PESTO

Morphological Parameters Explaining Stent Thrombosis assessed by OCT

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Conflits d’intérets :

- Consultant : Saint-Jude Medical, Terumo, Abbott

ACS with ST+ coronarography in acute phase (<12h) due to stent thrombosis
(all types of stents, acute;subacute;late and very late)

Optimal thrombectomie
(mechanical and/or médics : thrombo-aspiration, anti GpIIb-IIIa...)

TIMI 3 flow obtained

Consent patient

OCT faisable

If NO at 1 of 3 items :
No enrollment OCT, registry

YES

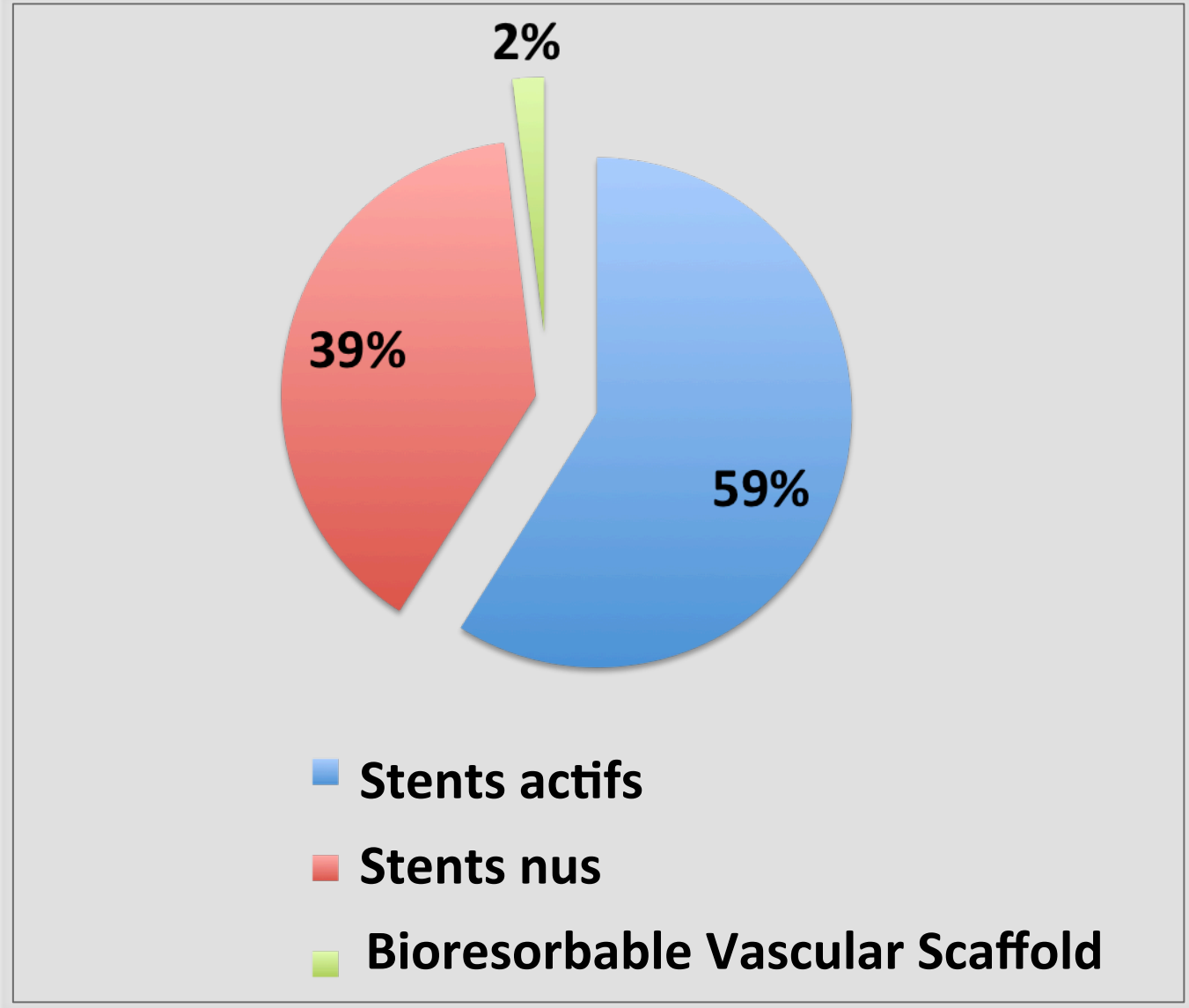
Note diagnose hypothesis and management before OCT
Immediate OCT or defered (MIMI, D1-D30) decided by the operator
Treatment guided by OCT

Caractéristiques patients

- N=120 sur 134 patients ont été inclus dans l'analyse finale (3 patients (2.4%) ont été exclus pour qualité d'imagerie non appropriée)

Age (yrs)	61.6 ± 1.1
Male gender (%)	89
Previous ACS (%)	68
Active smoking (%)	35
Dyslipidemia (%)	86
Hypertension (%)	56
Diabetes (%)	28
Recent (<15 d) modification of antiplatelet therapy (%)	22
Presentation mode:	
STEMI (%)	82
NSTEMI (%)	17
Unstable Angina (%)	1

