

# **Paradigm shift in Acute Aortic Syndrome.**

**From pathology to bedside.**

**Isidre Vilacosta MD. Madrid, Spain.**

**55th Annual Scientific Meeting of The  
Korean Society of Cardiology.**

**Daejeon, Korea. December 3, 2011.**

# Acute aortic syndrome: from pathology to bedside.

- Definition & classification.
- Clinical presentation & diagnostic algorithm.
- Anatomoclinical correlations in daily clinical practice:
  - Classic dissection.
  - Intramural aortic hematoma.
  - Penetrating aortic ulcer.
  - Incomplete dissection.

# AAS-Definition.

ht153650 Module 1 Heart 2/2/09 11:03:01

Topics:

Review

## Acute aortic syndrome: a new look at an old conundrum

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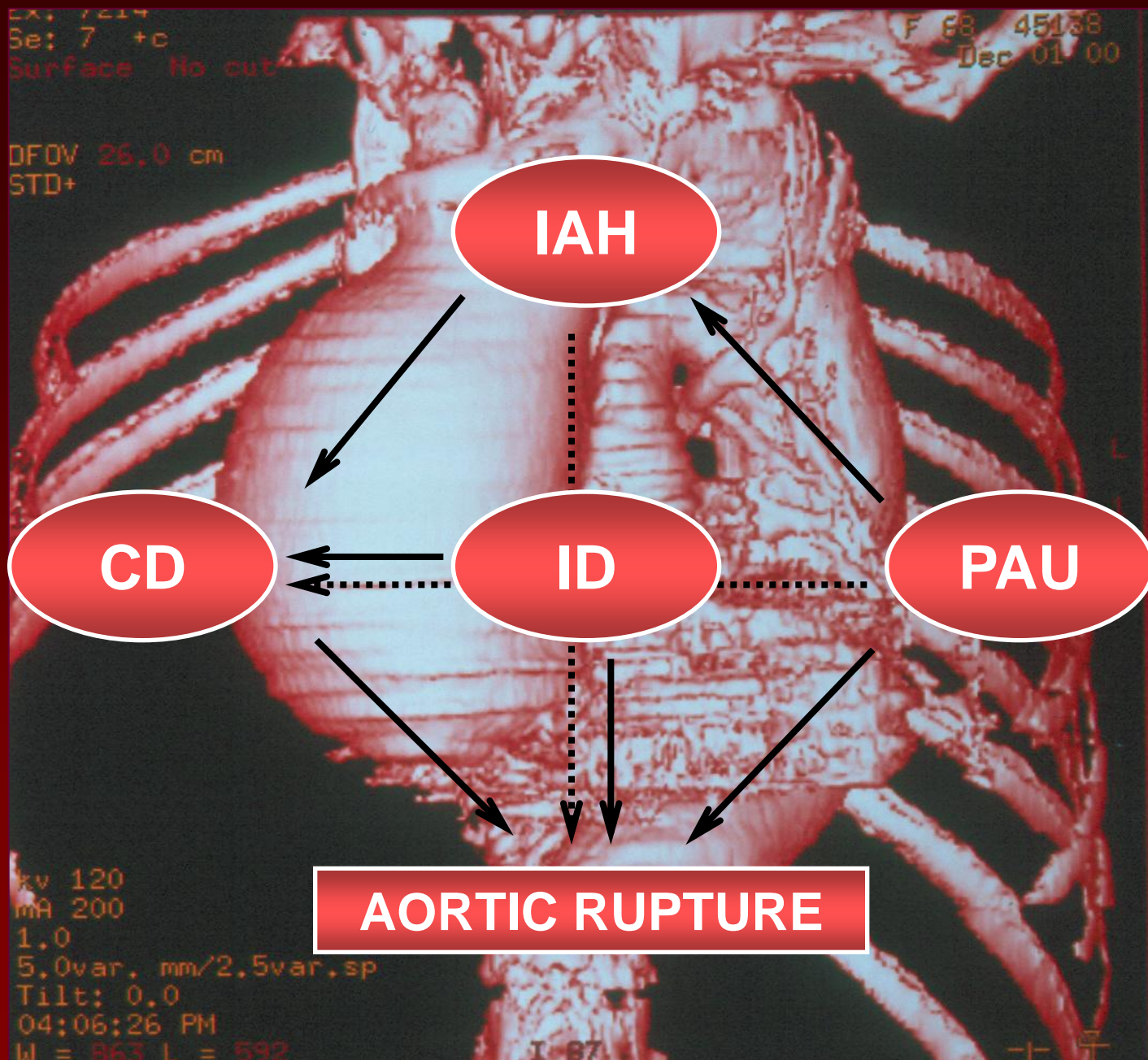
### ABSTRACT

The term acute aortic syndrome (AAS), coined several years ago, is now widely recognised. In the light of new findings in aortic pathology and in an era when modern imaging techniques are widely available and interventional management of AAS is increasing, some morphological and diagnostic aspects of acute aortic pathology have been examined and the syndrome updated. This article provides a new, comprehensive overview of the pathology, diagnosis, evolution and management of patients with AAS. As acute aortic disease is the most common fatal condition in patients with chest pain, prompt recognition and treatment is of paramount importance.

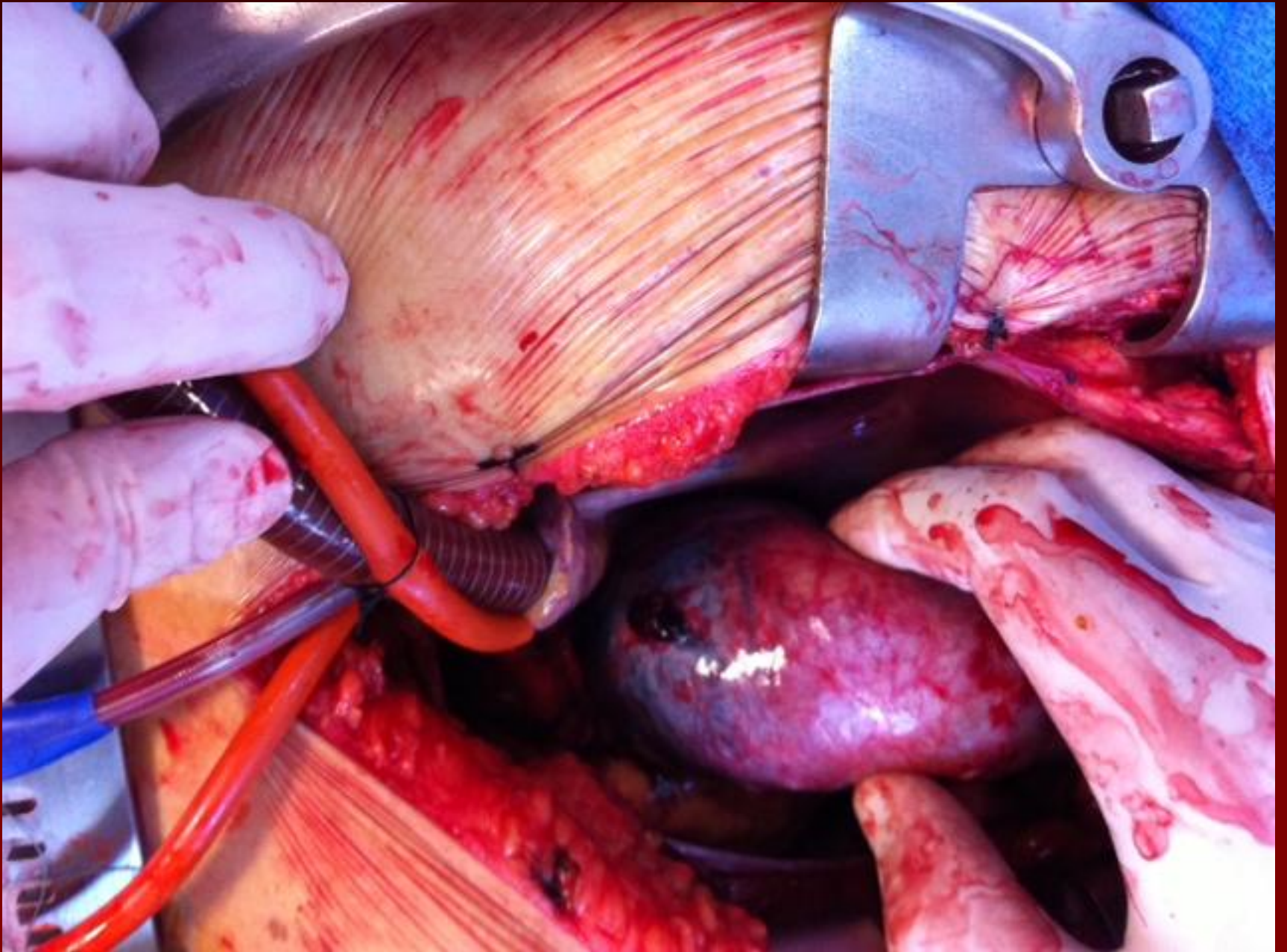
previous facts strongly suggest the existence of a link between them.

In this revision, the morphology, distinctive features, diagnostic clues, natural history (patterns of evolution) and management of these lesions are discussed. A systematic classification of acute aortic pathology seems advantageous to describe AAS more precisely, to better relate aortic morphological characteristics to surgical intervention and to gain more insight into the intimal relation of the four elements that comprise this syndrome. We have not considered aortitis and traumatic aortic rupture as part of AAS because they have a totally distinct clinical and pathophysiological profile.

Fig. 1



Vilacosta I, Aragoncillo P, Cañadas V, et al. Heart 2009



**AORTIC RUPTURE**

# AORTIC PAIN



### First Step

#### Clinical Suspicion of AAS

Aortic Pain + Hypertension or underlying connective tissue disease (Marfan Syndrome)



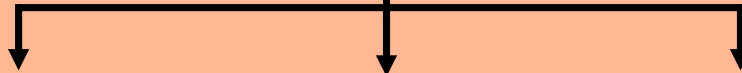
#### Physical Examination

Murmur of Aortic Regurgitation, Pulse deficits



### Second Step

#### Basic Evaluation



#### ECG

Absence of ischemic changes

#### Chest X-ray

Aortic dilatation

#### CPK, troponins, D-dimers

Normal myocardial enzymes

Elevated D-dimers

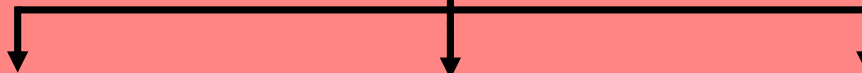


#### High Probability of AAS



### Third Step

#### Imaging evaluation: CT + TTE



#### Confirmed diagnosis

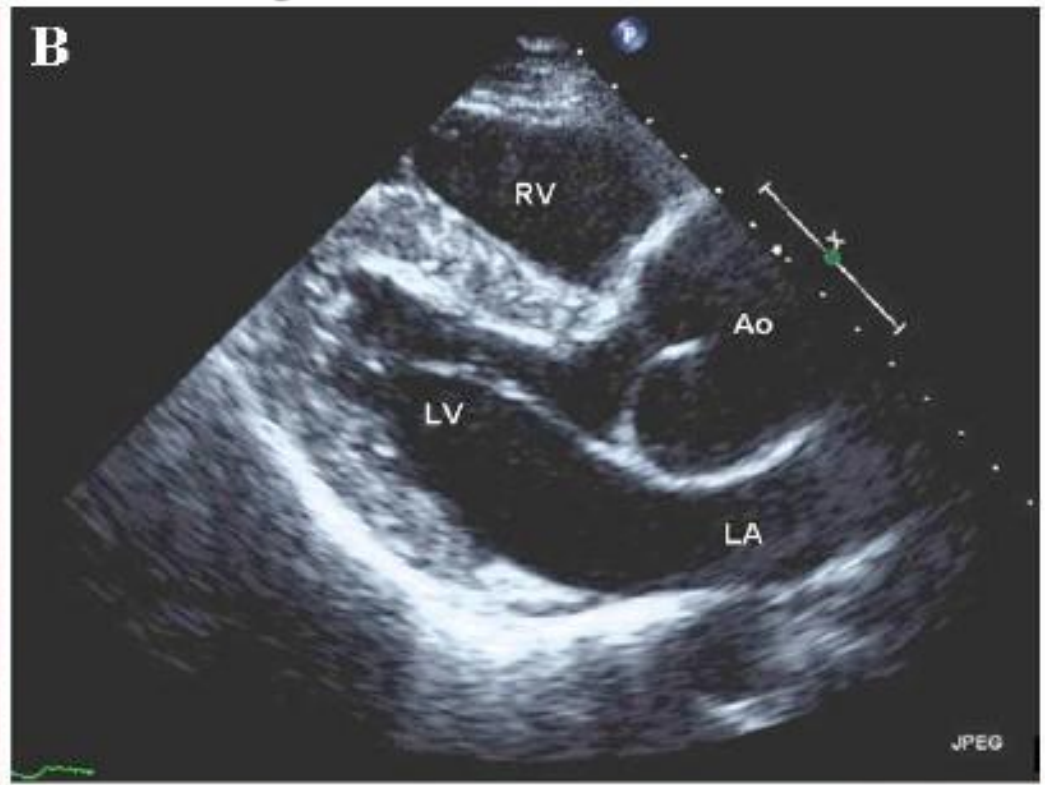
#### Excluded diagnosis

Consider other causes  
of  
chest pain

#### Non-conclusive diagnosis or diagnostic queries

TEE/MR

# Loeys-Dietz syndrome





### First Step

#### Clinical Suspicion of AAS

Aortic Pain + Hypertension or underlying connective tissue disease (Marfan Syndrome)



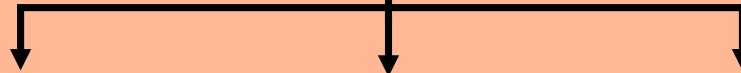
#### Physical Examination

Murmur of Aortic Regurgitation, Pulse deficits



### Second Step

#### Basic Evaluation



#### ECG

Absence of ischemic changes

#### Chest X-ray

Aortic dilatation

#### CPK, troponins, D-dimers

Normal myocardial enzymes

Elevated D-dimers

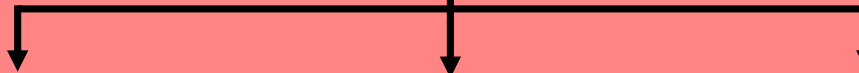


#### High Probability of AAS



### Third Step

#### Imaging evaluation: CT + TTE



#### Confirmed diagnosis

#### Excluded diagnosis

Consider other causes  
of  
chest pain

#### Non-conclusive diagnosis or diagnostic queries

TEE/MR

# D-dimers

- Degradation product of cross-linked fibrin.
- High sensitivity & NPV: 500 mcg/l.
- Low specificity. (Initial 6 h: >1600 ng/ml).
- Correlation with the extension of the lesion.
- Inverse relation: symptoms-test time & D-d.
- Evolution of D-dimer value during AAS.
- Acute vs chronic AAS. Predictor of mortality.

**Weber T, Chest 2003; Eggebrecht H, JACC 2004;**

**Ohlmann P, Crit Care Med 2006; Sodeck G. Eur Heart J 2007;**

**Suzuki T, (IRAD-Bio) Circulation 2009.**

### First Step

#### Clinical Suspicion of AAS

Aortic Pain + Hypertension or underlying connective tissue disease (Marfan Syndrome)



