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APPAC
ACTUALISATIONS ET PERSPECTIVES EN PATHOLOGIE CARDIOVASCULAIRE

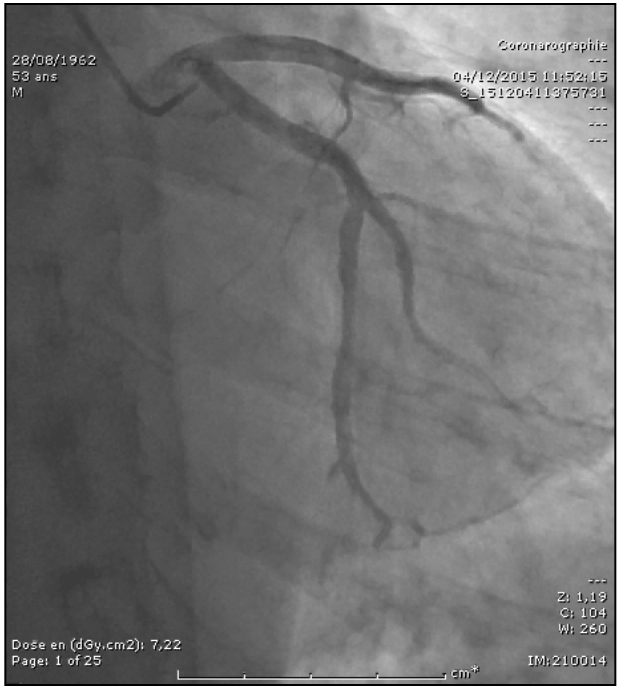
LE BOA AVAIT TROP MANGE

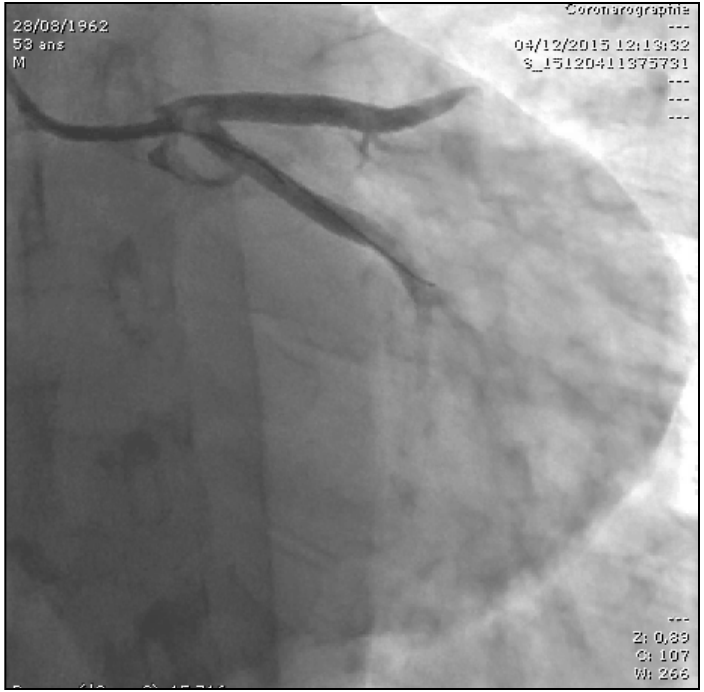
F. DE POLI CH Haguenau

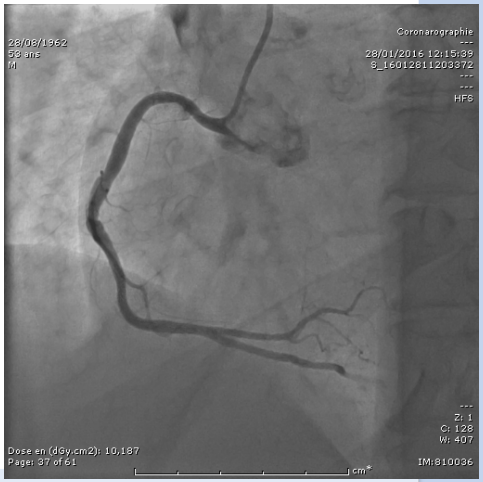
Cas clinique 1

- M. 58 ans, admis la nuit en USIC pour SCA ST- Tropo + (0,3µg/l n<0,15),
- Angor crescendo avec douleurs de repos,
- FDR : tabac,
- ECG : T- en inf , écho normale,
- Coro par voie radiale en 6F le lendemain (15/12/15).