Type de stents



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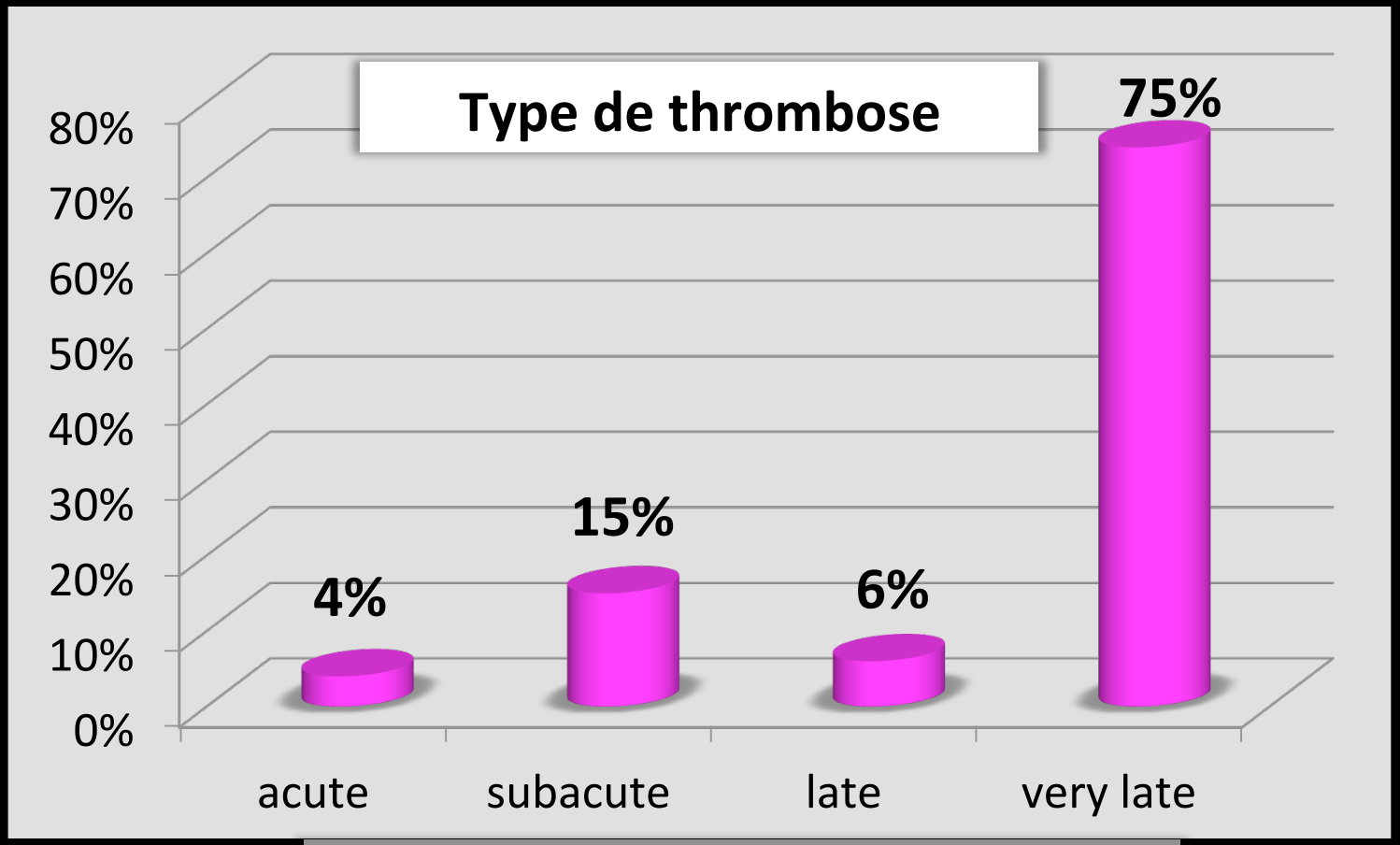
Analyse de 120 patients



Délai Médian 4.3 ans

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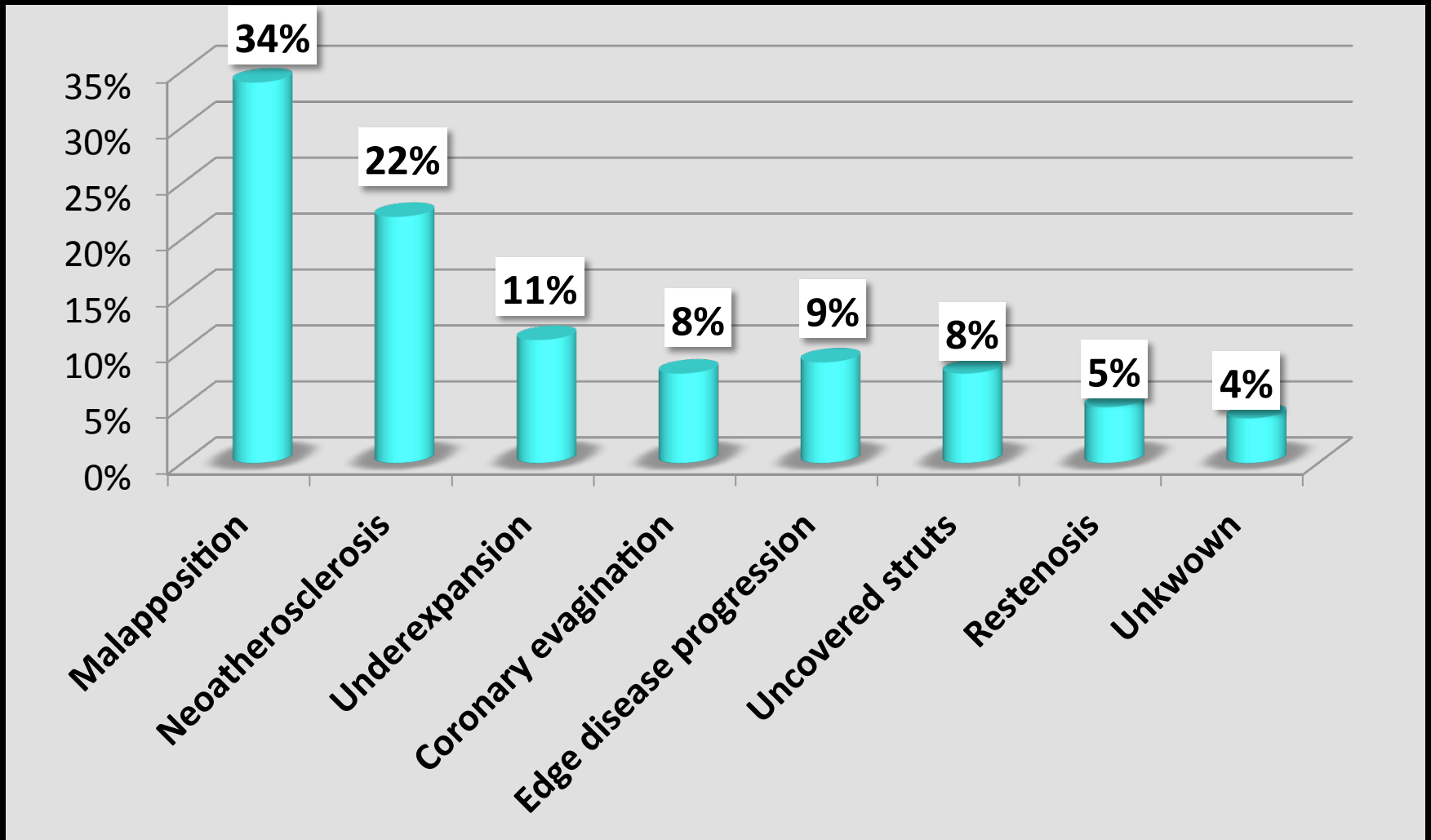
Anomalies morphologiques