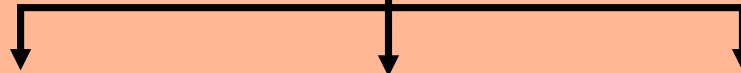
#### Physical Examination

Murmur of Aortic Regurgitation, Pulse deficits



### Second Step

#### Basic Evaluation



#### ECG

Absence of ischemic changes

#### Chest X-ray

Aortic dilatation

#### CPK, troponins, D-dimers

Normal myocardial enzymes

Elevated D-dimers

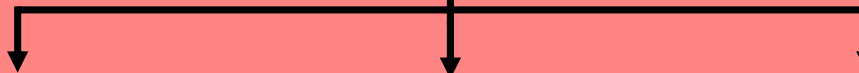


#### High Probability of AAS



### Third Step

#### Imaging evaluation: CT + TTE



#### Confirmed diagnosis

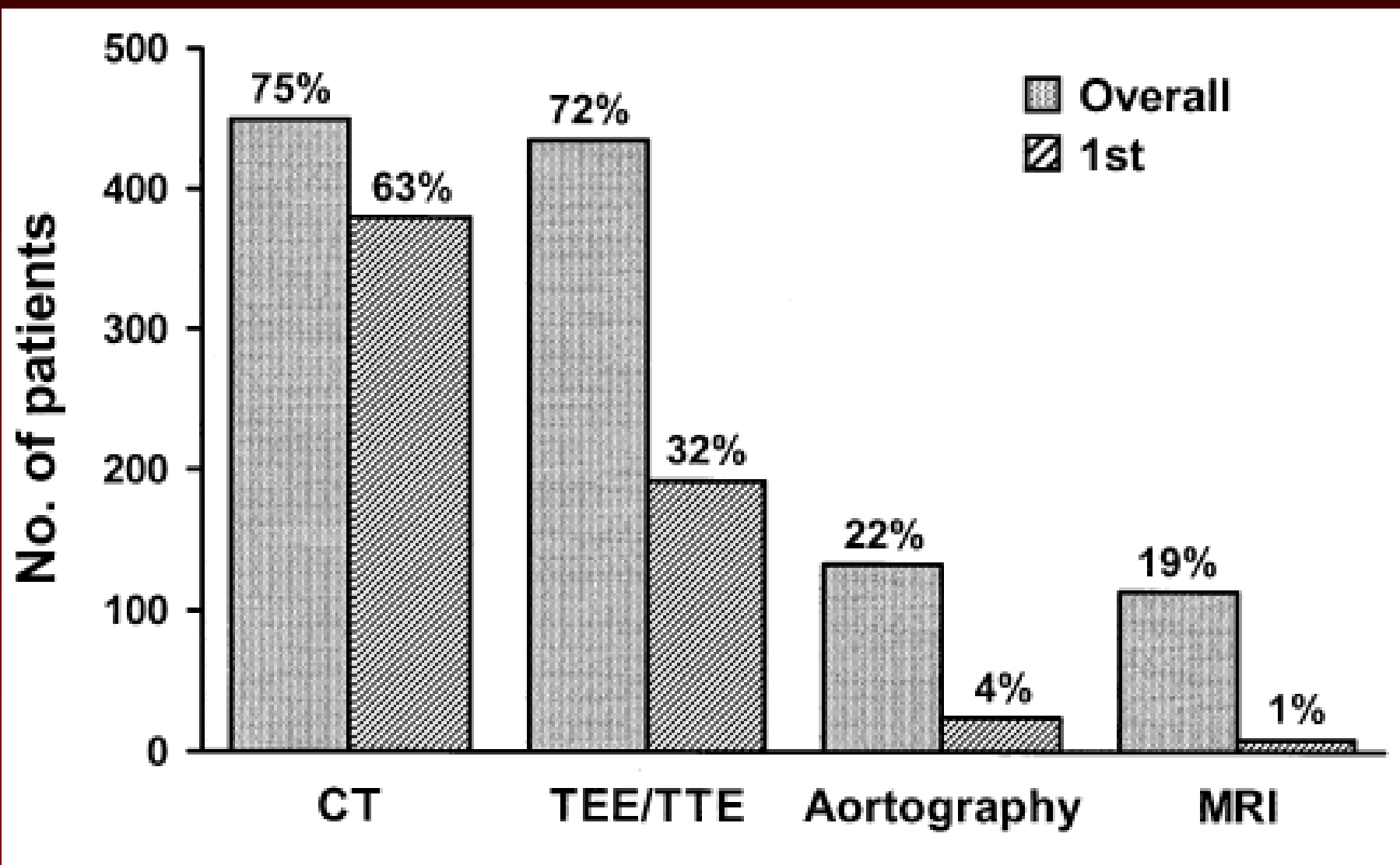
#### Excluded diagnosis

Consider other causes of chest pain

#### Non-conclusive diagnosis or diagnostic queries

TEE/MR

# IMAGING TECHNIQUES IN AAS.

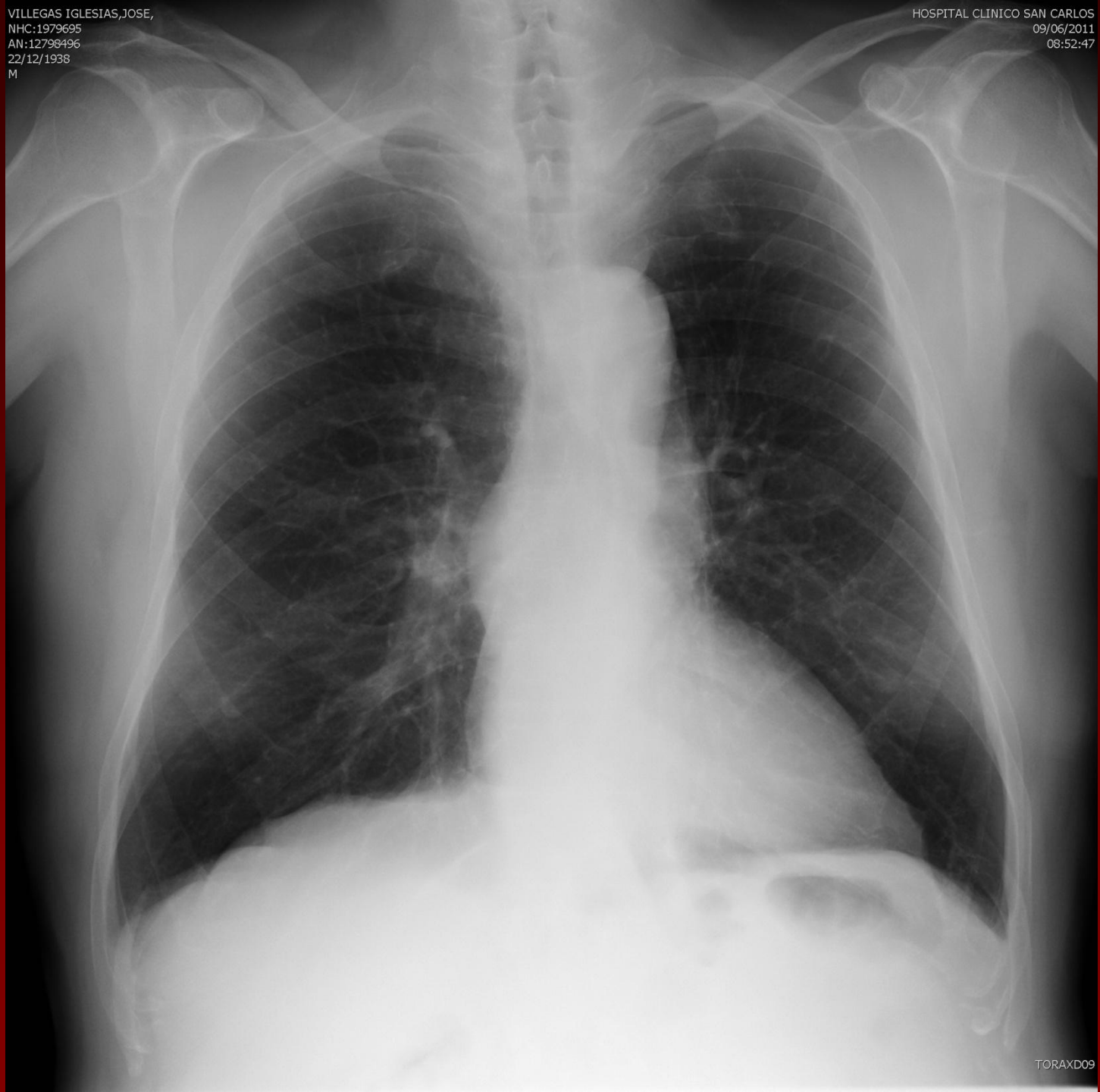


# Clinical scenario-1

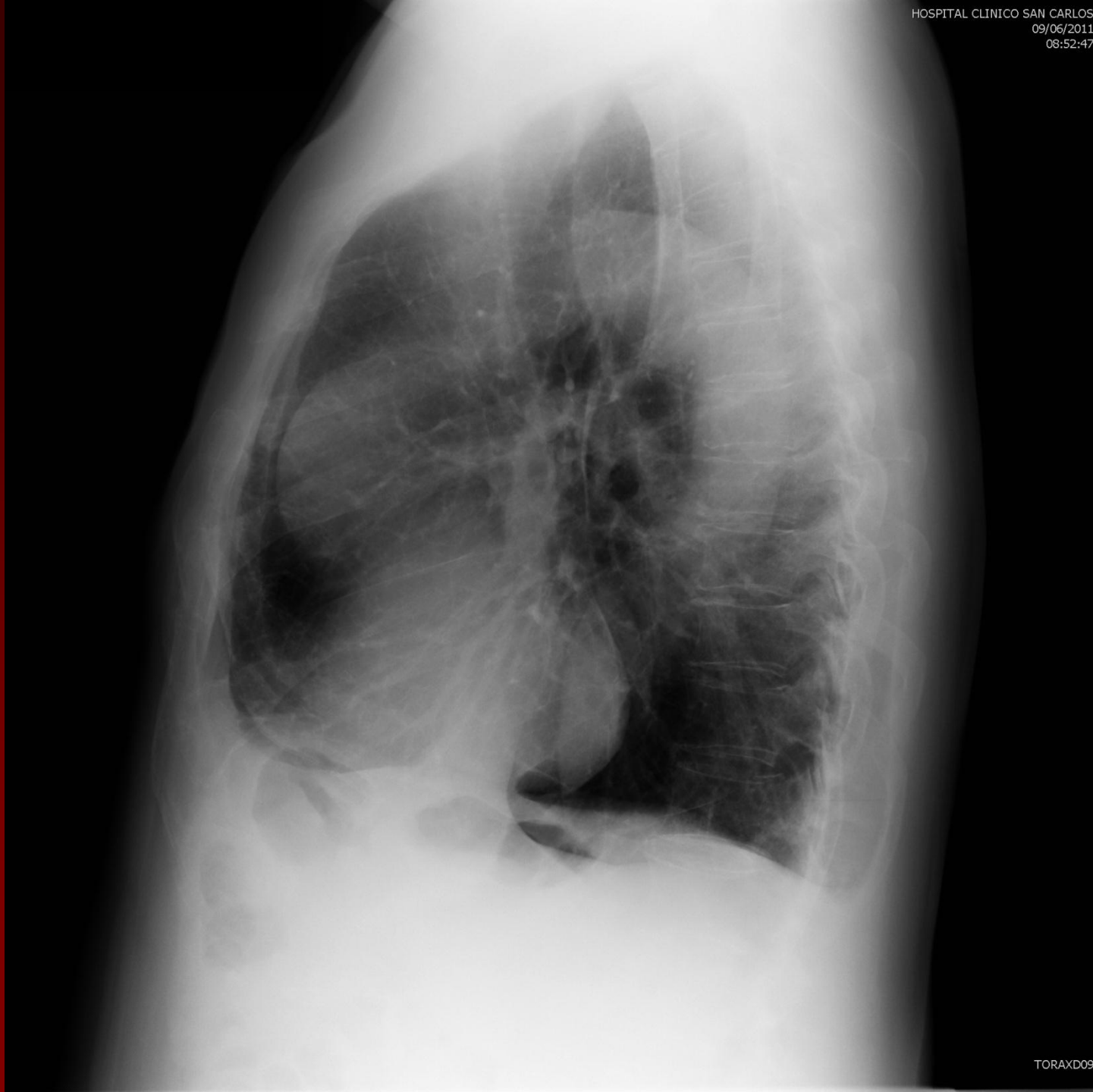
- 72 year-old male, HTA, smoker, COPD, DLP.
- Acute & intense chest pain (feb-2-2011):
  - **ER**: Normal EKG & enz.: atypical chest pain + respiratory infection.
- One month later: similar, but milder episode.
  - **ER**: Normal EKG & enzymes.
    - Normal physical exam.
    - Aortic dilation: X-ray & CT.
    - Lab.: Gl: 125 mg/dl, hemogram: N, **DD:4959**.
    - **Diagnosis**: Ao. aneurysm--> Surgery (6-22-11).

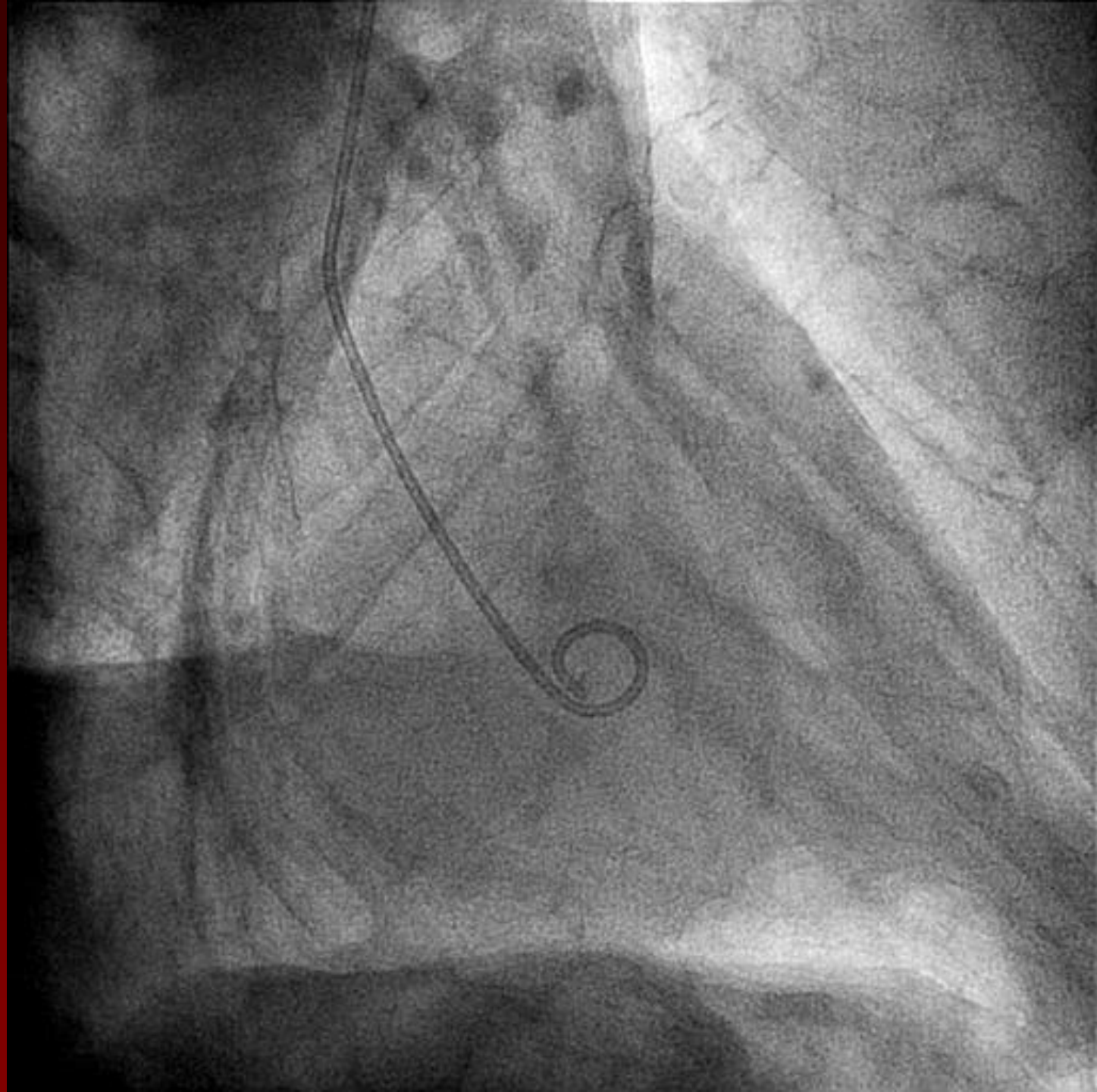
VILLEGAS IGLESIAS,JOSE,  
NHC:1979695  
AN:12798496  
22/12/1938  
M

HOSPITAL CLINICO SAN CARLOS  
09/06/2011  
08:52:47

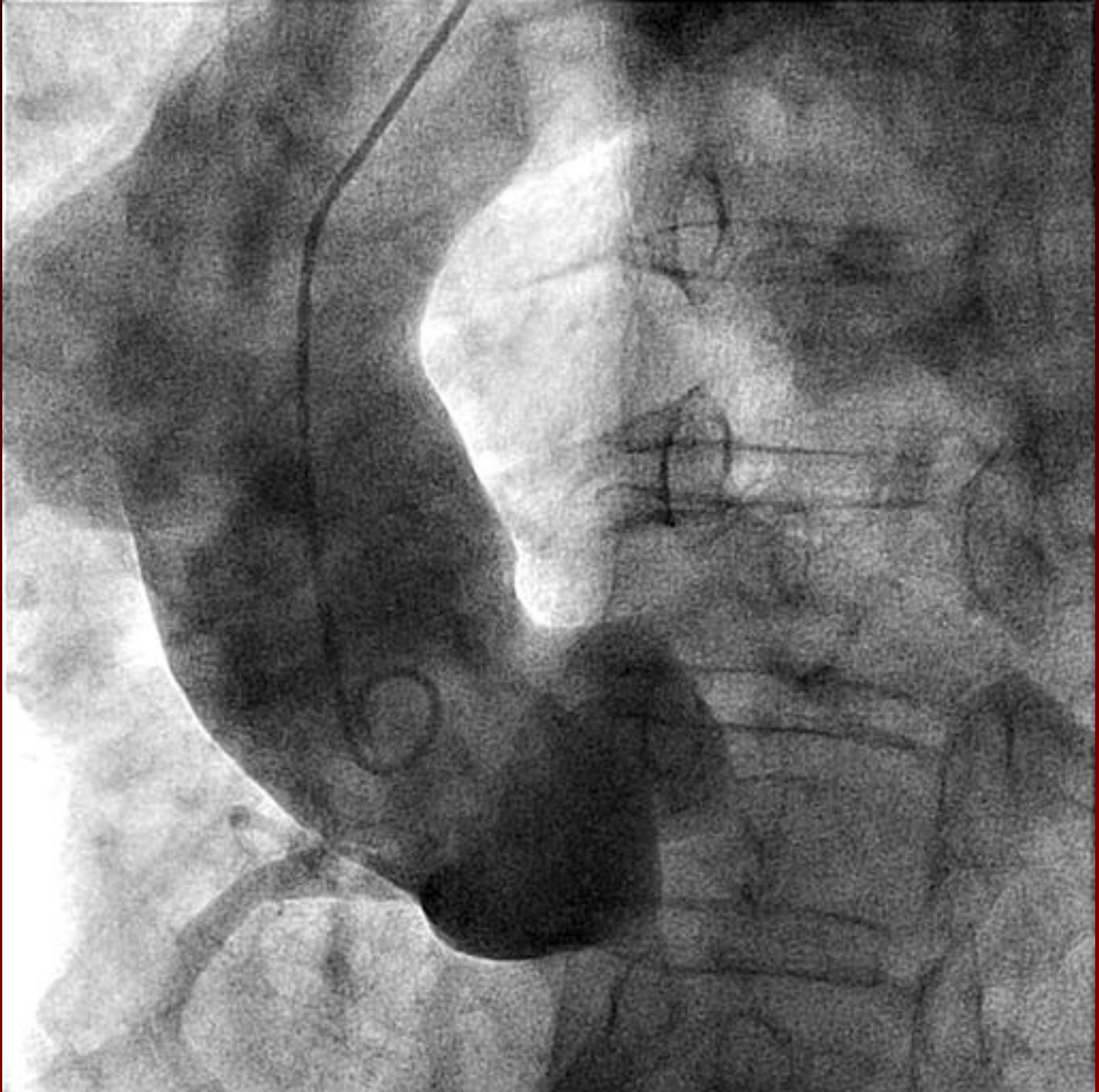


TORAXD09









H. C. SAN CARLOS  
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15/06/2011  
13:32:25  
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22/12/1938  
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R  
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L  
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738msec  
KV:120  
MA:108

