Calcium

- Barrier to optimal dilatation
- Barrier to optimal drug absorption
- Key cause of severe dissections
- Bilateral / circumferential calcium ranked as most severe by different calcium grading systems
- Highly prevalent in:
  - Elderlies
  - Diabetics
  - Kidney disease


DCB and Calcium

Calcium: potential barrier to optimal drug absorption
Circumferential distribution strongest influencing factor

N=60
- SFA lesions ~ 6 cm (de-novo)
- CTO: 31.7%
- DCB with standard pre-dilatation
  - a = <3 cm; b = >3 cm
  - Calcium evaluation by CTA (circumf.) and DSA (longitud.)

N=91 (retrospective)
- SFA lesions ~ 5.7 cm
- Restenotic: 45.1%
- CTO: 33.0%
- 6-month LLL (primary endpoint) by Angio Core lab adjudication

Ex-vivo experiments confirm that degree of fem-pop circumferential calcification predicts arterial drug uptake

N=5 cadaveric human lower limbs with observed arterial calcification
Paclitaxel absorption kinetics scale inversely with calcification scores

DCB and Calcium

Ex-vivo and pre-clinical experiments confirm Calcium, not plaque burden, remains the real barrier for DCB drug uptake


Plaque Scoring in calcified SFA

Supporting the role of plaque scoring for vessel prep in calcific lesions and the hypothesis that degree of calcium does not predict patency

- ASC Technical Success* = 100% (w/out pre-dil)
- Overall Primary Patency = 81.2% (69/85)

*successful scoring = ability to cross the lesion and inflate ASC, at least at NP w/out balloon rupture

Primary Patency (KM) per degree of calcification

DISRUPT PAD

Acute Effectiveness

Acute Gain

% Patency Freedom From TLR

Conclusions

- Circumferential distribution of calcium represents the main barrier for drug uptake
- Proper lesion preparation can increase the patency rate of DCBs in heavy calcified lesions
- Atherectomy devices increase the luminal gain and may also improve drug uptake
- Vessel preparation with plaque scoring and lithoplasty do not debulk calcium but are able to increase vessel permeability, drug absorption, and patency rate