Anomalie morphologique retrouvée dans 97% cas

Resultats : analyse OCT de 120 patients



Mécanismes de thrombose de stent en fonction du délai

	Global (n=120)	Acute+ Subacute ST (n=23)	Late+ Very Late ST (n=97)	p
Malapposition (%)	34.2	47.8	30.9	0.12
Ruptured Neoatherosclerosis (%)	22.5	**0**	**27.8**	**0.004**
Underexpansion (%)	10.8	26.1	7.2	0.02
Coronary Evagination (%)	8.3	0	10,3	0.11
Edge related disease progression (%)	9	4.3	8.2	0.45
Isolated uncovered struts (%)	8.3	0	10.3	0.11
Neointimal hyperplasia (%)	5	0	5.2	0.34
Edge dissection (%)	1	4.3	0	0.19
No cause identified (%)	4	**13**	**1**	**0.02**

Mécanisme de thrombose en fonction du type de stent

	BMS (n=47)	DES (n=71)	p
Acute + Subacute ST (%)	19.1	18.3	0.91
Late + Very Late ST (%)	80.9	81.7	0.91
Index PCI to ST delay (y)	6.5±0.9	3.1±0.4	<0.001
Malapposition (%)	31.9	35.2	0.71
Ruptured Neoatherosclerosis (%)	36.2	14.1	0.005
Underexpansion (%)	6.4	12.7	0.22
Coronary Evagination (%)	2.1	12.7	0.04
Edge related disease progression (%)	12.8	4.2	0.09
Isolated uncovered struts (%)	4.3	11.3	0.16
Neointimal hyperplasia (%)	4.3	4.2	1.0
Edge dissection (%)	0	1.4	0.61
No cause identified (%)	0	5.6	0.13

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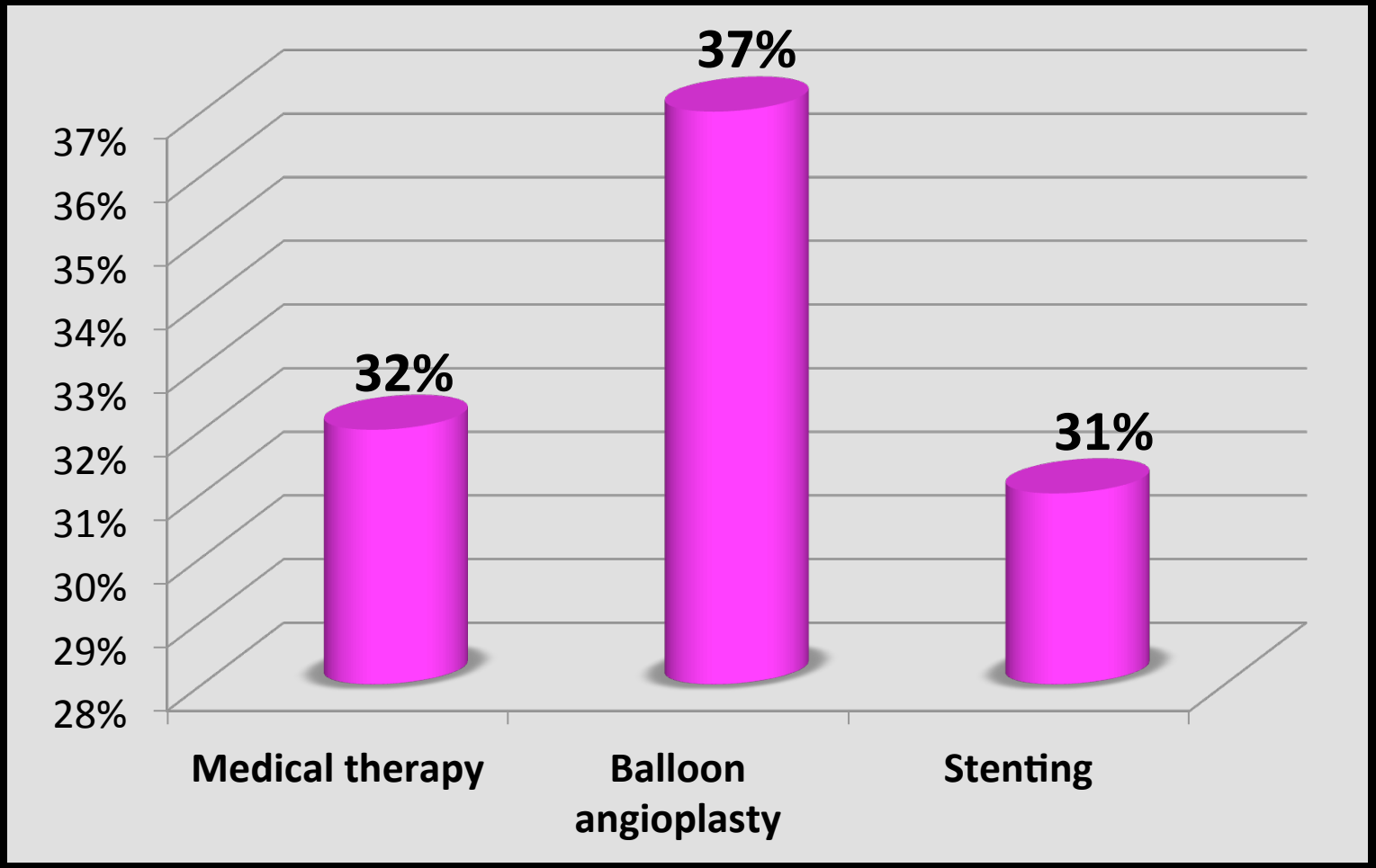
Décision thérapeutique