IM:193  
SE:4  
ALG:B

pitch:

201.4  
1mm

P372

R  
2  
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L  
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3  
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738msec  
KV:120  
MA:108

IM:195  
SE:4  
ALG:B

pitch:

203  
1mm

P372

H. C. SAN CARLOS  
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738msec  
KV:120

IM:187  
SE:4

VILLEGAS IGLESIAS, JOSE,  
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22/12/1938  
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A64

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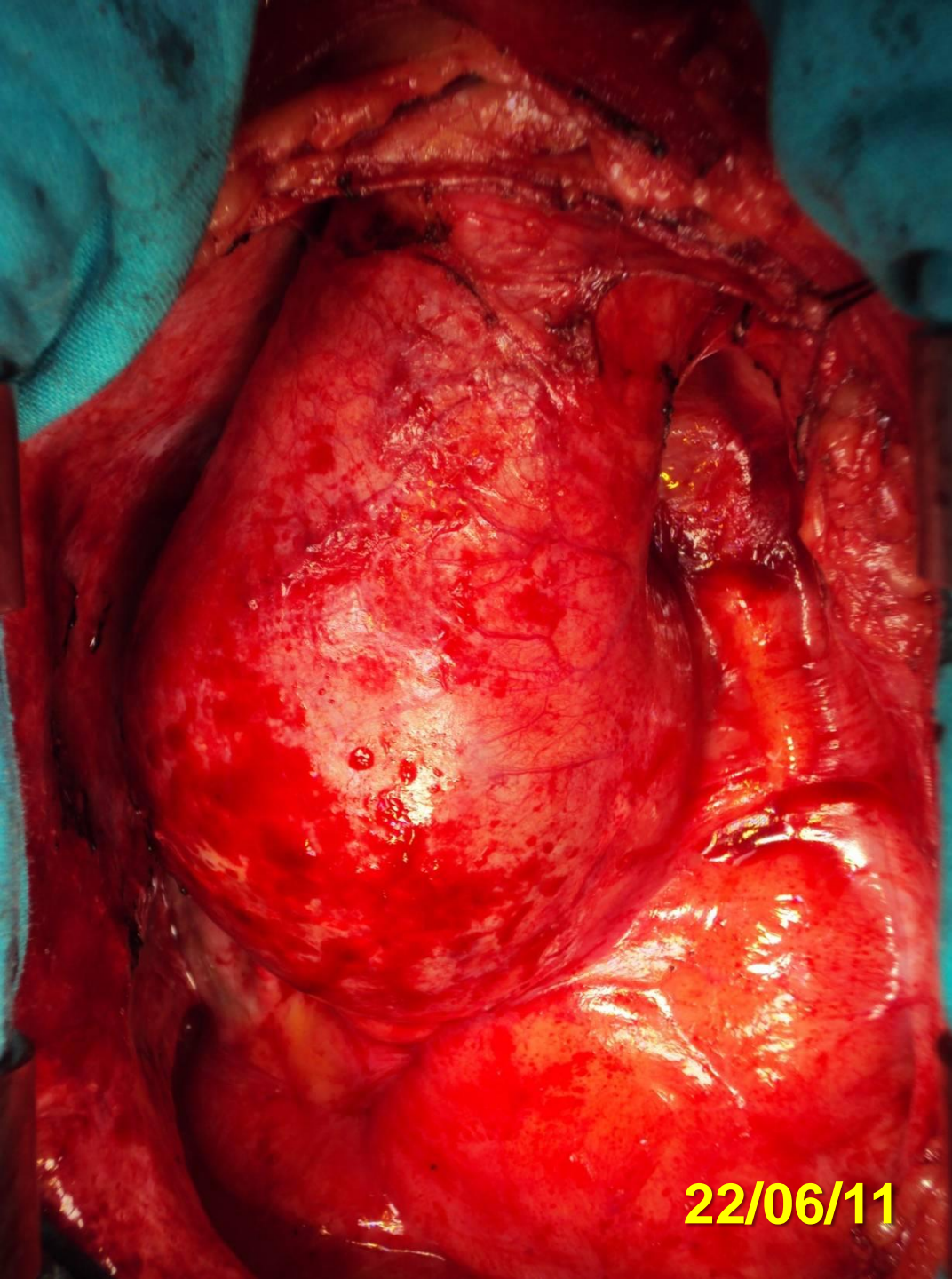
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KV:120  
MA:108

IM:199  
SE:4  
ALG:B

pitch:

206.2  
1mm

P372



22/06/11



inch 0 inch

cm 0 cm 1

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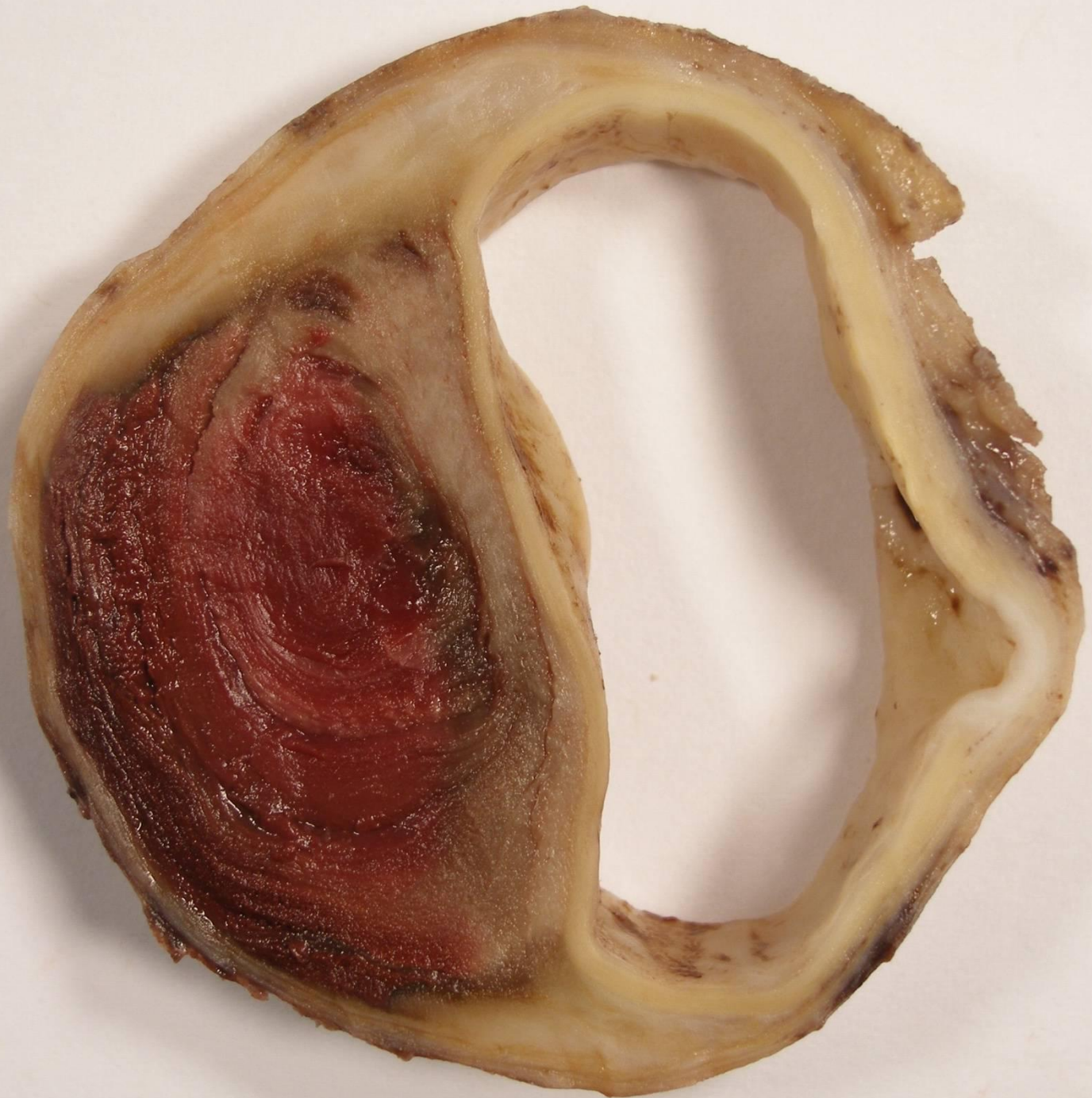
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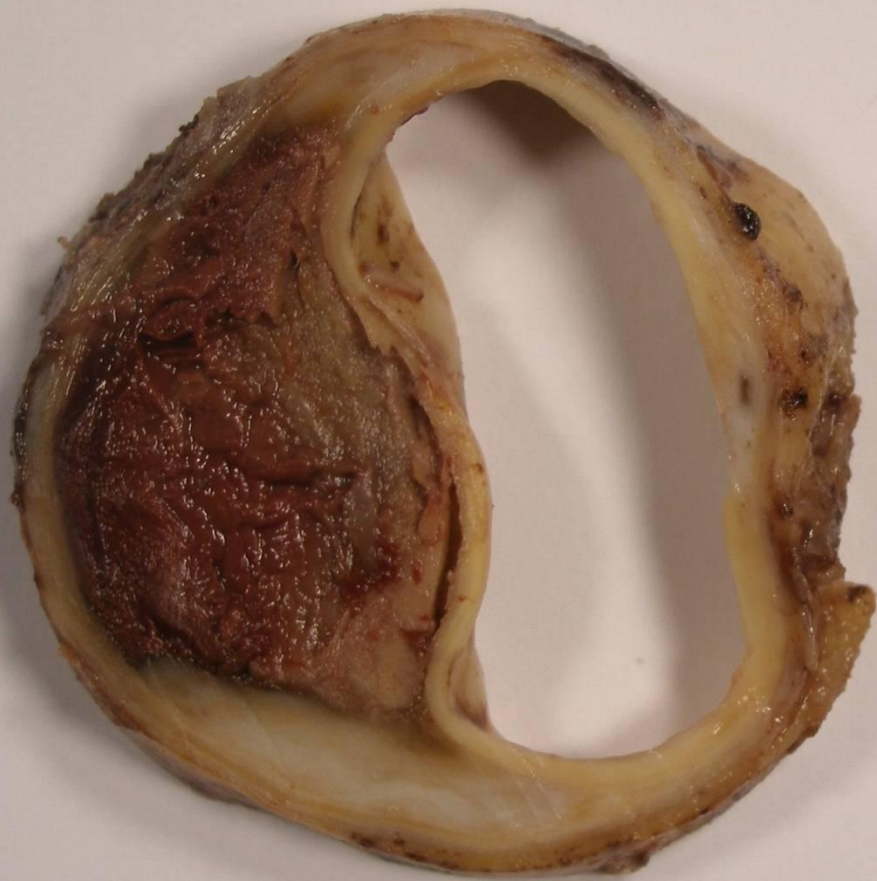
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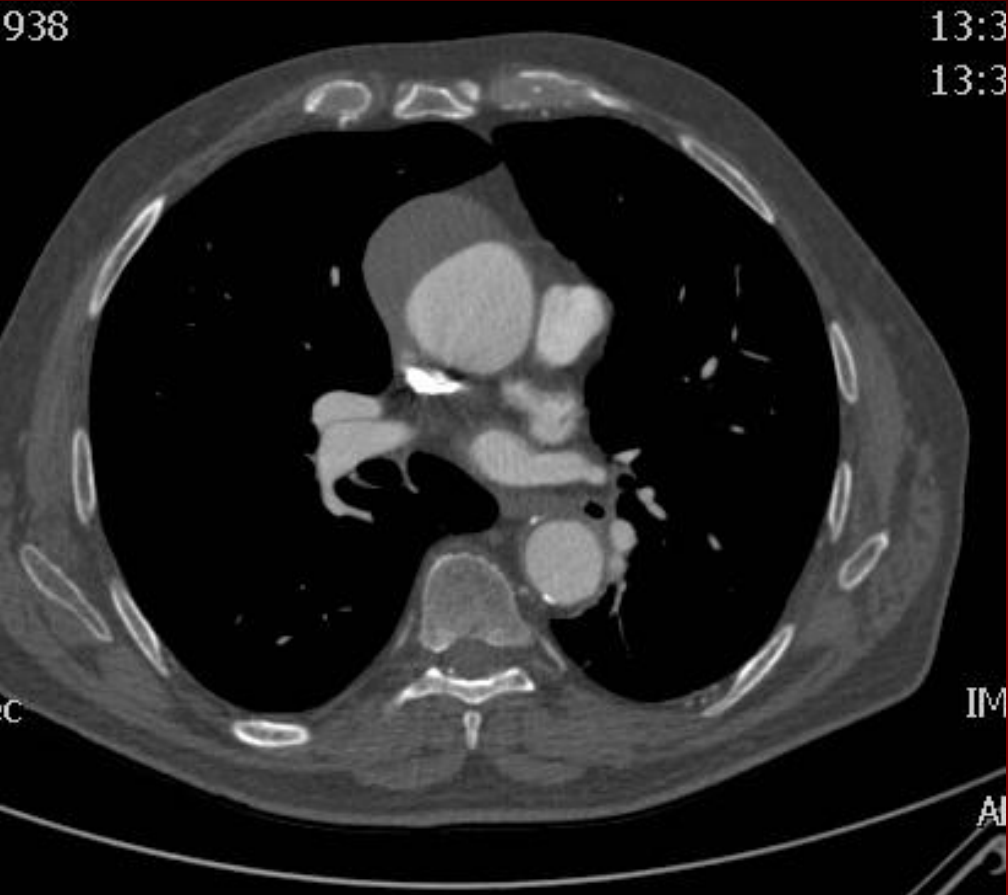




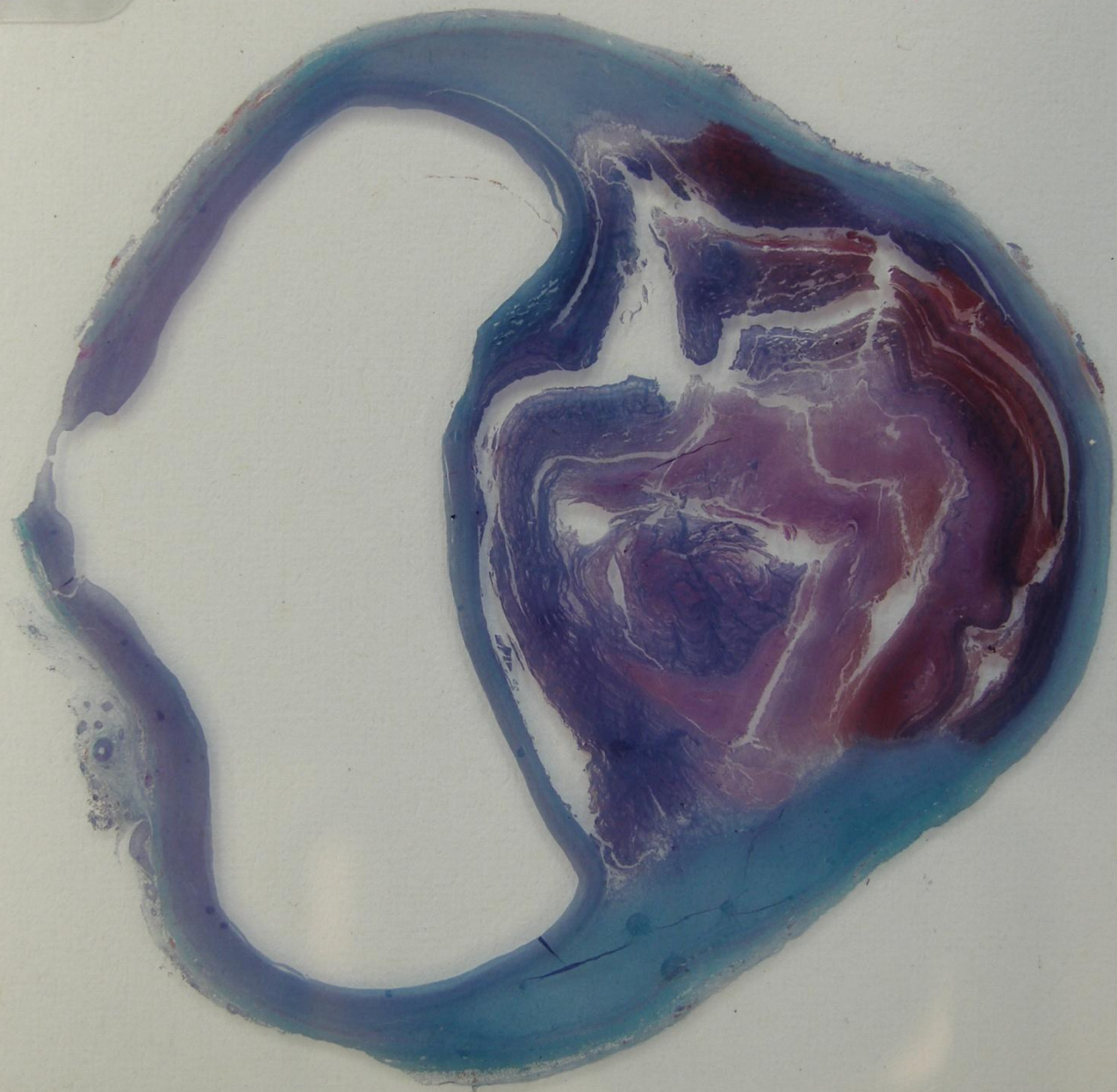




# Patho-tomographic correlation







# Classic aortic dissection

- **Definition:** separation of the aortic media.
- **Entrance tear:**
  - Areas of greatest hydraulic stress.
- **Intimomedial flap.**
- **Double-channel aorta.**
- **Re-entrance tear.**
- **Several communicating points.**