Cas clinique 1



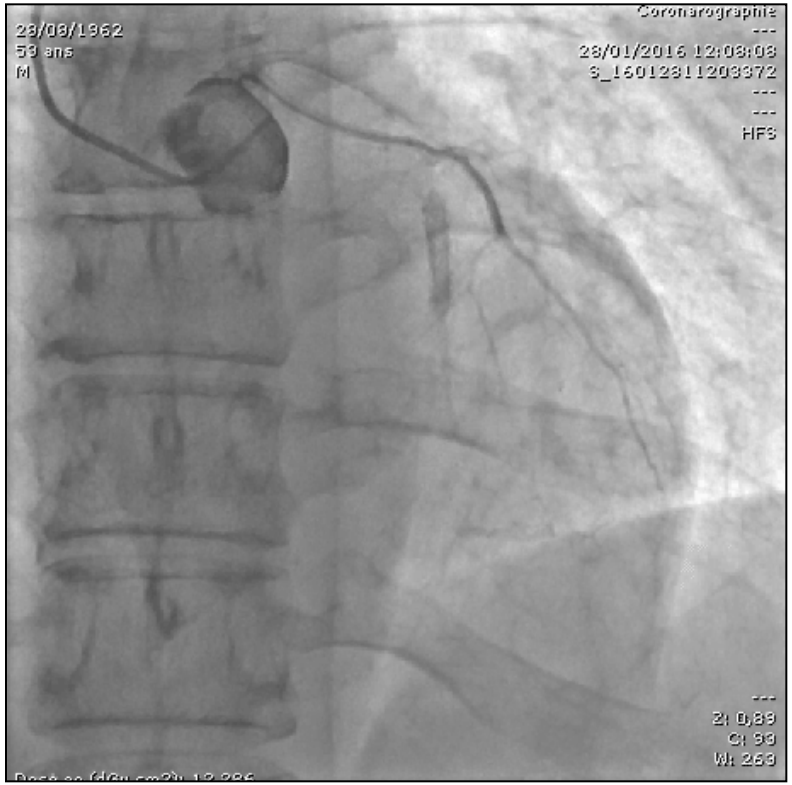


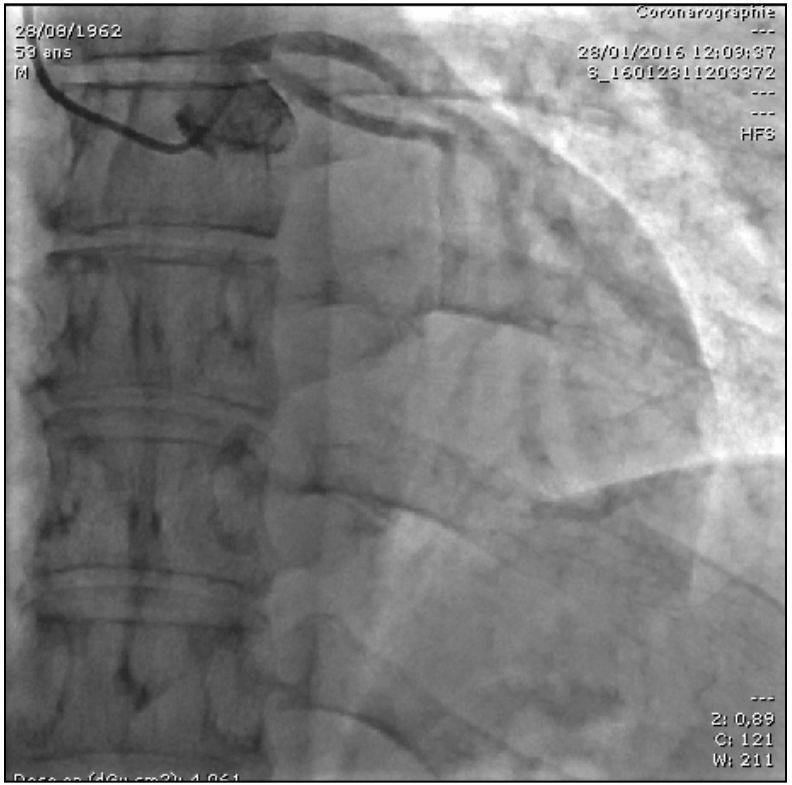


Cas clinique 1

- Un mois plus tard (le 27/01/2016) ré-adressé par le service de réadaptation fonctionnelle pour persistance de douleurs angineuses et test d'effort litigieux,
- Ttt en cours : Duoplavin, Bisocé 1,25 mg,
  Tahor 10 mg,
- Coro par voie radiale en 6F.MEOPA car patient tres anxieux

Cas clinique 1





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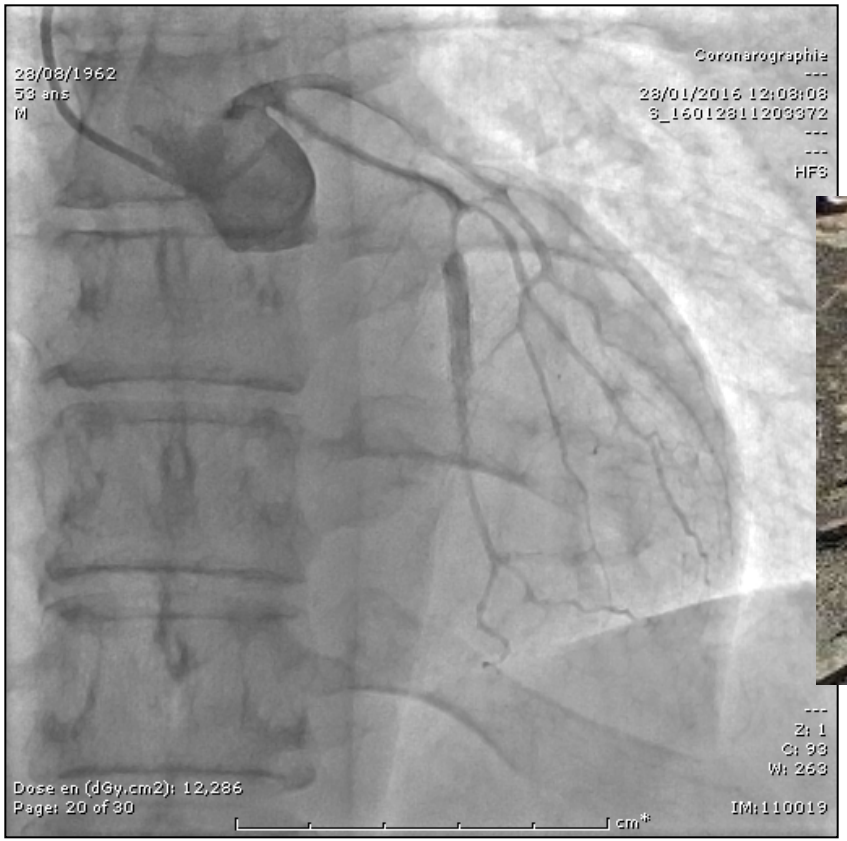

Spasme coronaire

- Fréquence variable selon les ethnies,
- Mécanismes multiples : dysfonctions endothéliales, hyper-réactivité des cellules musculaires lisses vasculaires, polymorphismes phénotypiques, rôle de l'inflammation et de ses médiateurs, rôle des plaquettes,
- La présentation classique (douleur de repos, avec sus-ST transitoire, chez une femme fumeuse, avec des coronaires saines) n'est pas toujours respectée.

Le spasme coronaire

- Rôle des sondes dans la provocation (taille, intubation profonde, voie d'abord, courbure),
- Test de provocation quand, comment et dans quelles conditions:
  - IV ou intra-coronaire,
  - par voie radiale ou par voie fémorale,
  - diagnostique ou pour évaluation du traitement,
- Maladie masquée (traitement vasodilatateur lors des coro par voie radiale, ttt anti HTA),
- Spasme radiale et coronaire intriqué?