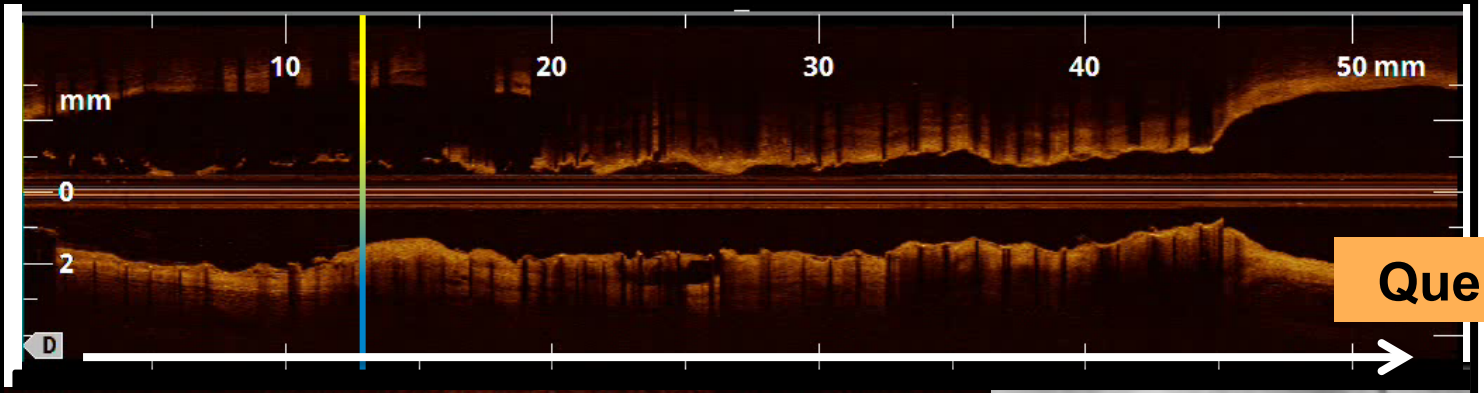
Analyse 120 patients



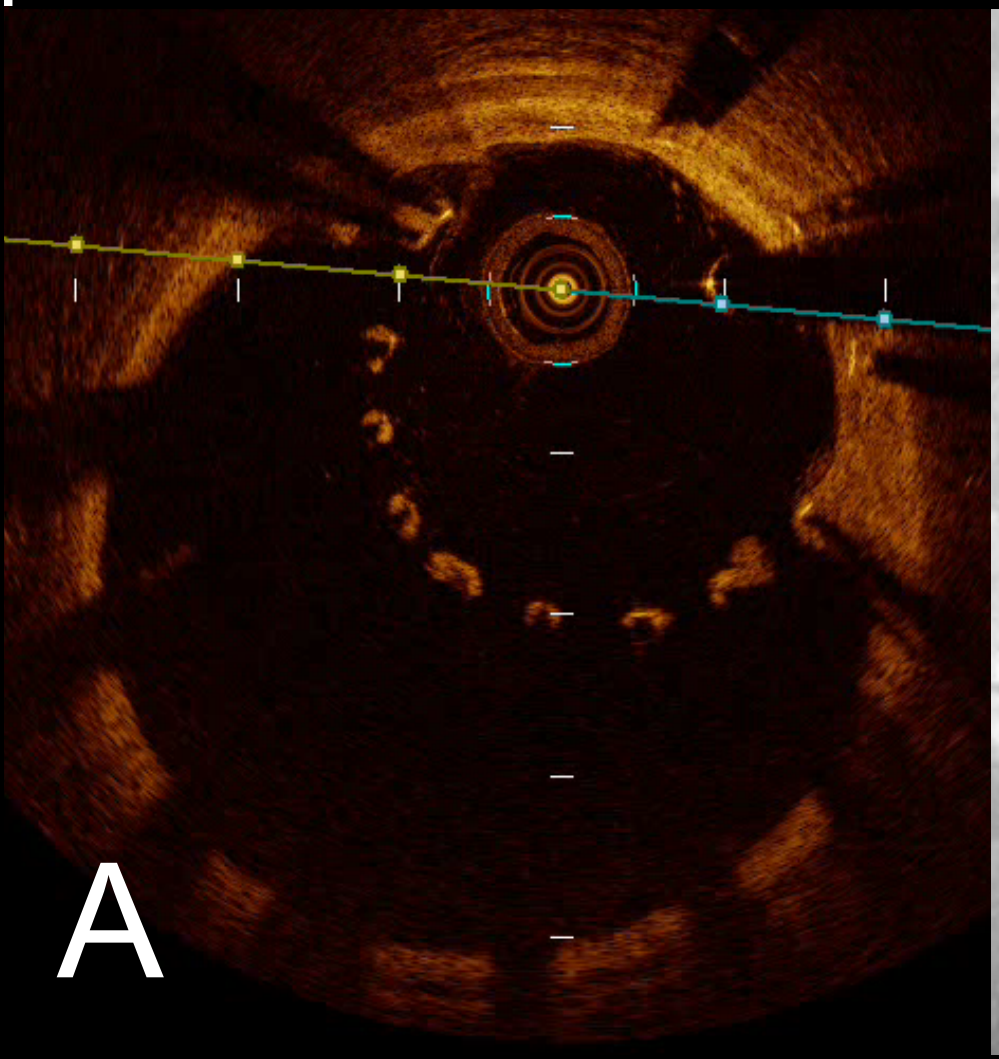
Analyse 120 patients

Cause	Coronarography
Undetermined	42 %
Probable	36 %
Certain	12 %

L'OCT influence la prise en charge dans 55% des cas