# CLASSIC DISSECTION

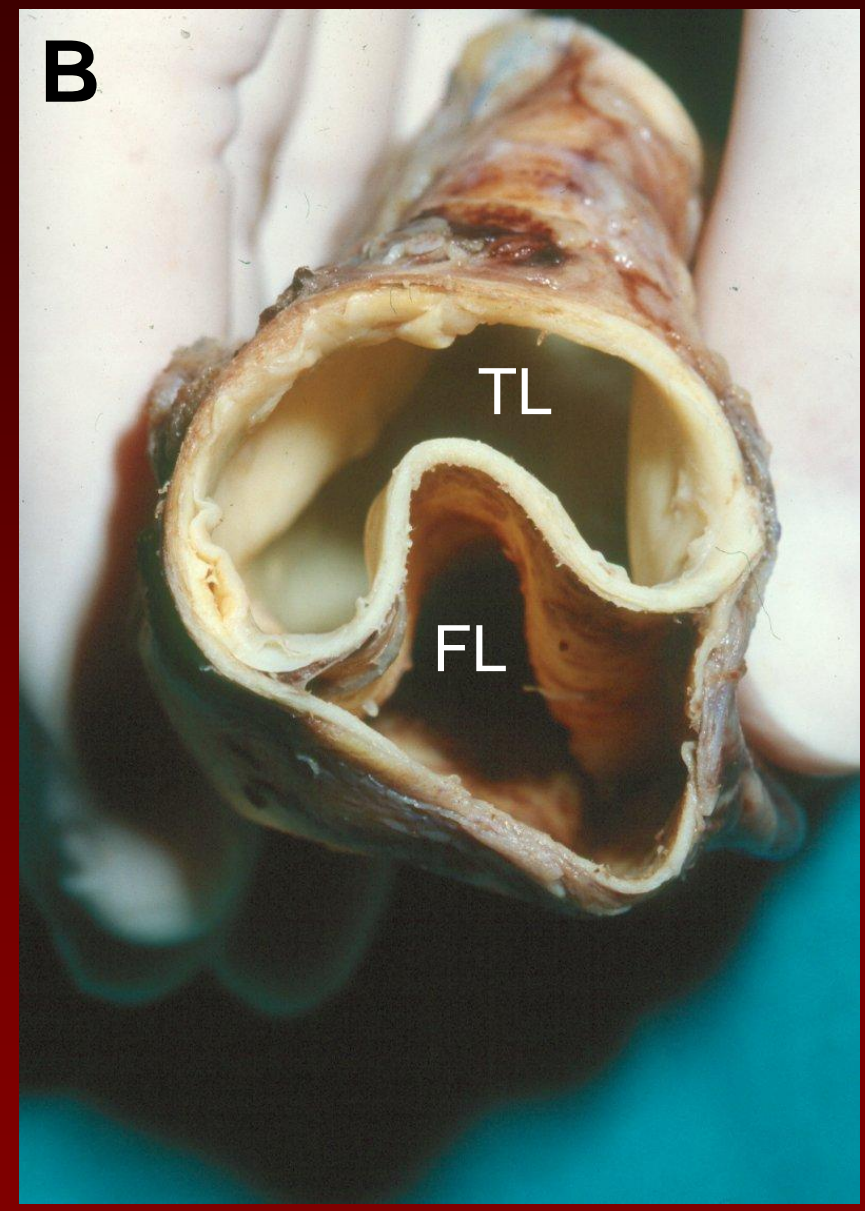
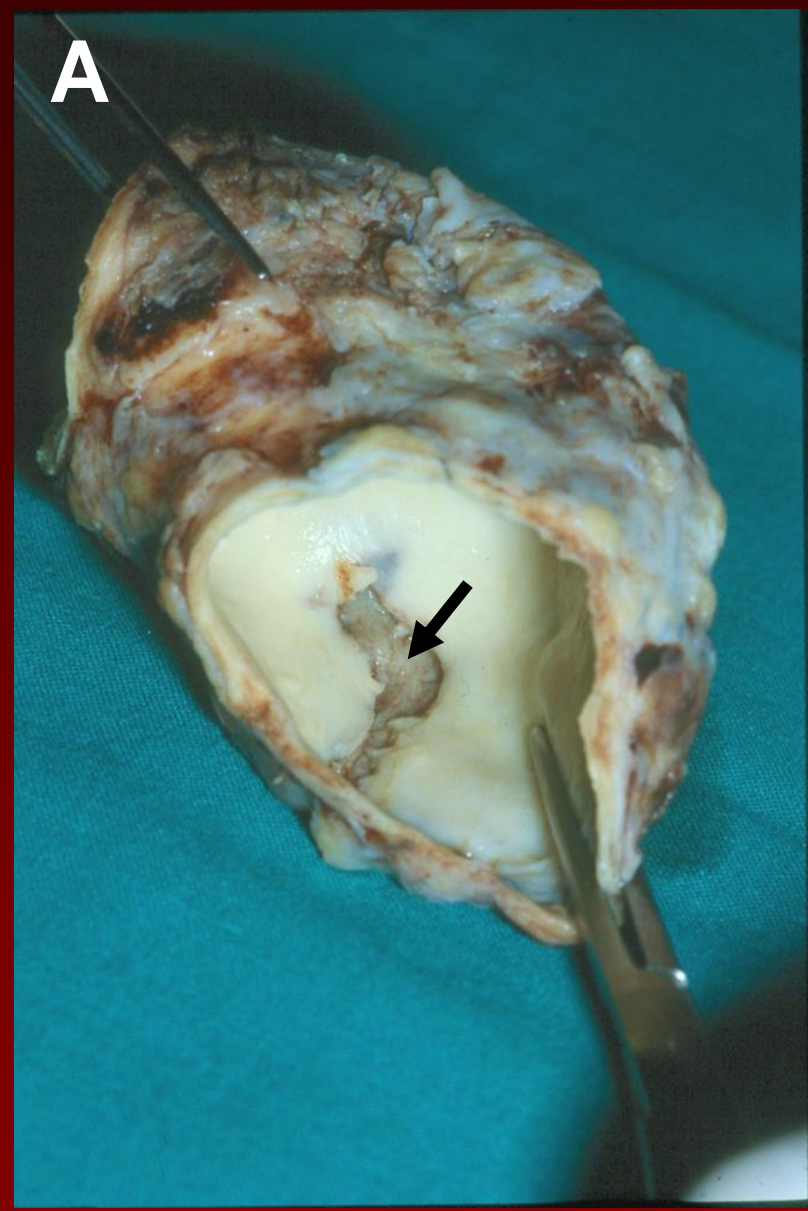
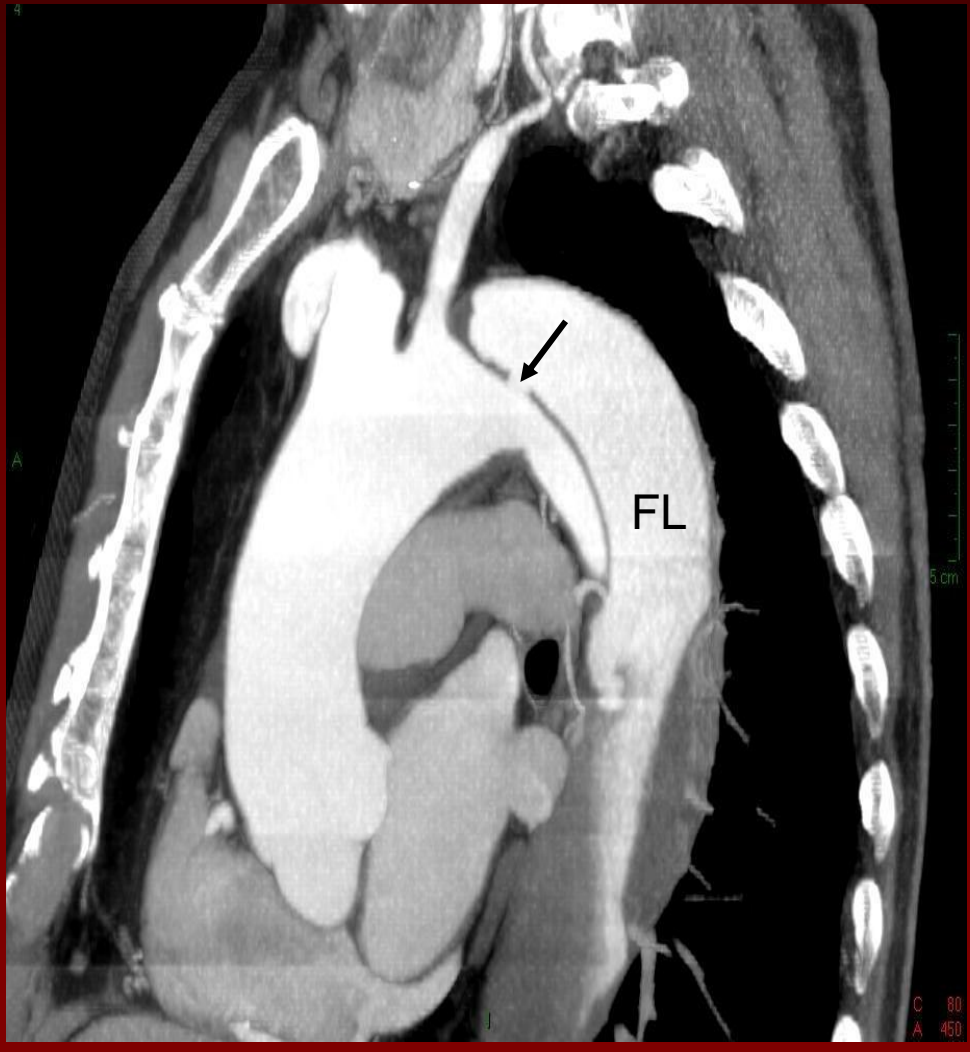
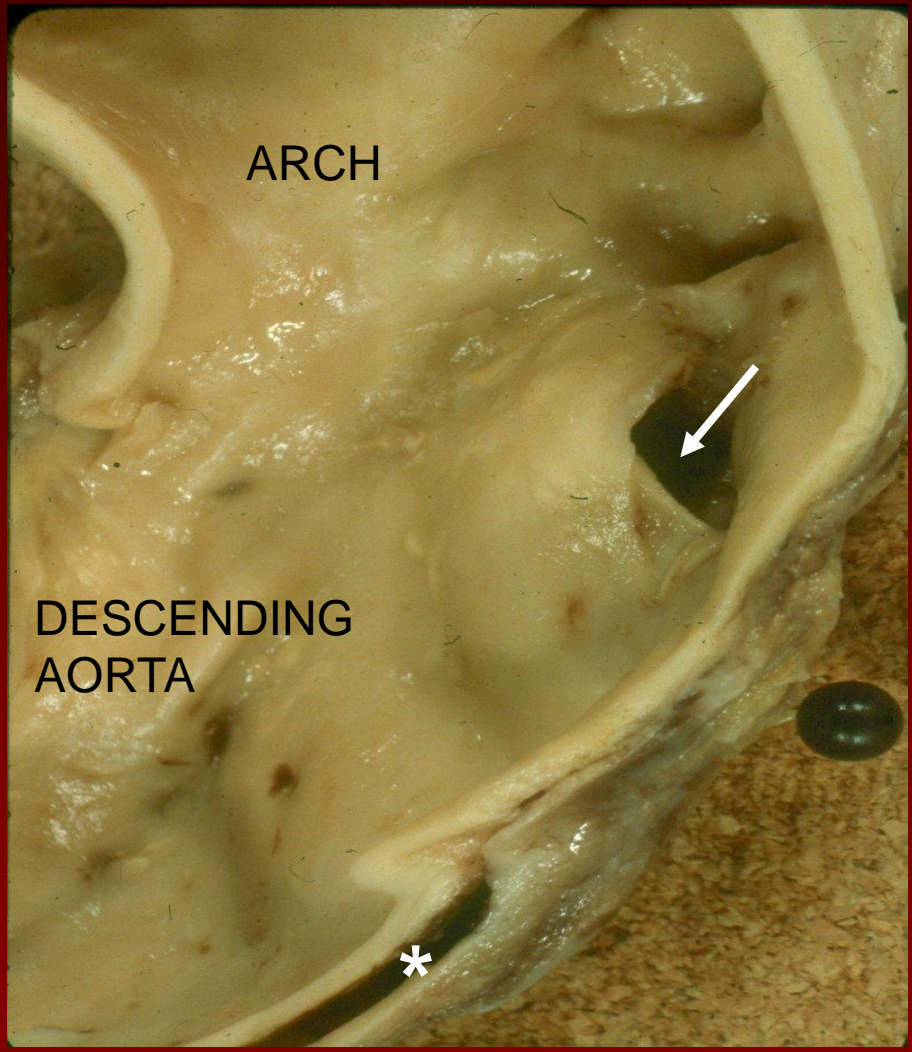
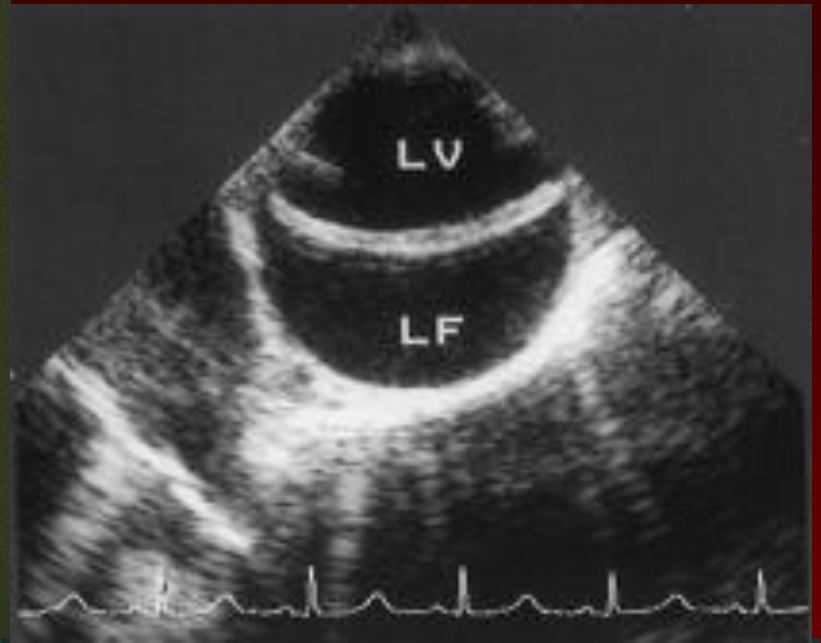
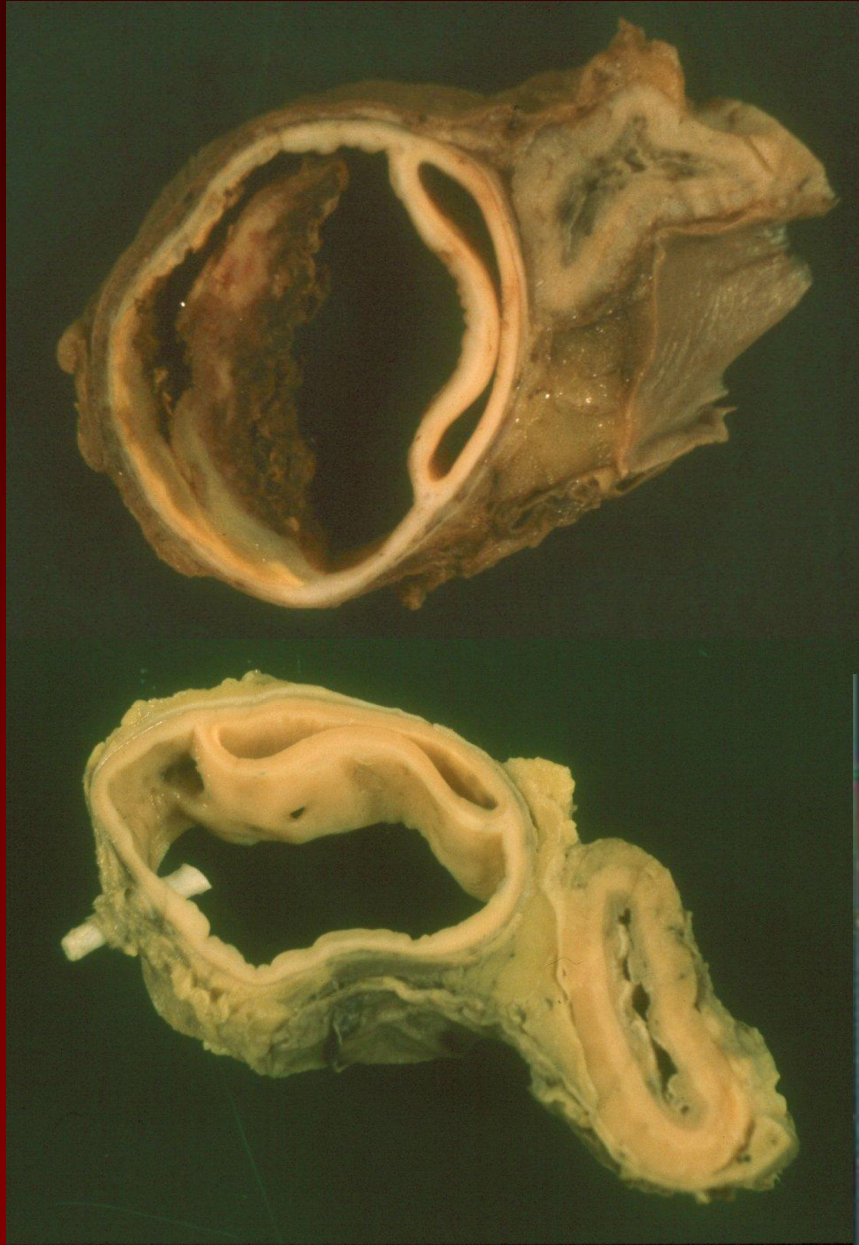