Le spasme coronaire

- Pour mieux comprendre:

Eur Heart J. 2015 Aug 4. pii: ehv351. [Epub ahead of print]

International standardization of diagnostic criteria for vasospastic angina.

Beltrame JF[1], Crea F[2], Kaski JC[3], Ogawa H[4], Ong P[5], Sechtem U[5], Shimokawa H[6], Bairey Merz CN[7]; Coronary Vasomotion Disorders International Study Group (COVADIS).

Author information

Abstract

The Coronary Vasomotion Disorders International Study Group (COVADIS) was established to develop international standards for the diagnostic criteria of coronary vasomotor disorders. The first symposium held on the 4-5 September 2013 addressed the criteria for vasospastic angina, which included the following (i) nitrate-responsive angina, (ii) transient ischaemic electrocardiogram changes, and (iii) documented coronary artery spasm. Adoption of these diagnostic criteria will improve the clinical diagnosis of this condition and facilitate research in this field.

KEYWORDS: Coronary artery disease; Coronary artery spasm; Ischaemic heart disease; Vasospastic angina

Contemporary Reviews in Cardiovascular Medicine

Mechanisms of Coronary Artery Spasm

Gaetano Antonio Lanza, MD; Giulia Careri, MD; Filippo Crea, MD

The term coronary artery spasm (CAS) refers to a sudden, intense vasoconstriction of an epicardial coronary artery that causes vessel occlusion or near occlusion. Although CAS may be involved in other coronary syndromes, it represents the usual cause of variant angina.

The variant form of angina was first described in 1959 by Prinzmetal et al,[1] who used this term to indicate that angina attacks, unlike the most common form of effort angina, occurred at rest and were associated with ST-segment elevation, rather than ST-segment depression, on the ECG (Figure 1). Because myocardial ischemia occurred in the absence of any change in myocardial oxygen demand, the authors hypothesized that it was caused by an increased tonus of vessels at the level of coronary stenoses.[1]

Some years later, in fact, coronary angiography, performed during spontaneous angina attacks, demonstrated that CAS is the usual cause of variant angina.[2–4] Coronary angiography also showed that CAS could occur at the site of a stenosis (either minor or severe) or in angiographically normal coronary arteries,[5] usually at a localized segment of an epicardial artery (focal spasm) (Figure 2).[6] However, sometimes CAS involves 2 or more segments of the same (multifocal spasm) or of different (multivessel spasm) epicardial coronary arteries, or may also involve diffusely one or multiple coronary branches.[7]

Careful assessment of clinical history and 24- to 72-hour ambulatory ECG monitoring are usually sufficient to achieve the diagnosis of variant angina, whereas the use of provocative tests of CAS (eg, intravenous ergonovine, intracoronary ergonovine, or acetylcholine administration) is required in about 10% of patients.[7]

Transmural myocardial ischemia caused by occlusive CAS can be complicated by malignant ventricular arrhythmias,[8,9] which can result in sudden death, or, if prolonged, by acute myocardial infarction.[10] Accordingly, a prompt diagnosis would be essential to prevent these serious complications, even though calcium antagonists are very effective in preventing CAS.[11,12] However, the diagnosis of variant angina is often overlooked for several months after its manifestation.[7]

Sixty years after the first description of variant angina, the causes and the mechanisms of CAS are still poorly defined. The research in this field has indeed been refrained by several factors, including the low incidence of the disease and the considerable efficacy of nonspecific vasodilator therapy.[12]

However, in 10% to 20% of patients, CAS is refractory to standard treatment, or high doses of calcium antagonists are needed to effectively prevent its recurrence. Thus, elucidating the mechanisms responsible for CAS could make treatment of difficult or refractory cases easier.[13]

In this article, we review the state of knowledge regarding the etiopathogenesis of CAS in patients with the clinical syndrome of Prinzmetal variant angina.