Quelques exemples



PESTO Study

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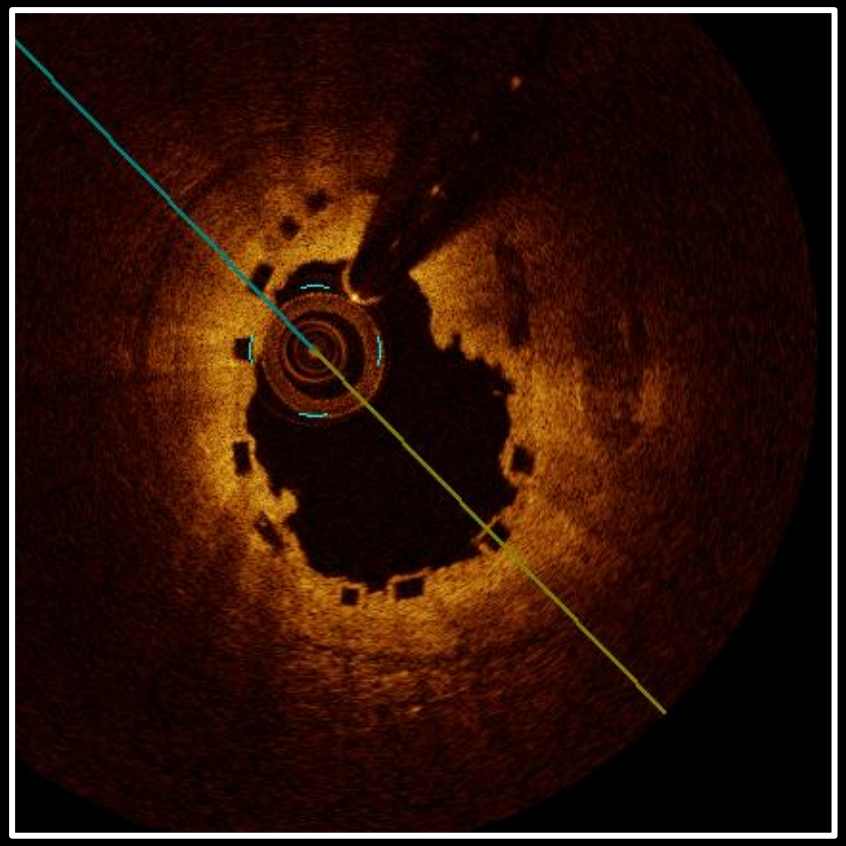
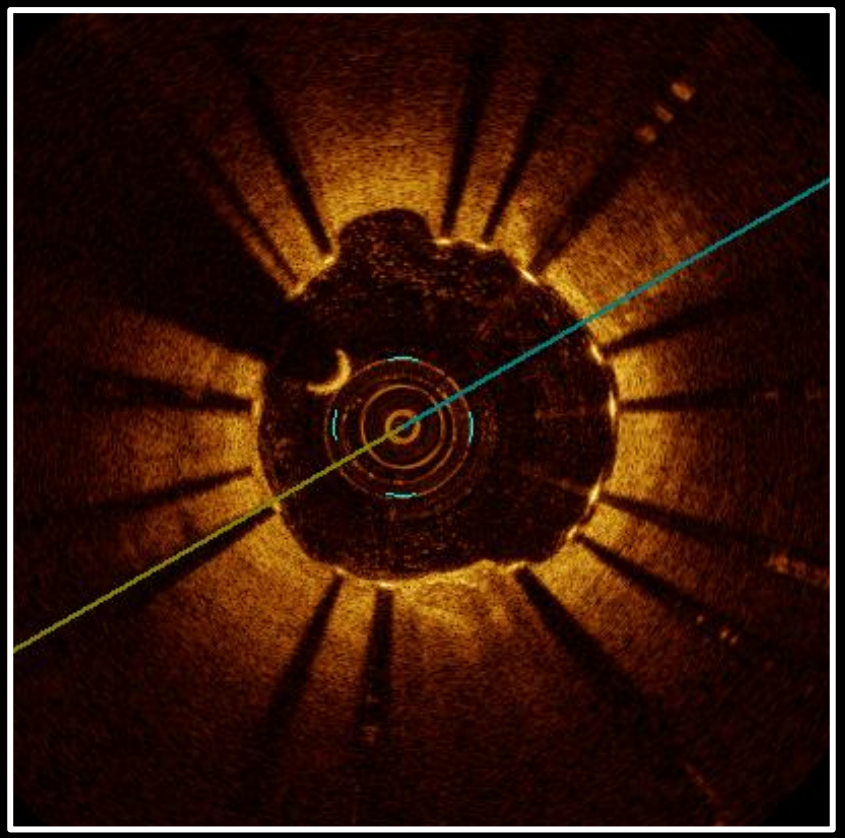
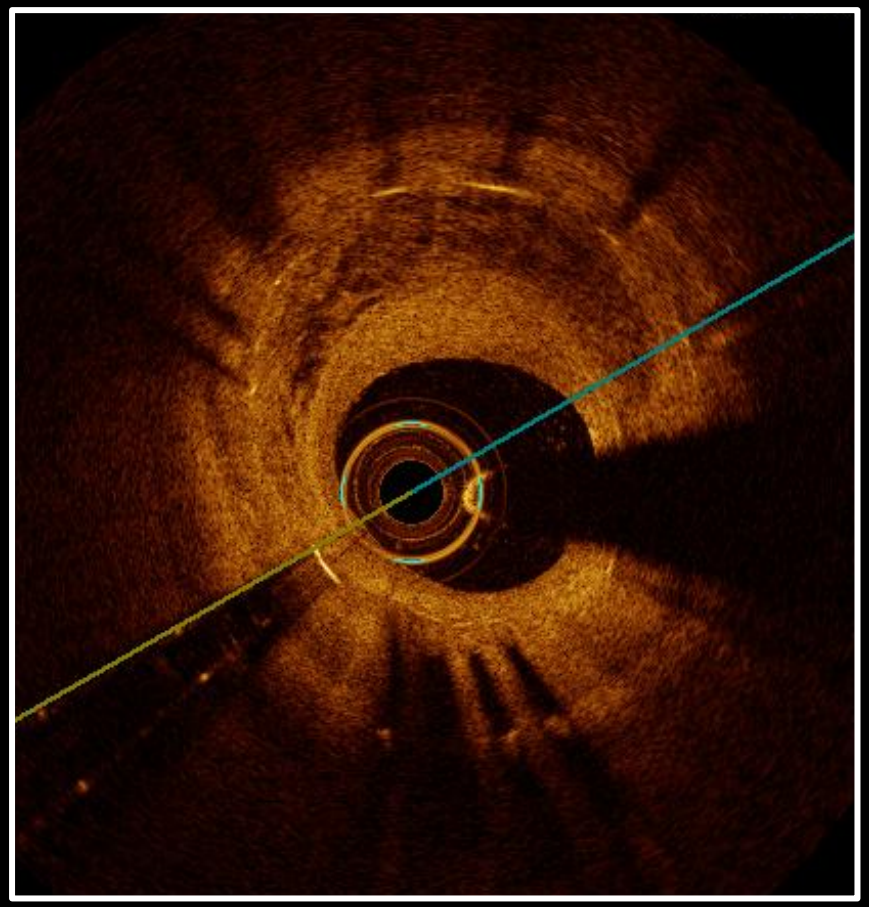