Fig. 3

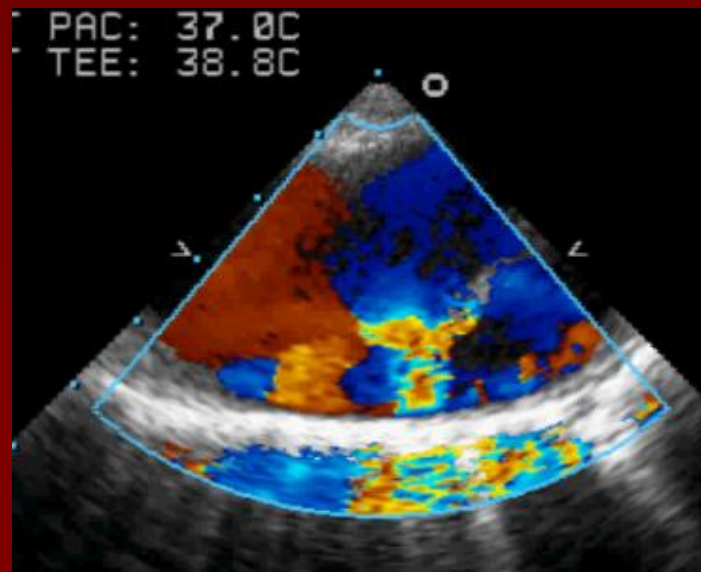
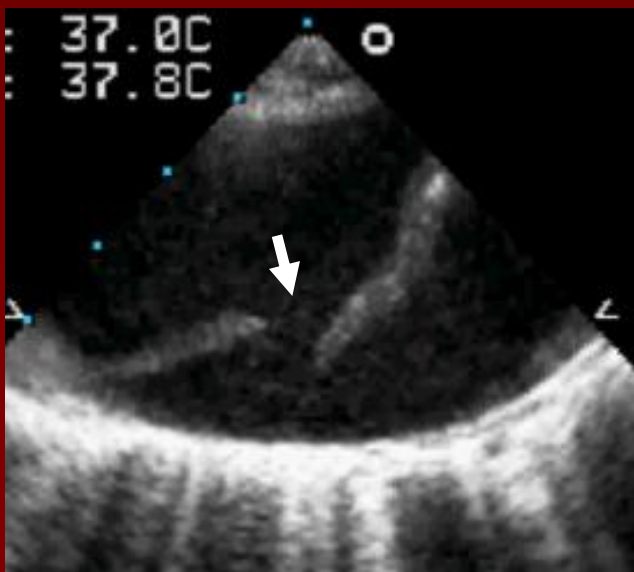
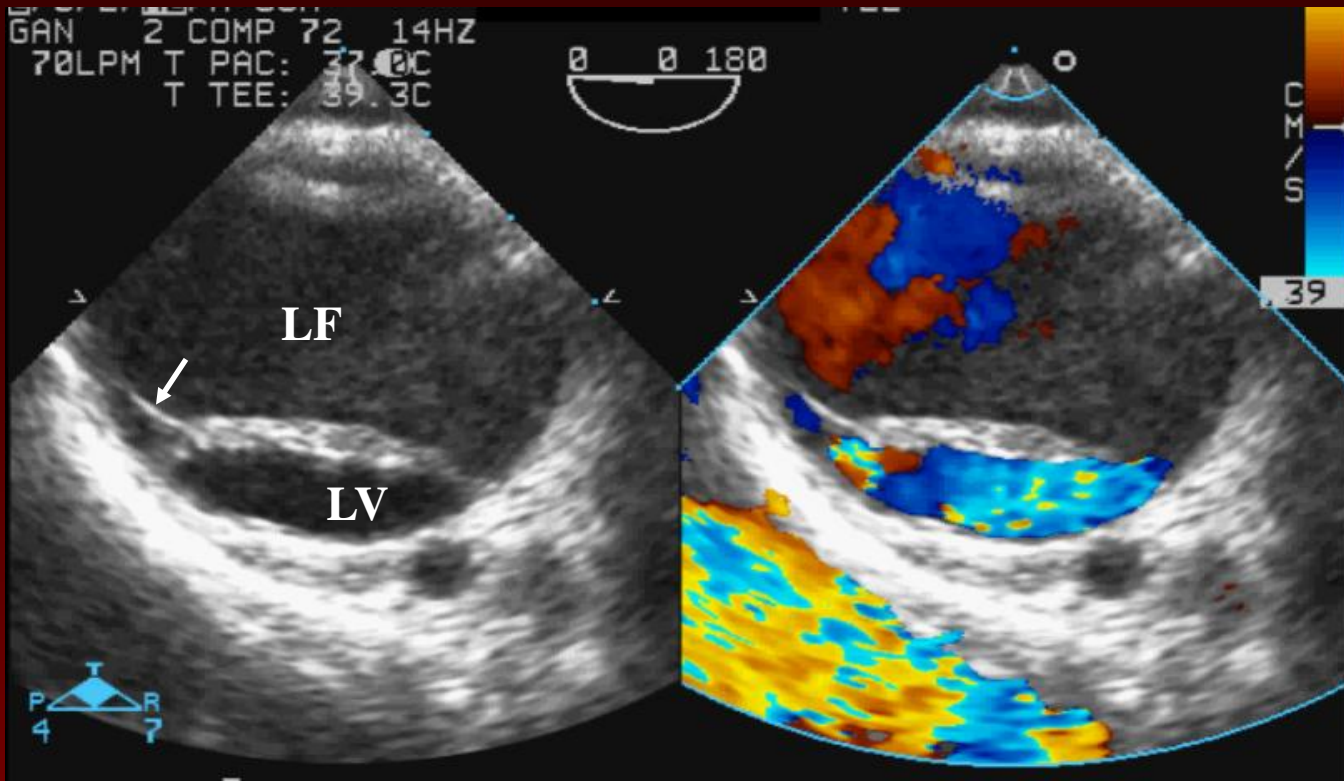
# CLASSIC DISSECTION TYPE B



# CLASSIC DISSECTION

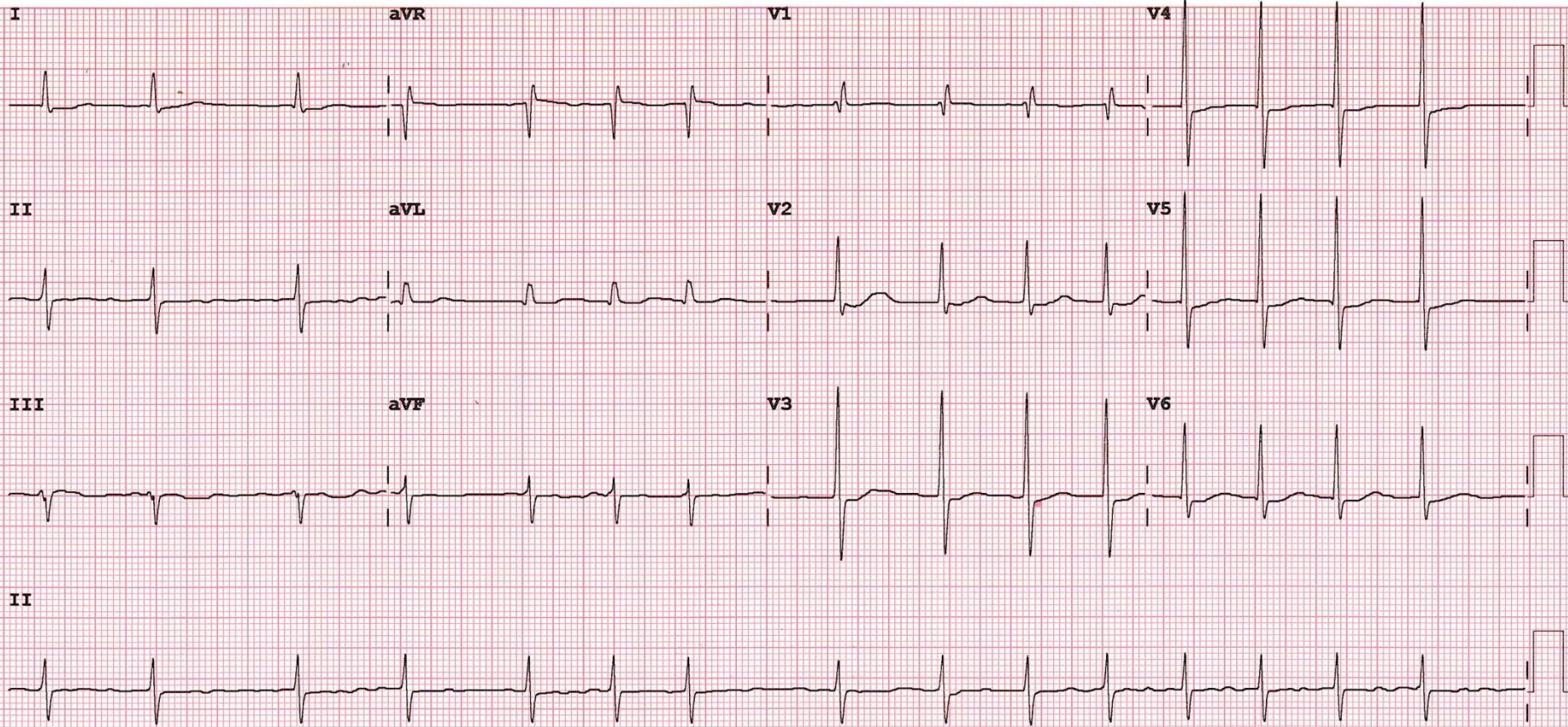


# CLASSIC DISSECTION



# Clinical scenario-2

- 78 year-old male, **HTA**, chronic AF, COPD.
- Acute & intense **chest pain** (april-24-2011):
  - **ER**: AF & normal cardiac enzymes.
  - **H.:** **Angio**: no CAD, **CT**: AA: 45 mm., AAA: 38 mm.
- Two weeks later: similar, but mild **chest pain**.
  - **ER**: AF & ST abnormalities.
  - Normal physical exam. TA: 125/80 mmHg.
  - Lab.: **INR: 2,3**, **Hb: 10,1**, **Htc:31,9%**, plat: 457000, WBC:14400 (N:86%), **DD:5010**, Tr I: 0,03, Gl: 118, Cr: 0,65.



Equip:

Veloc: 25 mm/s

Miemb: 10 mm/mV

Prec.: 10,0 mm/mV

F 50~ 0,50-100 Hz W

PH100B bCL P?





A141

H. C. U. San Carlos

TCU04

AN:12762698

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13:27:43

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500msec

KV:120

MA:149

IM:113

SE:3

ALG:STANDARD

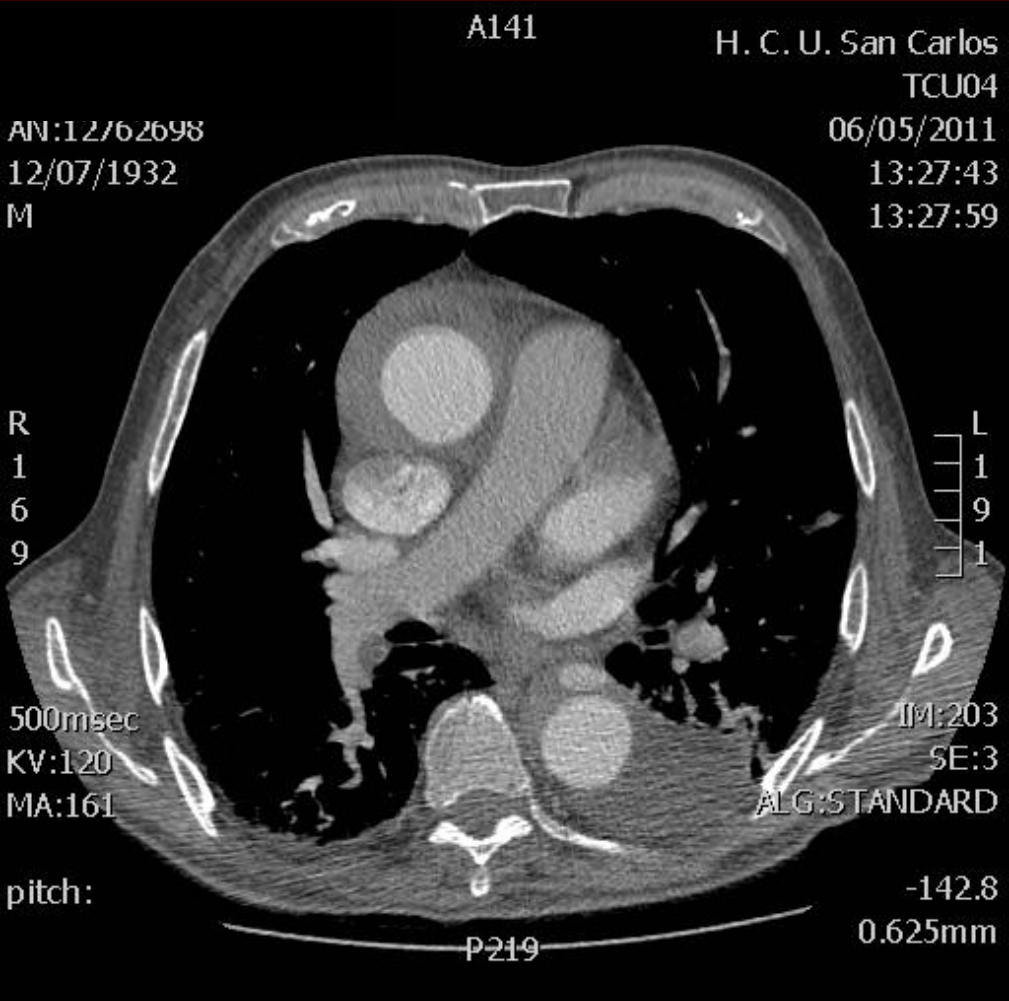
pitch:

