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RIVASCOLARIZZAZIONE CORONARICA versus CAROTIDEA:
VI SONO DIFFERENZE NEL PROFILO DI ATTIVAZIONE PIASTRINICA?


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Coronary stenting has become a mainstay of interventional cardiology.

Stent evolution

Drug Eluting Stent
- No Polymer
- < 2% Restenosis
- No late stent thrombosis
- No Plavix

Future

Drug Eluting Stent
Durable Polymer:
- <5% Restenosis
- Late thrombosis
- Plavix +Aspirin for life

SVV

Bare Metal Stent
- 30% Restenosis

Past

DES – Stent Thrombosis

The rate of stent thrombosis are increased with both PES and SES compared with their bare metal counterpart, a difference that emerges <1 year after stent implantation.


Late stent thrombosis, defined as any platelet rich thrombus occupying >25% of lumen >30 days after DES implantation.

Finn AV et al, ATVB 2007
Acquired platelet activation following stent implantation is well documented* and may affect both short- and long-term outcomes in patients treated with stents.

*By means of platelet aggregation measurement as well as by surface receptor expression in circulating blood

**PCI and platelet activation**

**Stent Thrombosis**

- Prevention is currently based upon the combination of **aspirin** (75-325 mg/day) plus **clopidogrel** (600 mg loading dose followed by 75 mg/day).

**DES, platelet activation and drug treatment**

- No data are available on platelet behaviour after stopping thienopyridine treatment.

**Platelet TF in DES treated patients – 10° GSP**

- During dual antiplatelet therapy TF-positive platelets are significantly higher than in medically treated (MT) patients.
- TF-positive platelets further increase at T1 and T2 (ASA only) remaining significantly higher than those of MT patients.
Follow up of DES–treated patients

Of the 48 patients enrolled, 16 (33%) underwent a *de novo* PCI on another coronary lesion over a 3 year period of time.

Patients who underwent a new PCI within three years (□) had TF levels in *resting platelets* significantly higher than patients who were event–free (○).

Patients' stratification according to the TF positive platelet value 30 days after clopidogrel withdrawal

- Carotid Artery Stenting (CAS) is an evolving method to treat carotid stenosis.
- Platelet activation in CAS occurs as a result of vessel wall damage and subendothelium exposure.
- The dual antiplatelet regimen has a significant impact on reducing adverse neurological outcomes.

No data on platelet activation during dual antiplatelet therapy and after thienopyridine discontinuation have been examined in CAS.
To compare platelet activation in patients who underwent carotid revascularization (n=38) versus stable angina patients who underwent coronary revascularization (n=16) with bare metal stent (BMS) implantation.

**Study Aim & Design**

- **Platelet activation markers:**
  - GPIIb/IIIa activated complex (PAC1)
  - P-Selectin (CD62P)
  - Tissue Factor (TF)
  - Percentage of total monocyte-platelet aggregates
  - Percentage of TF-positive monocyte-platelet aggregates

- Platelet activation markers were assessed by whole blood flow cytometry in resting conditions and after *in vitro* ADP stimulation (10 µM, 15 min).

**Methods**

- **Platelet activation markers during DAT—Resting conditions**

- **Platelet Tissue Factor expression during DAT—Resting conditions**
Platelet Tissue Factor expression during DAT – ADP-stimulated

Platelet activation markers during ASA – Resting conditions

Platelet Tissue Factor expression during ASA – Resting conditions

Platelet Tissue Factor expression during ASA – ADP-stimulated
• Significant higher levels of TF-positive platelets and TF-positive MPA were observed in peripheral blood of coronary patients who underwent revascularization with BMS implantation compared to patients with carotid artery stenting, both 1 month after stenting and 1 month after thienopyridine discontinuation.

• This prothrombotic platelet phenotype may have implications for thrombotic complication in coronary patients.

Conclusions