed that the patient’s position is an important factor in our ability to image an endoleak by colour duplex scan. The patient’s ambulatory status can also be a determining factor. When a patient had been mobile, an endoleak was detected but when he had been resting in the horizontal position for a short period of time the endoleak would disappear. We have also found that some endoleaks can only be imaged by changing the patient’s position on the examination table. These endoleaks can be imaged only by changing the patient’s position from supine to the right or left decubitus positions.

In all, 12 of 31 patients with unstable sacs were diagnosed with these endoleaks.

**Treatment:**
These intermittent/positional-dependent endoleaks were Type III. One endoleak underwent spontaneous resolution and 3, less than 5cm in diameter, were treated by surveillance. A further patient with advanced myeloma was also treated conservatively for 3 years until he succumbed from this disease. The remaining patients were treated by relining the endograft.

**Conclusion:**
We conclude that:

1) Intermittent / positional dependent endoleaks can masquerade as endotension of indeterminant origin.
2) Over one-third of patients with an unstable AAA sac and endotention after EVAR have intermittent / positional dependent endoleaks.
3) An awareness of their potential presence and method of diagnosis is necessary to avoid unnecessary conversion to open repair or late rupture.
4) B-mode and colour duplex ultrasound are the preferred diagnostic modalities to detect these endoleaks.
5) Endograft follow up requires careful scrutiny of aneurysm sac contents and wall as well as the graft device.