-86.5

0.625mm

P219





**Actual CT: May 6, 2011**

**Elsewhere CT,  
April 24, 2011**

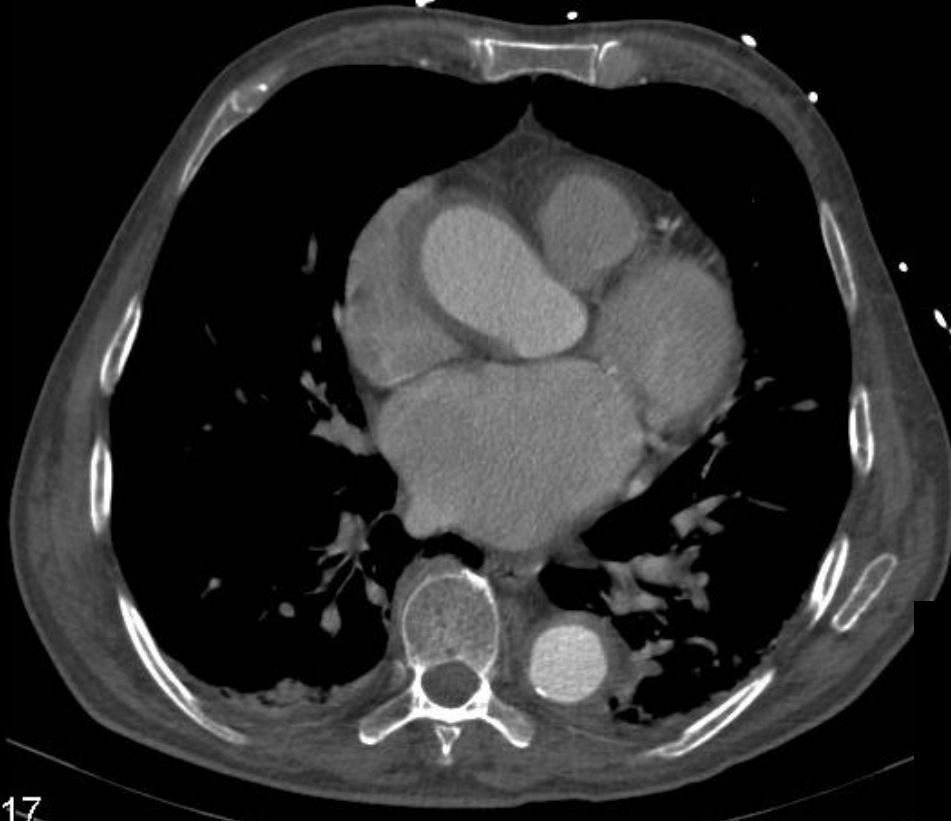


**78 yr-old male; HTA**

**Aortic pain**

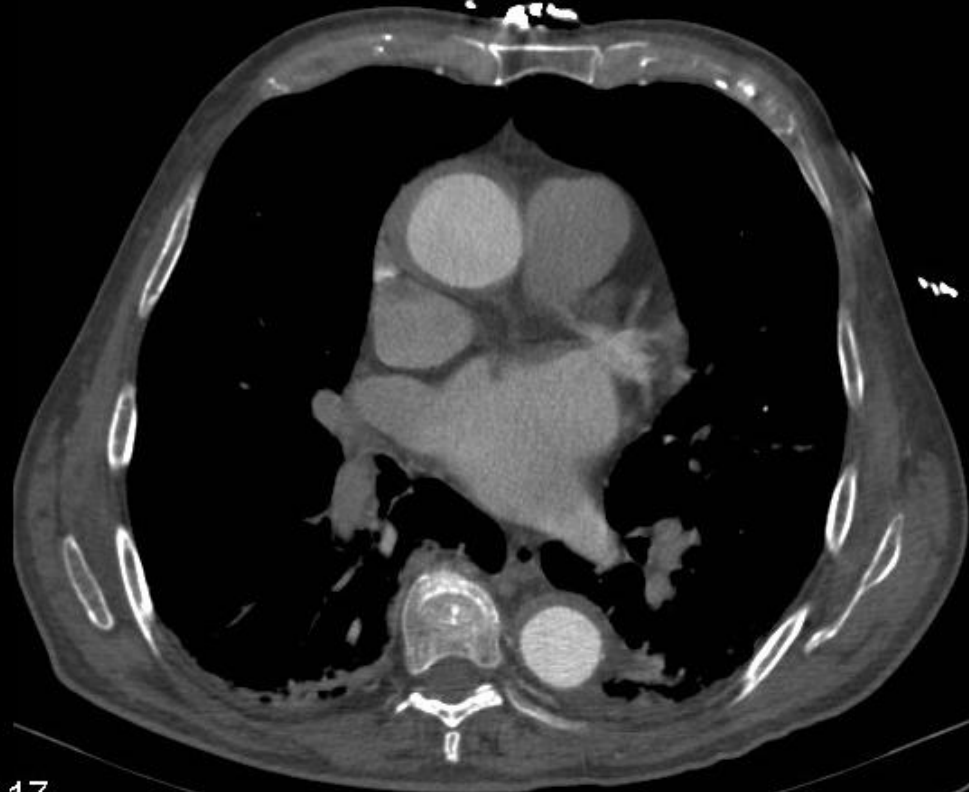
**No EKG ischemic changes**

**Normal myocardial enzymes**

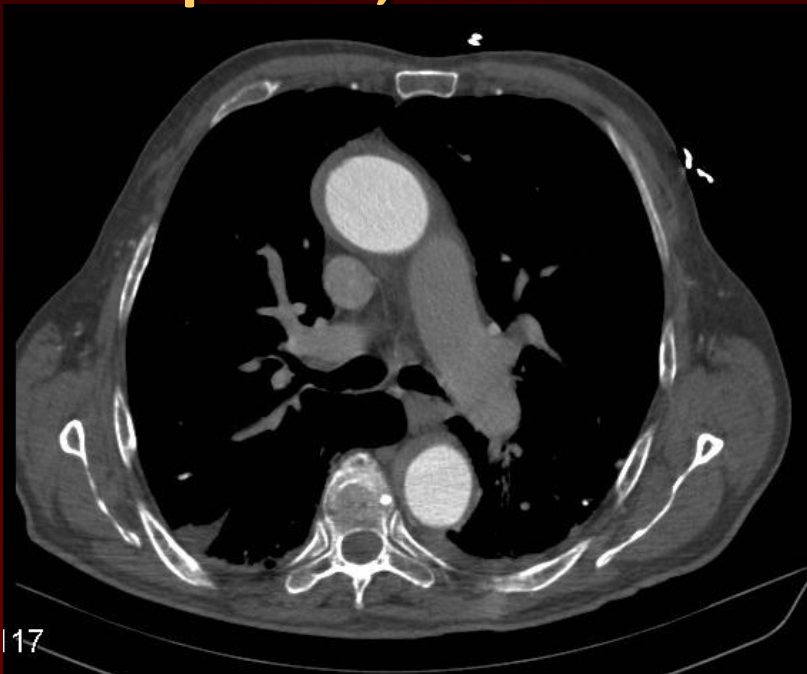


**Elsewere CT,  
April 24, 2011**

**78 yr-old male; HTA  
Aortic pain  
No EKG ischemic changes  
Normal myocardial  
enzymes**



April 24, 2011



May 6, 2011



# Intramural aortic hematoma



# Intramural aortic hematoma

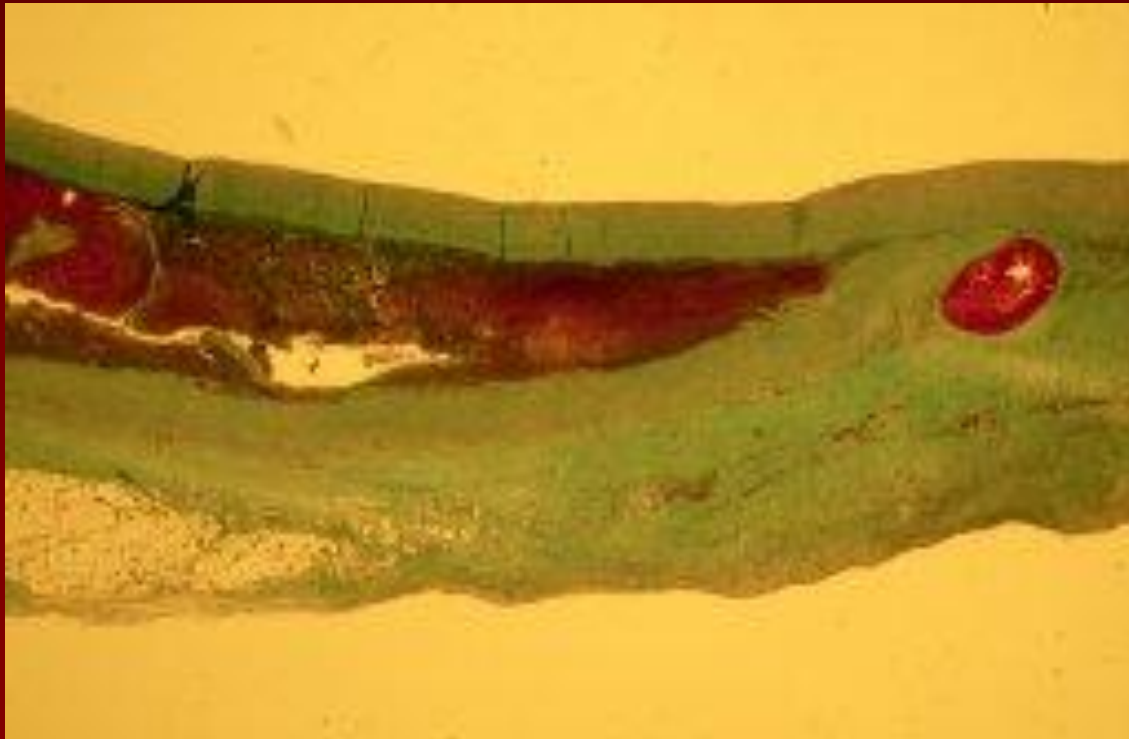


**Non-communicating type of aortic dissection**

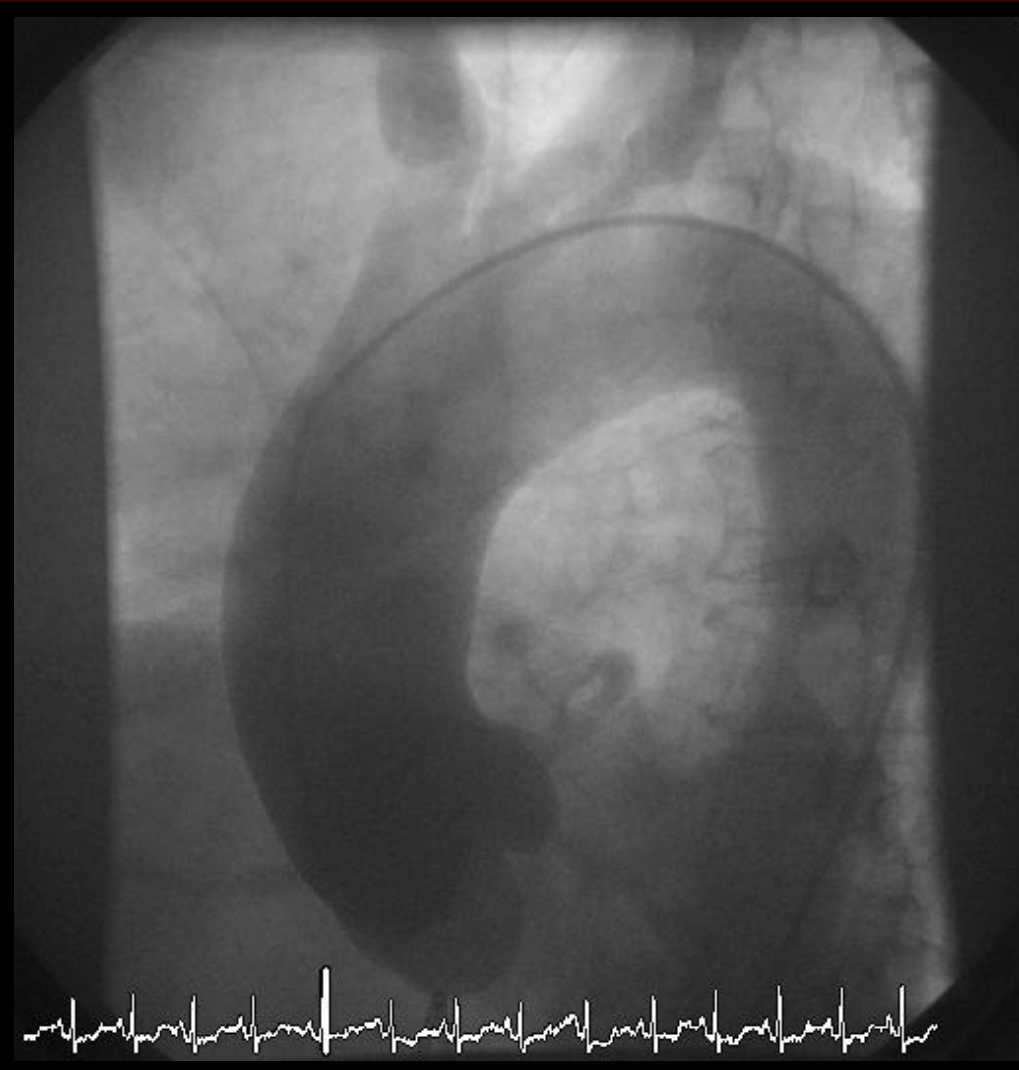
*Mohr-Kahaly S, et al. JACC 1994; Vilacosta I, et al. Am Heart J 1997*



# INTRAMURAL HEMATOMA



# Intramural hematoma

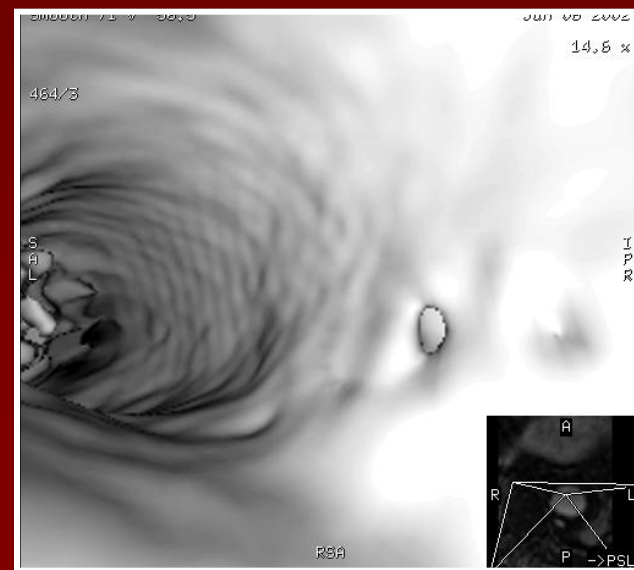
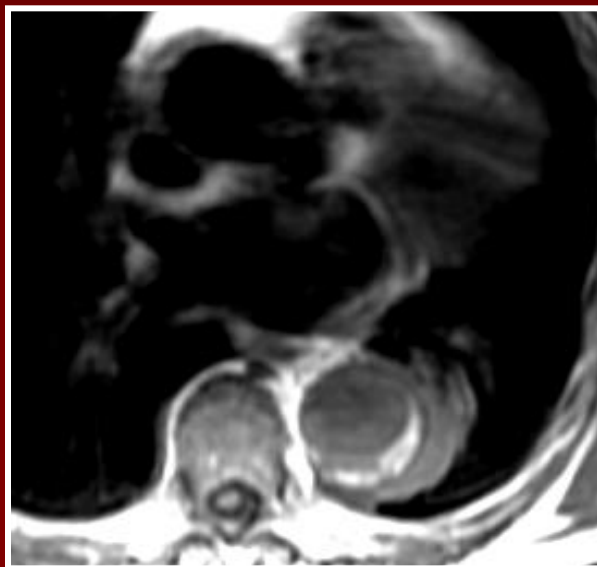


Song JK. Heart 2004.



**HIA**

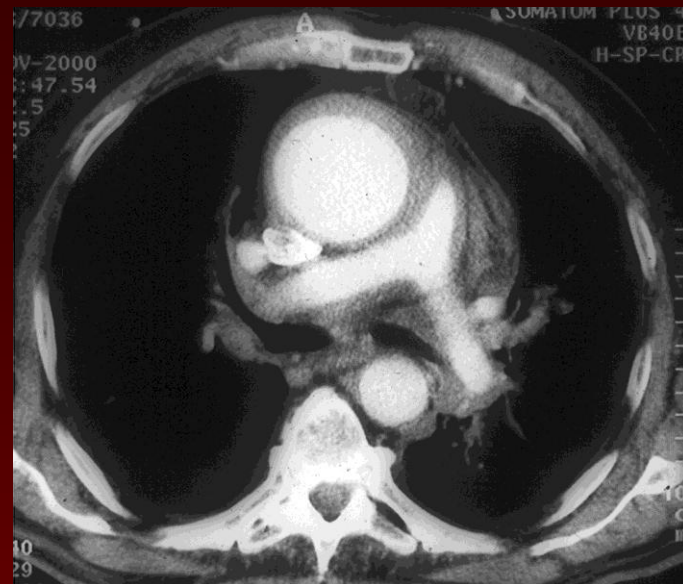
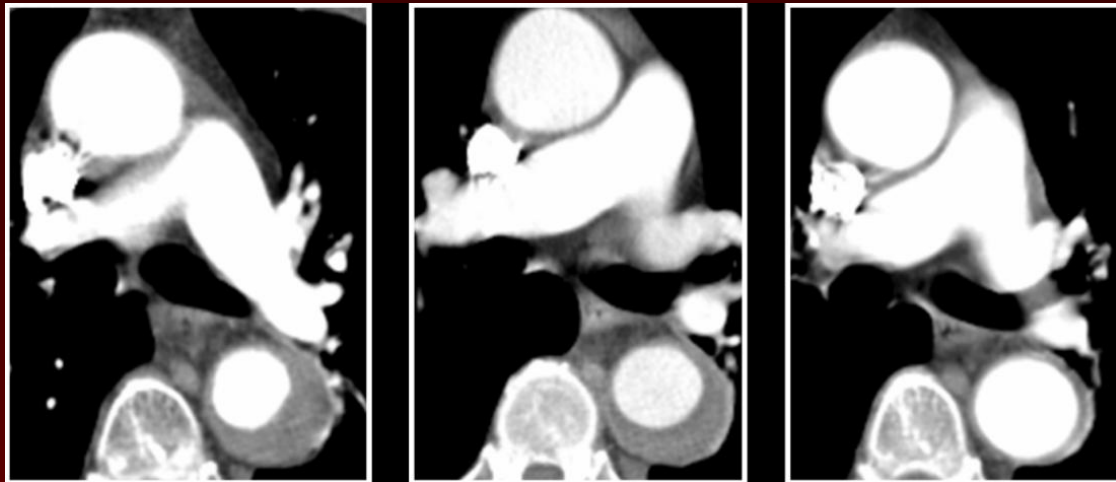
**ANGIO  
RM 3D**



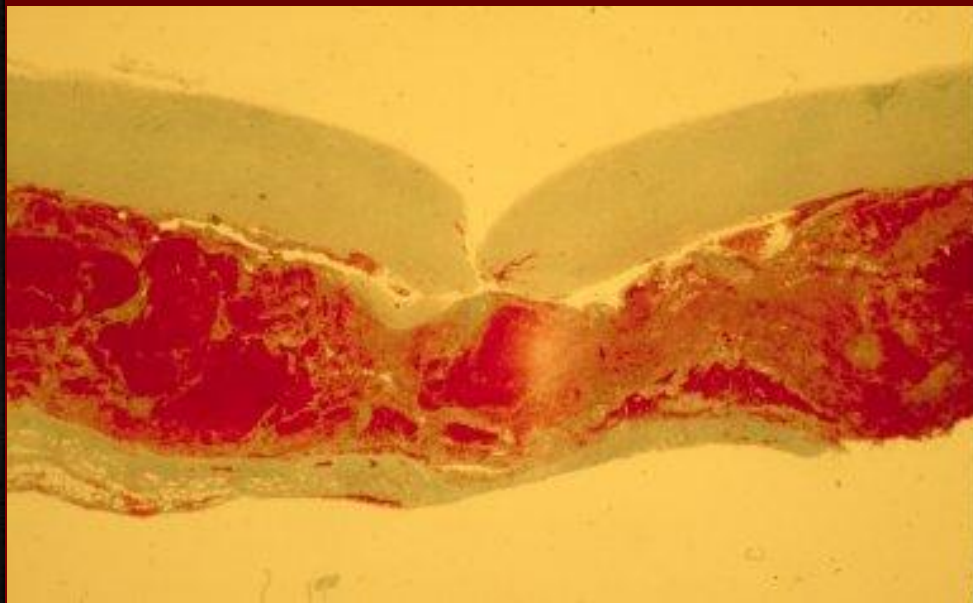
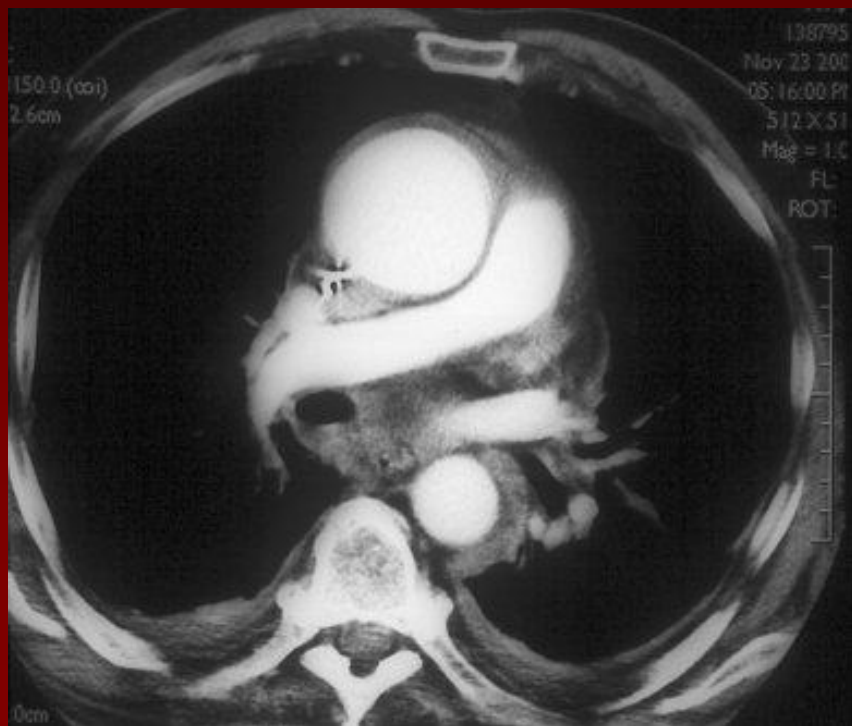
# Focal contrast enhancement on CT