BVS sous-expansion

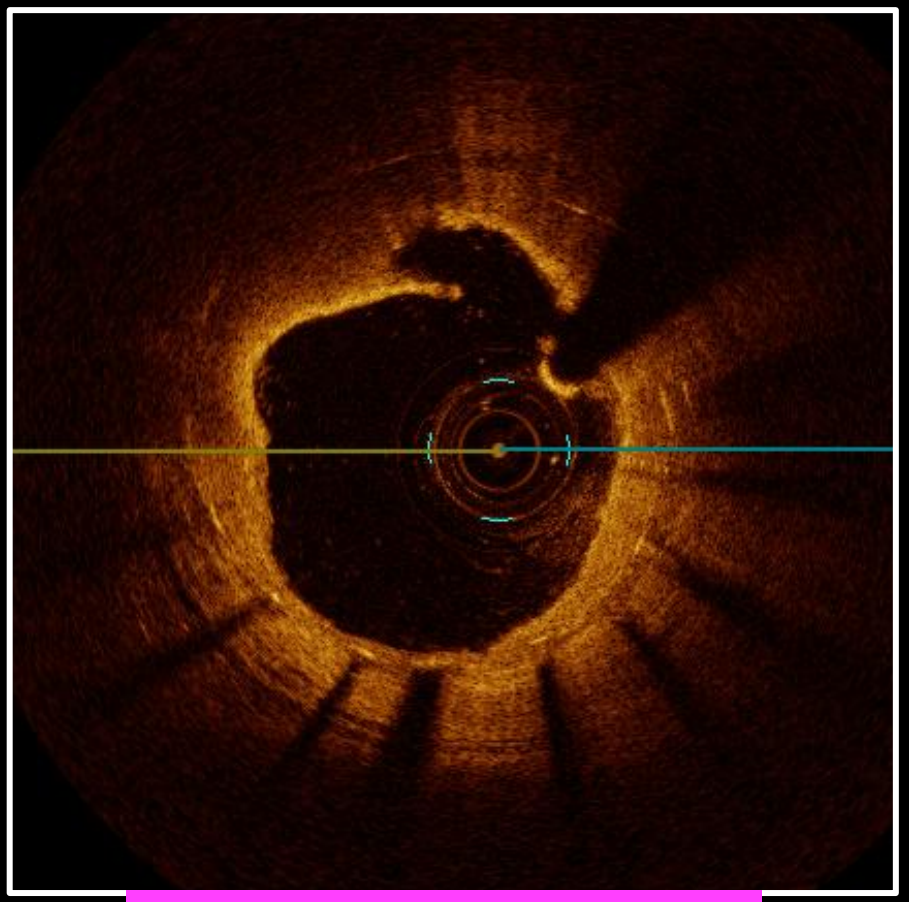


Struts non couvertes

PEST



Resténose avec néo-athérosclérose



Rupture de plaque avec neoatherosclerose