Pathogenetic Substrate of CAS

CAS results from the interaction of 2 components: (1) a usually localized, but sometimes diffuse, abnormality of a coronary artery that makes it hyperreactive to vasoconstrictor stimuli, and (2) a vasoconstrictor stimulus able to induce the spasm at the level of the hyperreactive coronary segment (Figure 3).

The mechanisms proposed to constitute the substrate for susceptibility to CAS include (1) endothelial dysfunction, (2) a primary hyperreactivity of vascular smooth muscle cells (VSMCs), and (3) other factors.

Endothelial Dysfunction

The endothelium has a crucial role in the physiological regulation of coronary vascular tone, mainly through the release of several vasodilators, the most important of which is nitric oxide (NO). Therefore, a significant endothelial damage might impair vasodilation, thus favoring CAS in response to vasoconstrictor stimuli.[14]

It is important to observe that various vasoactive stimuli (eg, acetylcholine, serotonin, histamine) cause vasodilation by inducing NO release by the endothelium, but, at the same time, they may cause vasoconstriction through direct stimulation of VSMCs. Thus, in the presence of endothelial dysfunction, their release in the vessel wall can lead to vasoconstriction or CAS.[15]

Experimental support of the possibility that endothelial dysfunction can play a major role in CAS can derive from an earlier porcine model developed by Shimokawa et al,[16] in which the authors managed to make a coronary segment susceptible to spasm in response to serotonin and histamine by the association of endothelium removal and high-cholesterol feeding, which also promoted the formation of atherosclerotic plaques in the vasospastic segment.

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Circulation is available at http://circ.ahajournals.org DOI: 10.1161/CIRCULATIONAHA.111.037283

Spasme coronaire

- **Critères diagnostics COVADIS** :
  - Angor spontané :
    - Répondant aux dérivés nitrés avec au moins un des critères suivants:
      - Angor de repos (particulièrement nocturne ou du petit matin),
      - Variation marquée de la tolérance diurne à l'exercice (réduite le matin),
      - Hyperventilation peut précipiter un épisode,
      - Inhibiteurs calciques font disparaitre les symptômes.

    Et

    - Modifications per-critiques de l'ECG (dans au moins 2 dérivations contiguës) :
      - Sus ou sous décalage ≥ à 0,1 mV,
      - Nouvelles ondes U négatives.