- Retrospective analysis of 107 pts. with distal IAH. **(assessment of the remodeling process & long-term outcomes): 5 types of evolution.**
- **FCE** (small contrast-enhanced islandlike lesion within the hematoma without direct communication with the true lumen): **42 of 107 pts (39,3%).** Two groups. Maximal dimension:  $7,2 \pm 2,8$  mm (3-15 mm).
- **IAH resorption was the most common (n=70) type of evolution in both groups** without any sig. difference.
- Development of localized dissection occurred more frequently in pts. with focal contrast enhancement (**microtear ?**).
- **Lower long-term survival rates were observed in patients with than in those without IAH progression,** but not between patients with and without FCE. **No need for urgent intervention for patients with FCE.**
- Maximal aortic diameter was associated with survival rates. **The optimal cutoff value for prediction mortality within 7 years was 41 mm.**

# Evolution of IAH



## Micro-entrance TEAR

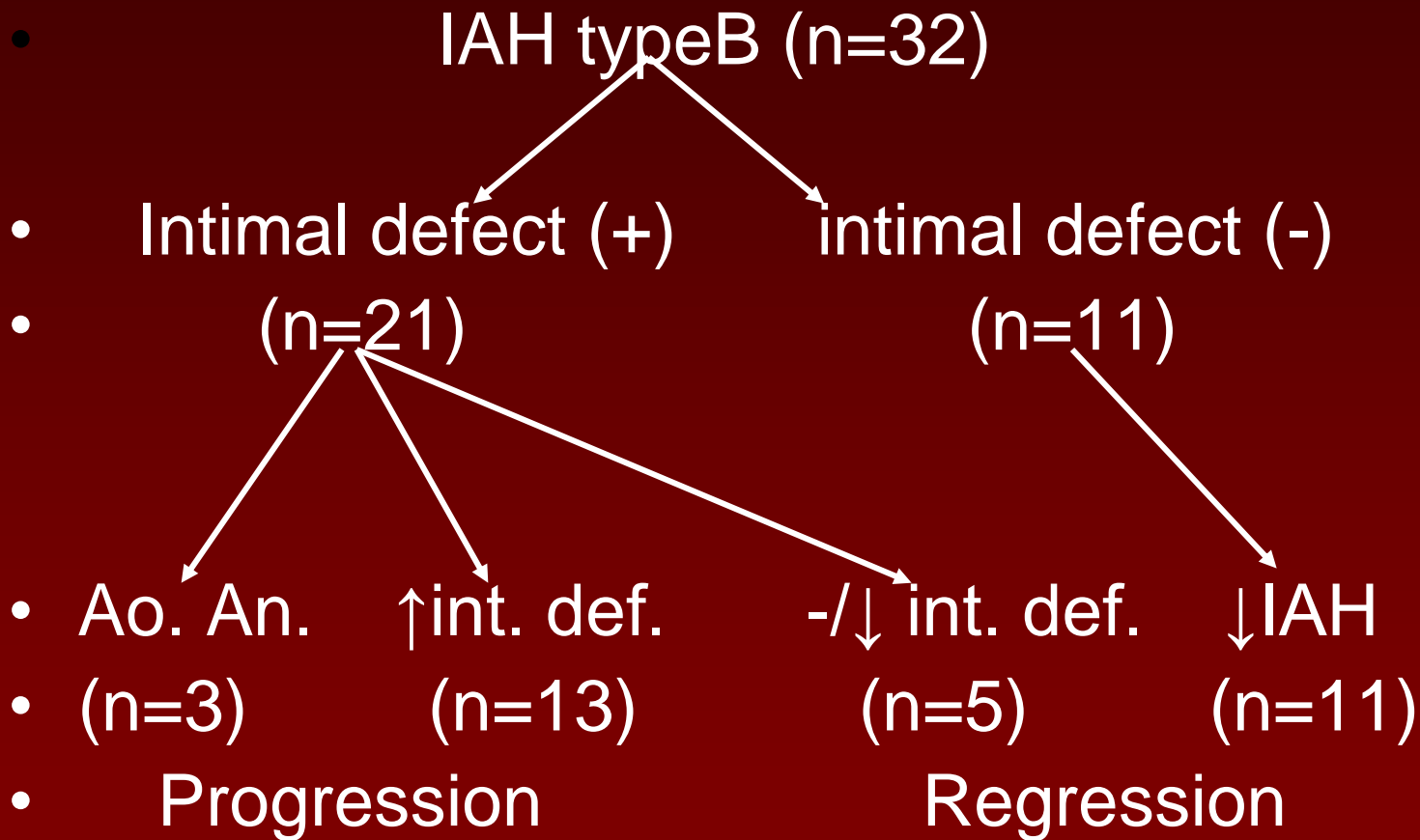


Vilacosta I, et al. Thoracic aortic diseases. Springer 2006.

# Detection of intimal defect by 64-row MDCT in patients with IAH

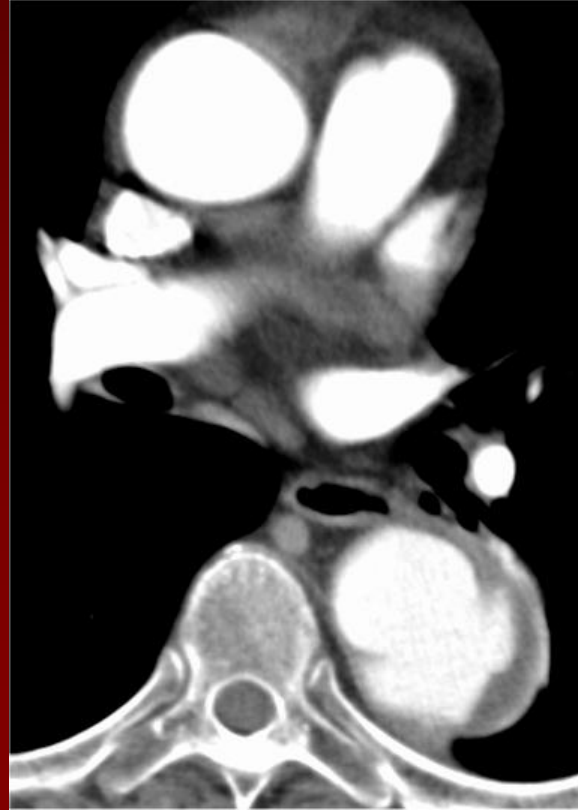
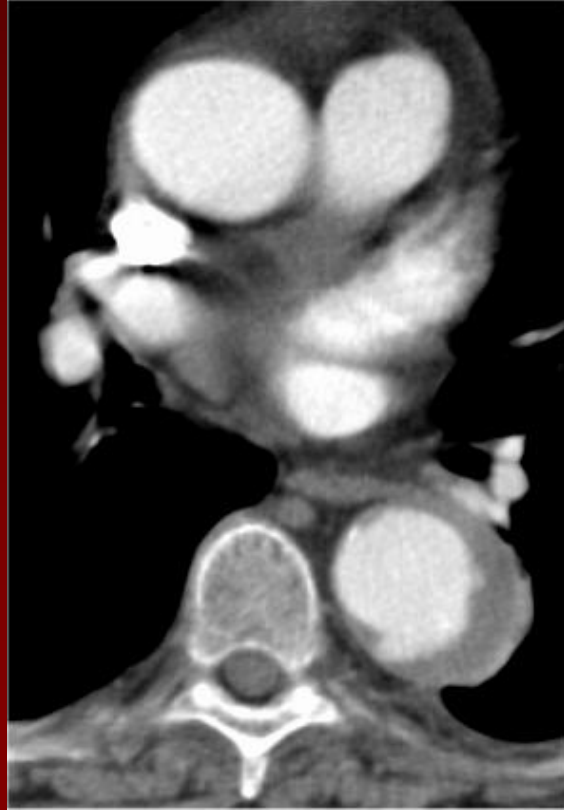
- Retrospective study of 38/60 patients IAH medically treated: IAH-A: 6; IAH-B: 32.
- 64-row MDCT with axial & longitudinal MPR images with 1 mm slice thickness during the acute phase (2 weeks).
- Intimal defect (continuity disruption of intimomedial flap) was detected by MDCT even at an early stage: 48 lesions in 27 p. (71%).
- Intimal defect was not completely identified by conventional 5 mm CT; MDCT: 20 lesions in 9.
- Intimal defect  $\geq 5$  mm in depth tended to progress.

# Detection of intimal defect by 64-row MDCT in patients with IAH





**Type B-IAH. Evolution of an intimal defect to an aortic aneurysm.**



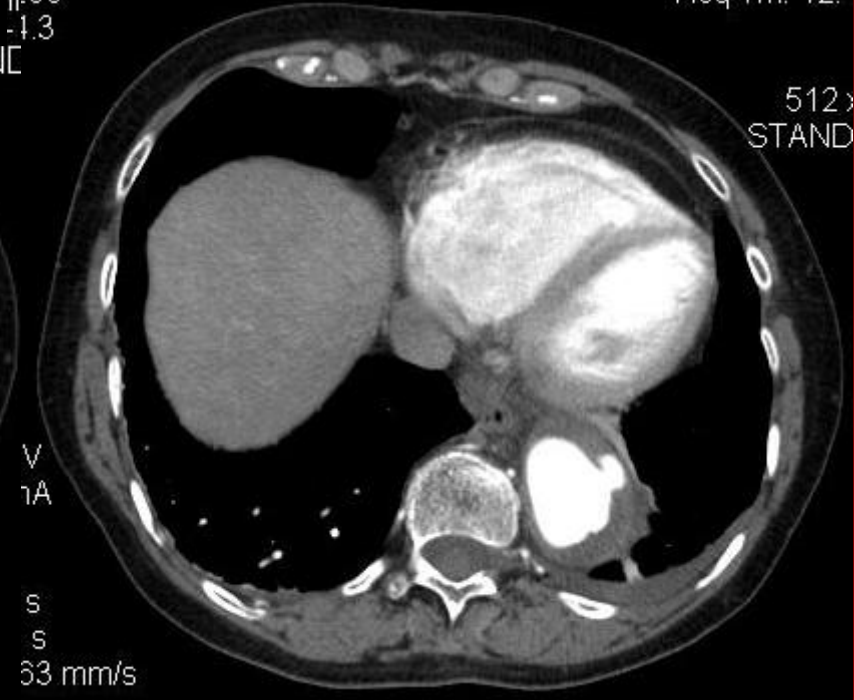
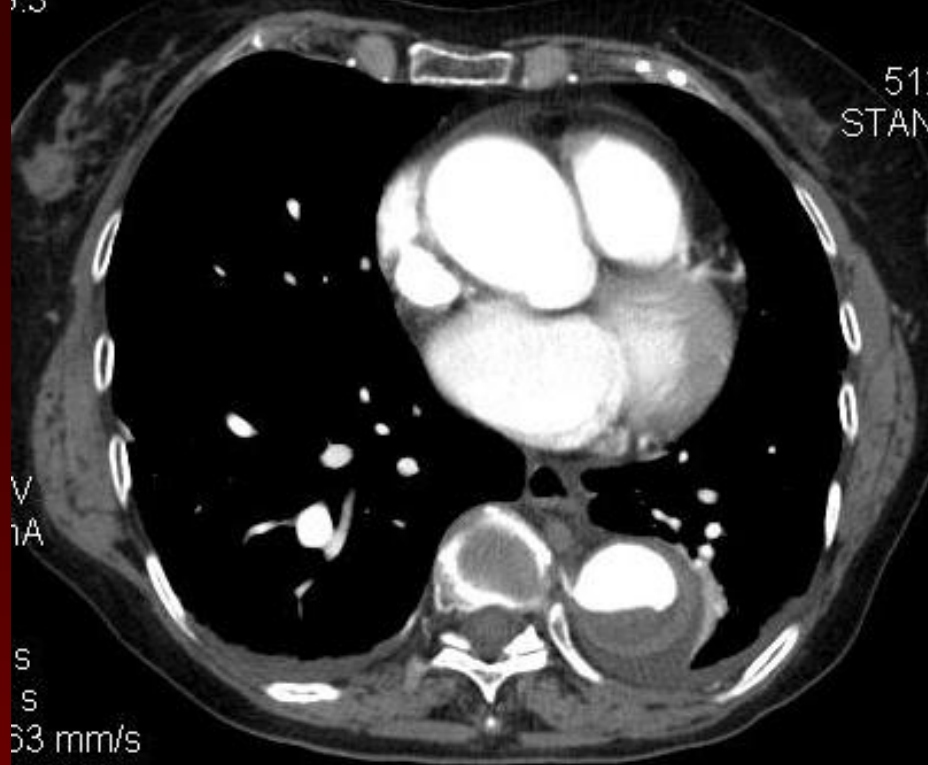


# Indicators of IAH progression

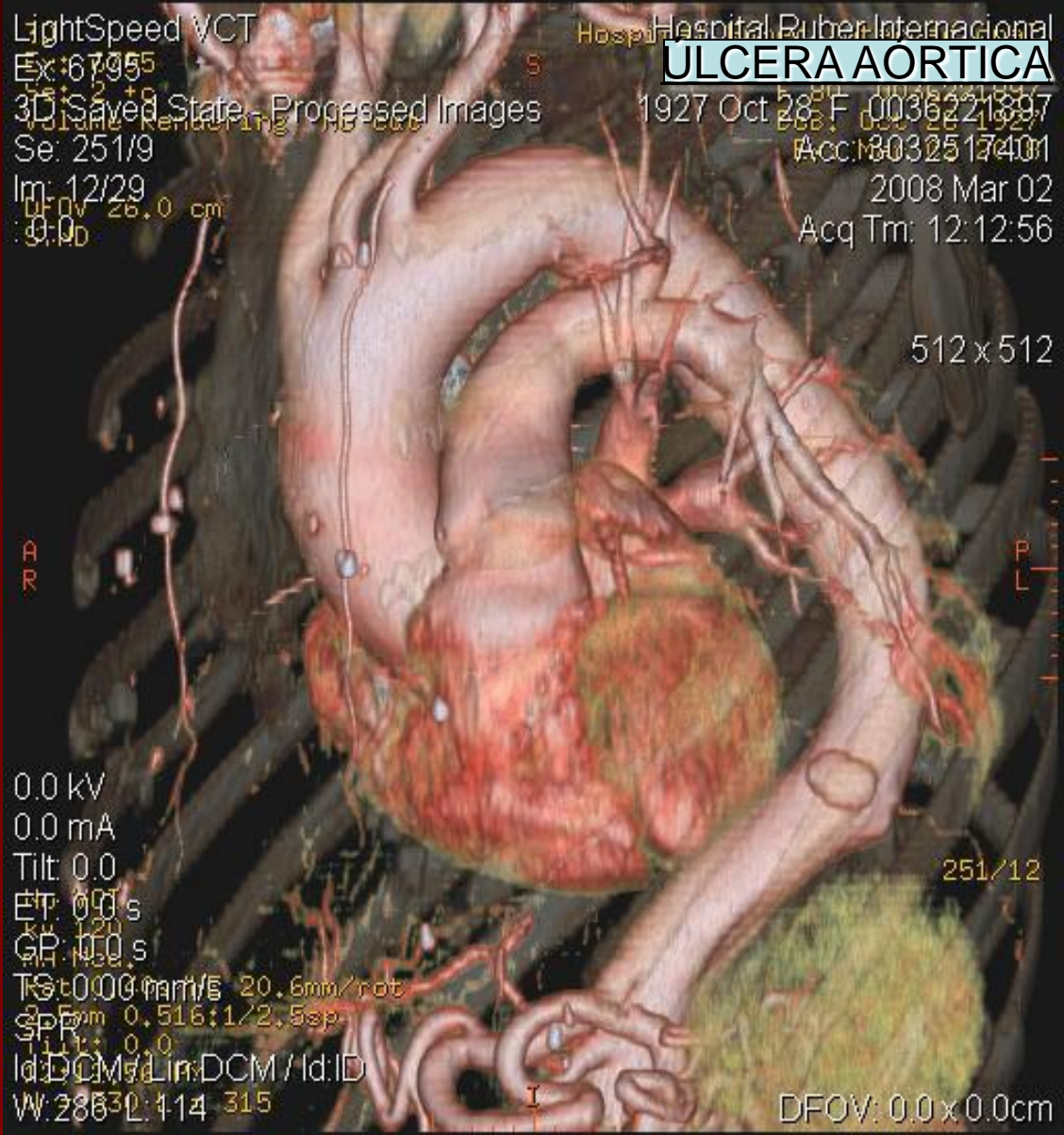
- **Involvement of the ascending aorta.**
- **Maximum aortic diameter (AA:  $\geq 50$  mm; DTA:  $\geq 40$  mm) on initial diagnostic imaging.**
- **Maximal hematoma thickness ( $\geq 16$  mm).**
- **Severe pericardial effusion.**
- **Huge or progressively increasing pl. effusion.**
- **Progressive aortic dilatation at follow-up.**
- **Persistent pain or haemodynamic instability, or both.**
- **Increment of the aortic wall thickness.**
- **Large intimal defect.**