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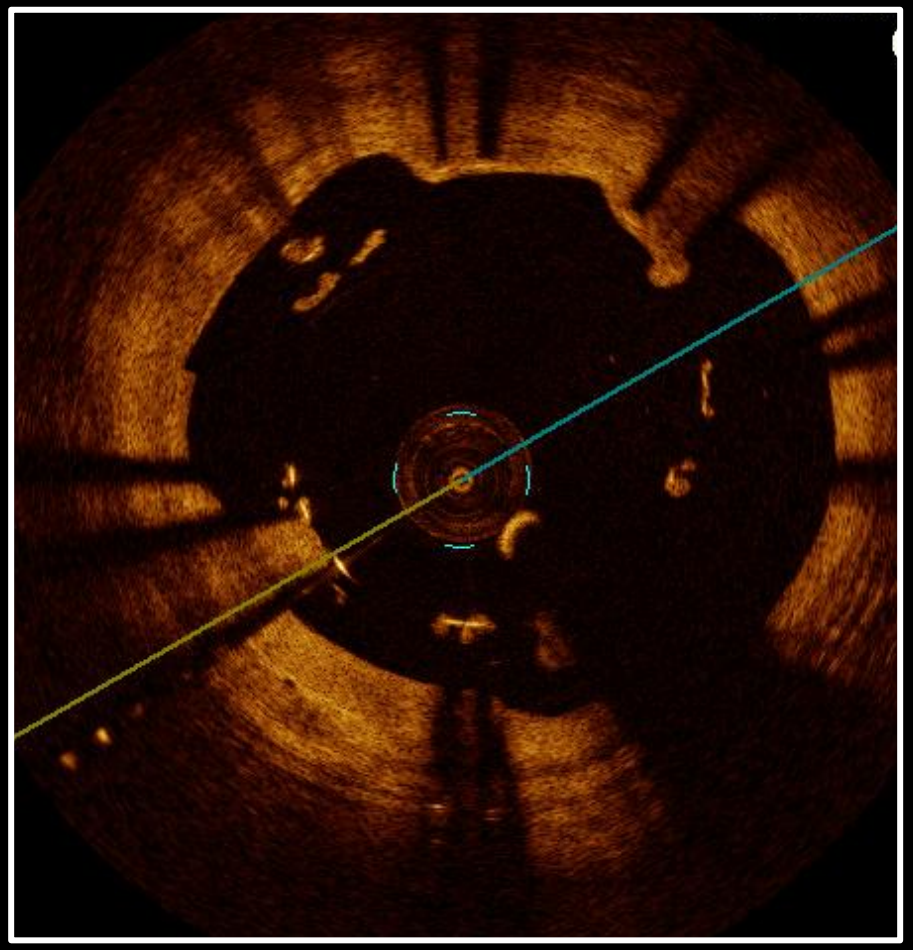
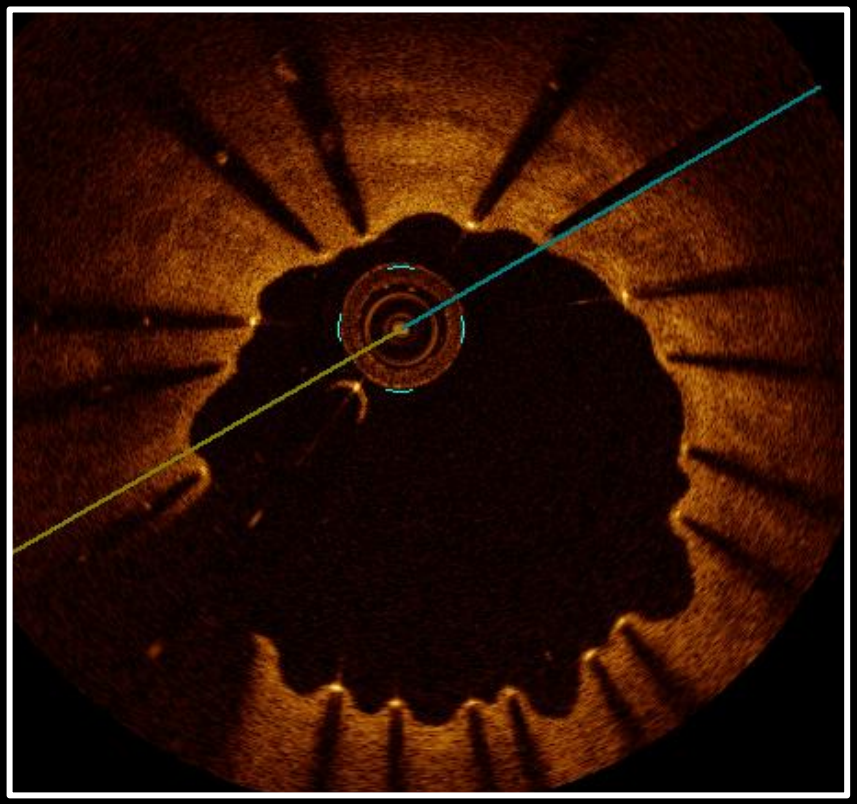
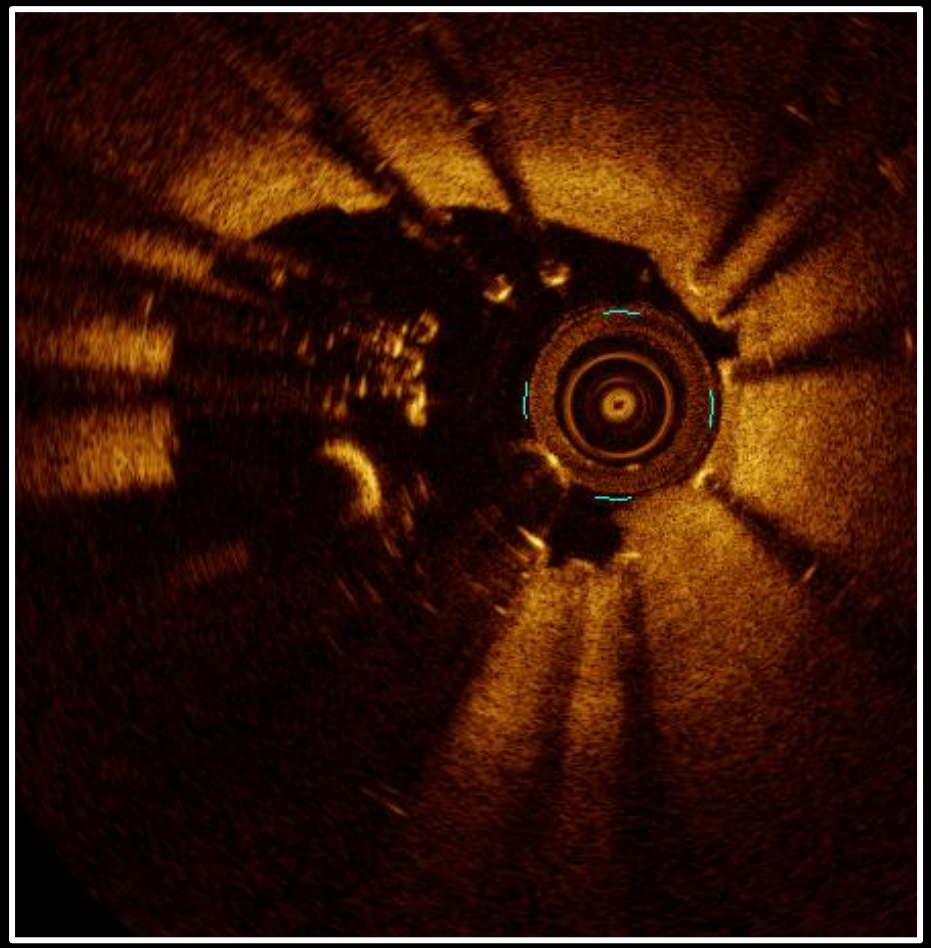