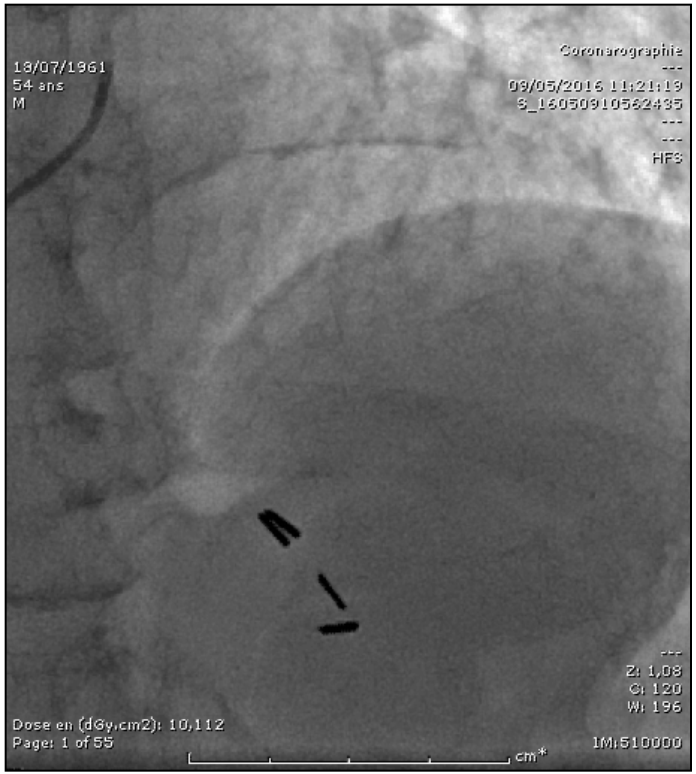
    Ou

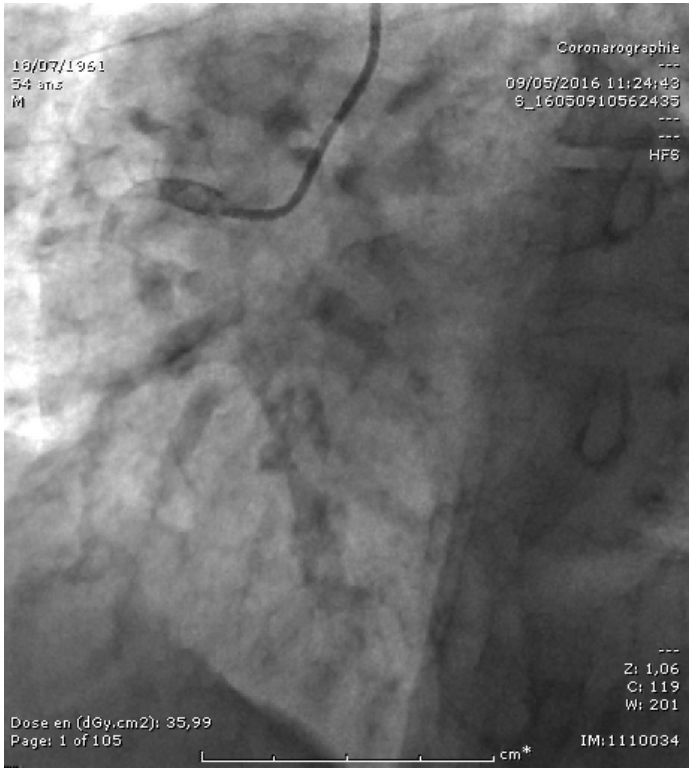
  - Test de provocation de spasme :
    - Occlusion totale ou subtotale coronaire (>90%) **et** douleurs angineuses **et** modifications ECG.

Cas clinique 2

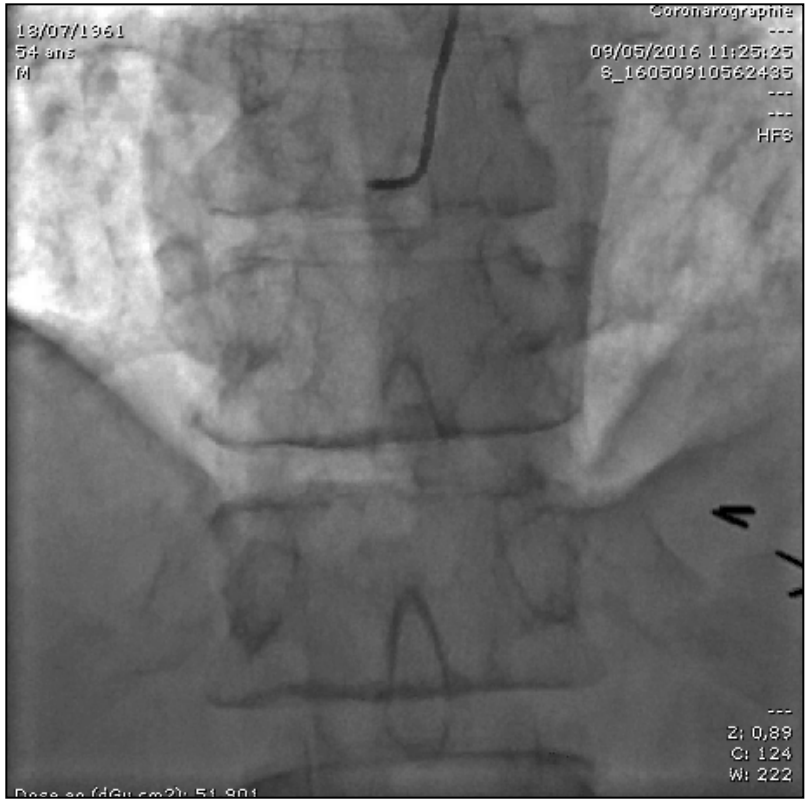
- M. 53 ans : adressé pour coro car découverte fortuite lors d'un examen de routine de séquelles IDM inf. à l'ECG avec une cinétique conservée en échographie,
- FDR : HTA, dyslipidémie, surcharge pondérale, tabac actif 60pa,
- ATCD : éthylisme chronique non sevré, splénectomie post traumatique, trouble érectile depuis 2 ans, SEP depuis 4 ans,
- Patient tachycarde dès le début de l'examen à 120 bpm.

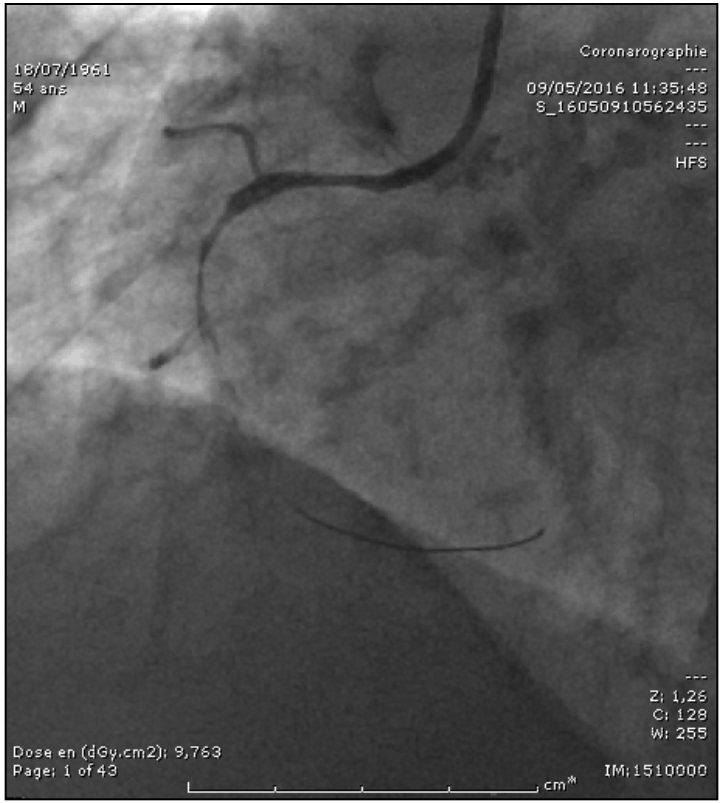
Cas clinique 2





Cas clinique 2





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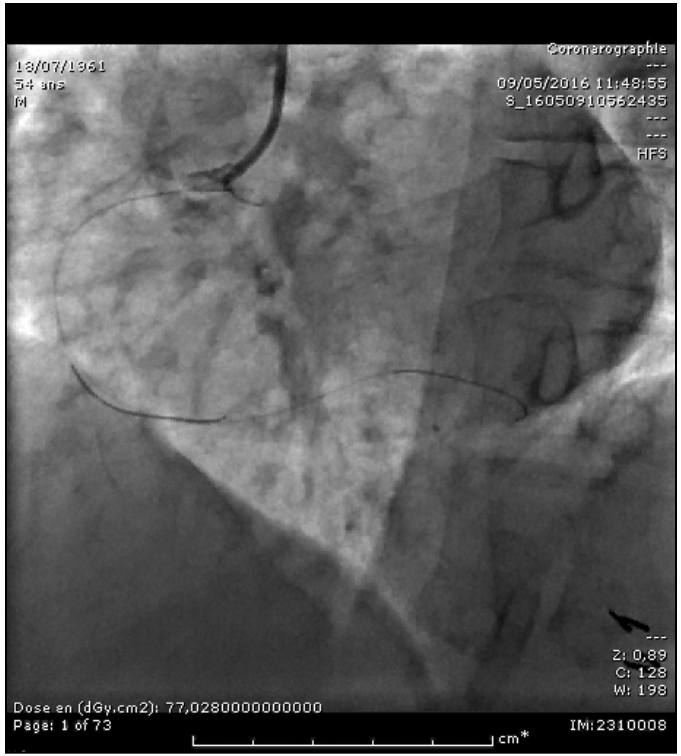