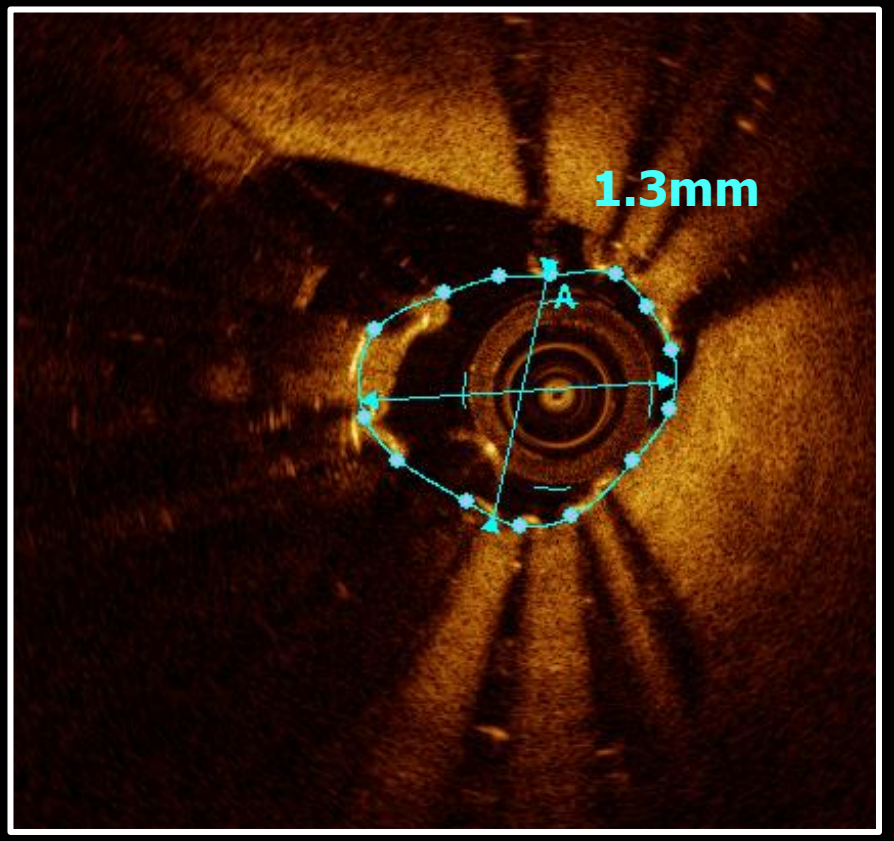
Malapposition



Mailles non couvertes et évagination

PEST





DES sous-expansion

Conclusion

Limites de PESTO

Pas de données sur la résistance à l'anti-agrégation plaquettaire

Tous les patients présentant une thrombose de stent n'ont pas été inclus dans l'étude durant la période (délais tardifs, pas de rétablissement flux TIMI 3..)

Limites de l'OCT : lésions distales, lésions non franchies

Conclusion

La thrombose de stent

L'angiographie ne semble pas suffisante dans la majorité des cas

Les mécanismes de thrombose de stent sont multifactoriels mais

- L'OCT aide au diagnostic et à la prise en charge

- La malapposition est la 1ère anomalie morphologique dans PESTO

- Avec l'aide de l'OCT un traitement médical est choisi dans 1 cas/3 dans PESTO

PEST

134 patients inclus

Merci aux centres

Merci

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STENT THROMBOSIS AND OCT

OCT Analysis in Patients With Very Late Stent Thrombosis

33 very late stent thrombosis
- **27 DES and 6 BMS**
- **61 months after implantation for DES – 109 for BMS**

OCT in acute phase– No thrombo-aspiration

First OCT system with proximal occlusion

	DES	BMS	
Proportion of frames with at least 1			
Uncovered strut, %	12.9 ± 15.5	0.5 ± 1.3	0.072
Malapposed strut, %	7.8 ± 11.1	0.0 ± 0.0	0.050
Proportion of lesions with at least 1			
Frame with uncovered strut	15 (56)	1 (17)	0.085
Frame with malapposed strut	14 (52)	0 (0)	0.020

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STENT THROMBOSIS AND OCT

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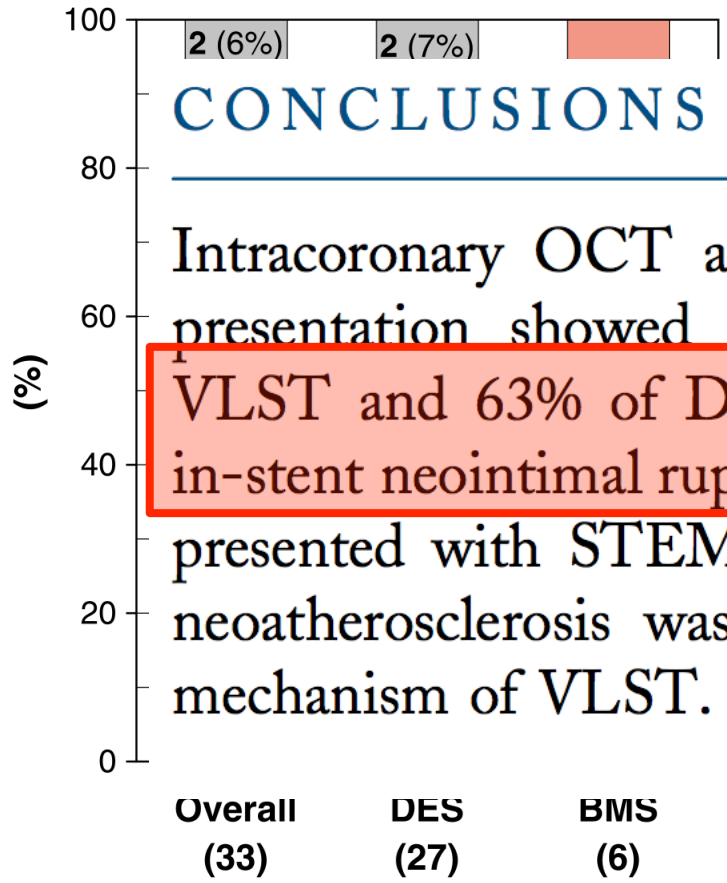

CONCLUSIONS

Intracoronary OCT at the time of acute clinical presentation showed that all BMS lesions with VLST and 63% of DES lesions with VLST had in-stent neointimal rupture; and two-thirds of them presented with STEMI, suggesting that advanced neoatherosclerosis was a common and aggressive mechanism of VLST.

al rupture

n BMS

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