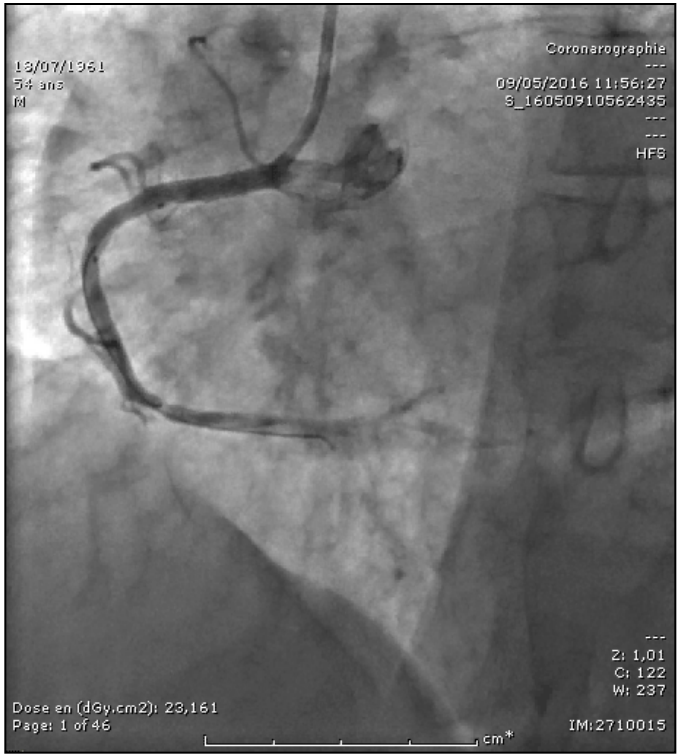

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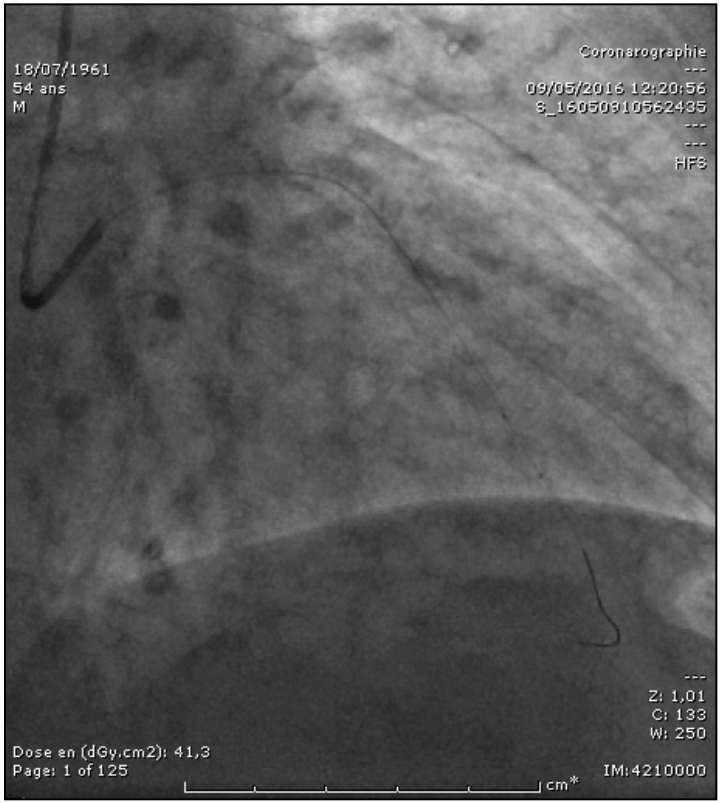

Evolution

- Rapidement favorable : un jour de réa, retour à domicile à J5,
- Élévation des tropo significative,
- Pas de séquelles en échographie,
- Bilan allergologique en cours.

Conclusion

- Un train peut en cacher un autre (rôles des histamines dans le spasme).
- Se méfier du réflexe occulo-sténotique.
- Faut-il refaire des tests de provocation à ces patients ?

Conclusions

- Et comme dirait Sabrina :

«Il t ‘arrive toujours de ces trucs…»