# Clinical scenario-3

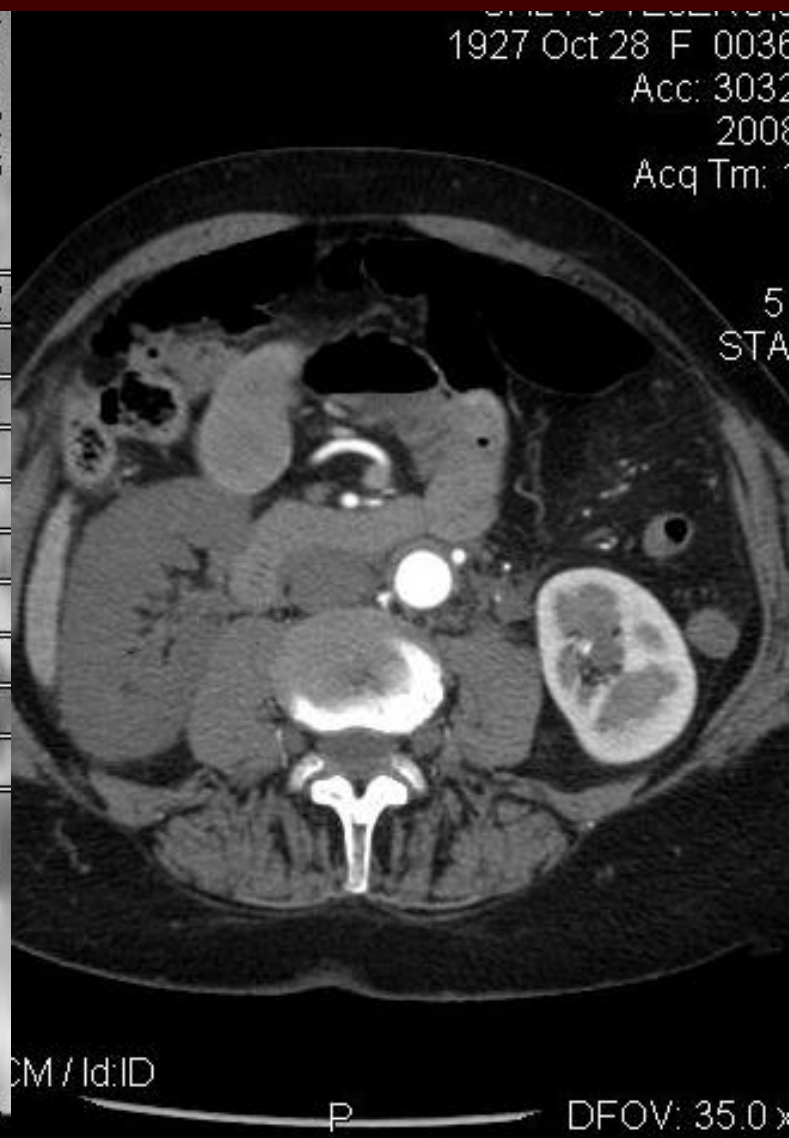
- 80 year-old woman with hypertension.
- **Acute chest pain** irradiating to the back and abdomen.
- **Physical exam:** BP: 170/80 mmHg, systolic murmur; pulses present; mild abdominal pain.
- **EKG:** sinus rhythm; no ST-T changes.
- **Chest-X ray:** prominent aortic Knob; no signs of heart failure.
- **Analysis:** AST:146; ALT:127; LDH:2870; CK: 40; Tr I:0,2; **D-dimers: 2251.**



# Clinical scenario-3

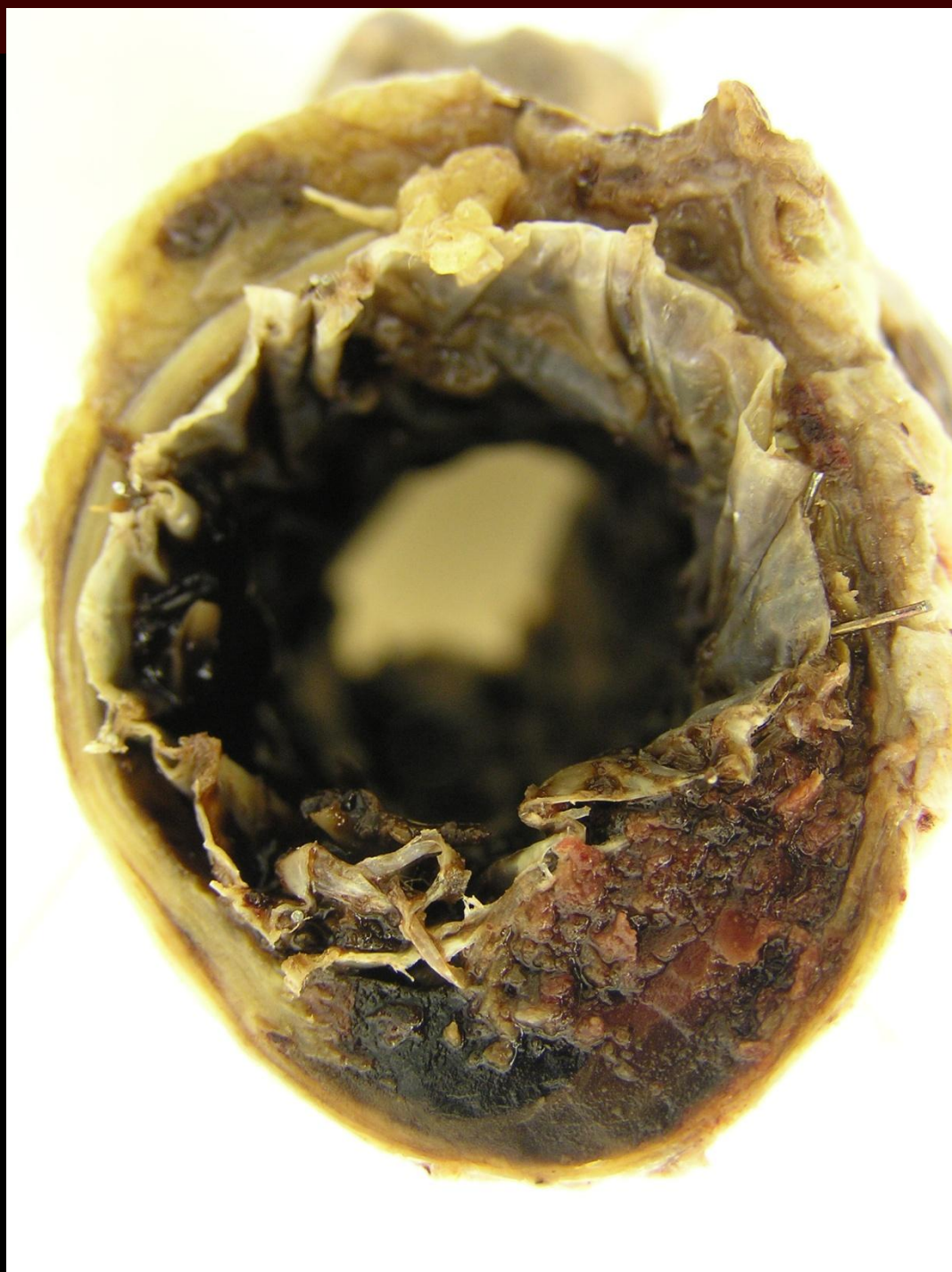


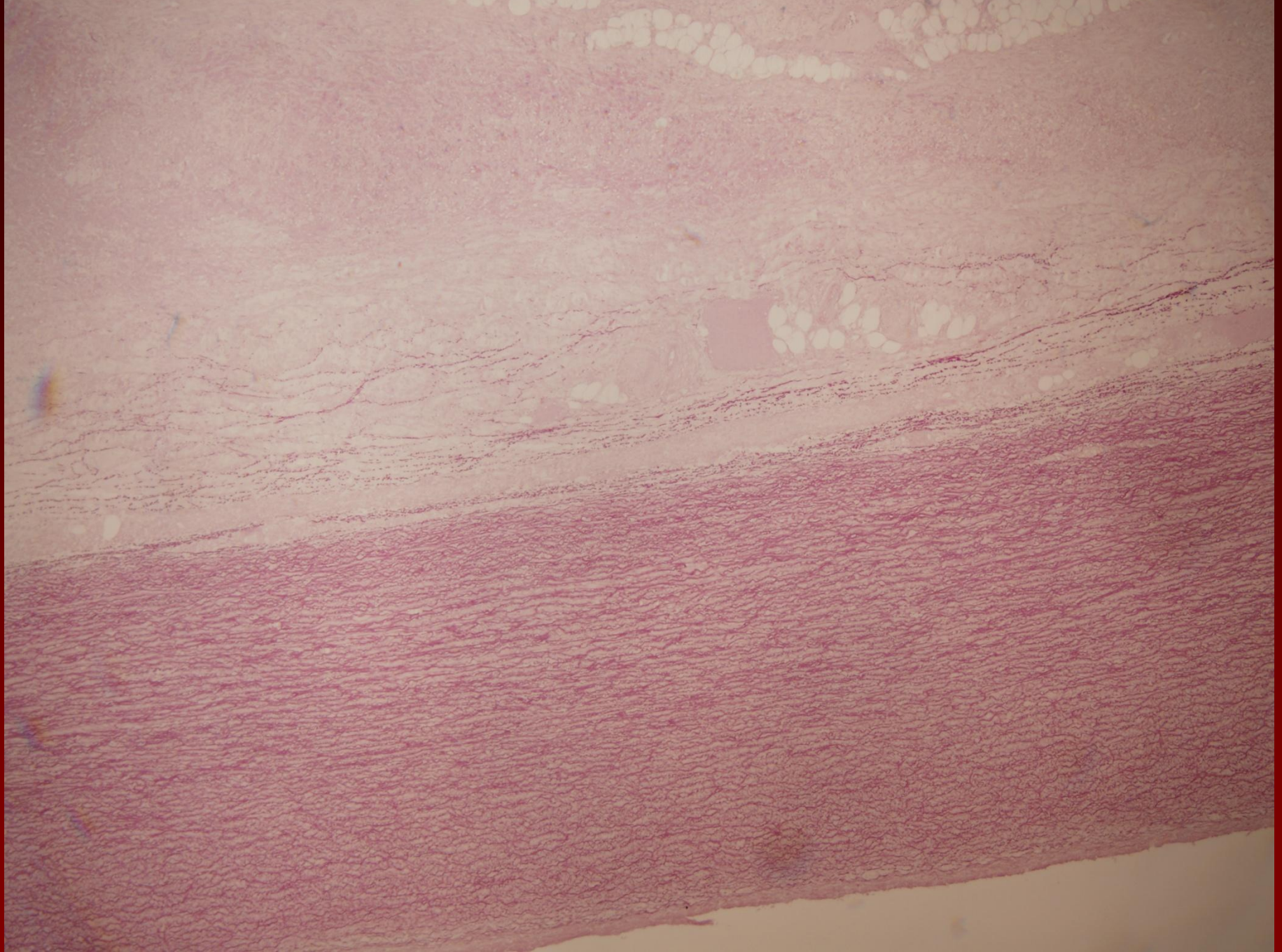
# Clinical scenario-3



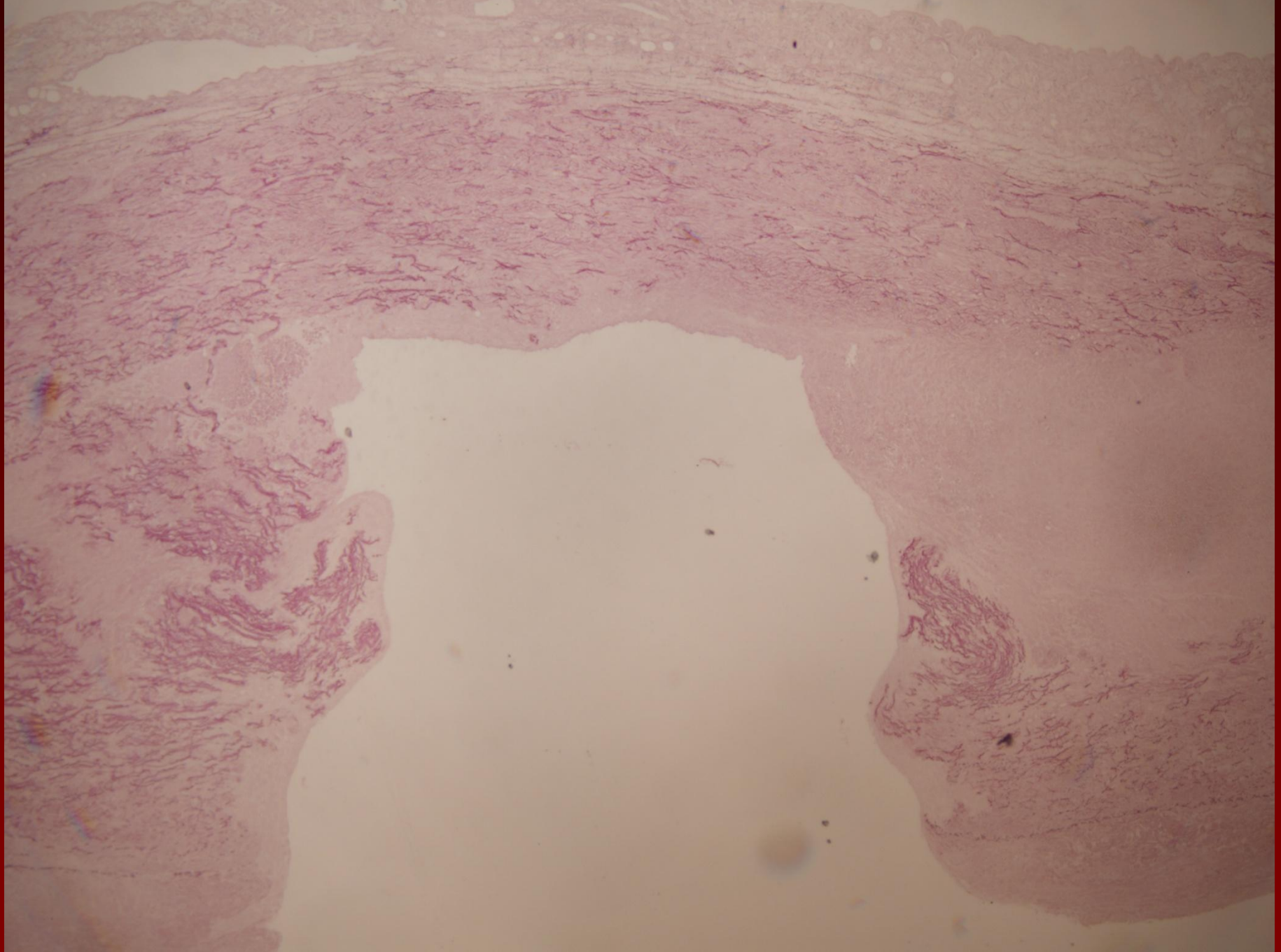
OV: 26.4cm  
FT: +

VOI  
120  
Mod.  
t: 0.50s / HF+ 39.4mm/rot



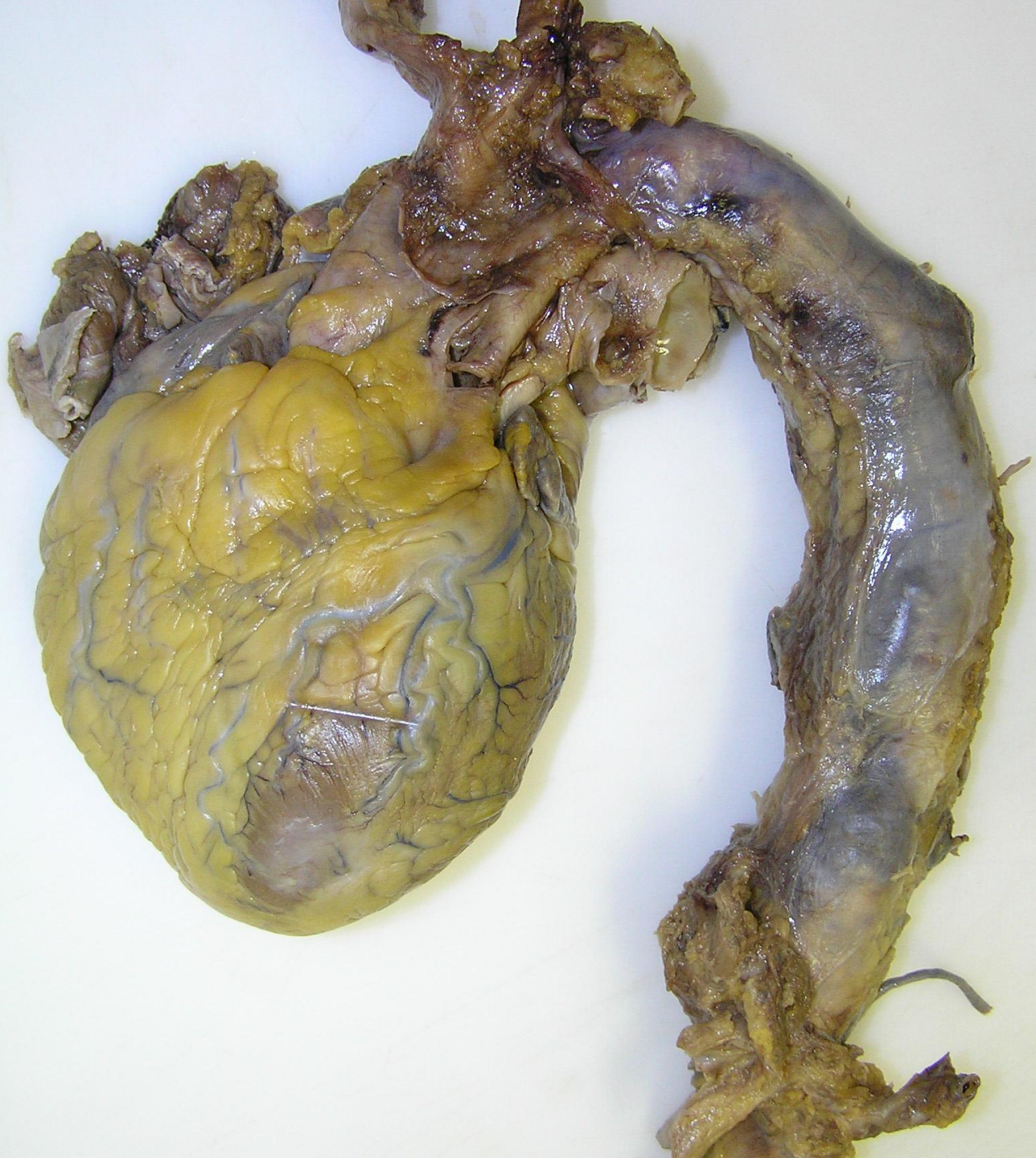


Normal aortic wall



Penetrating atherosclerotic ulcer

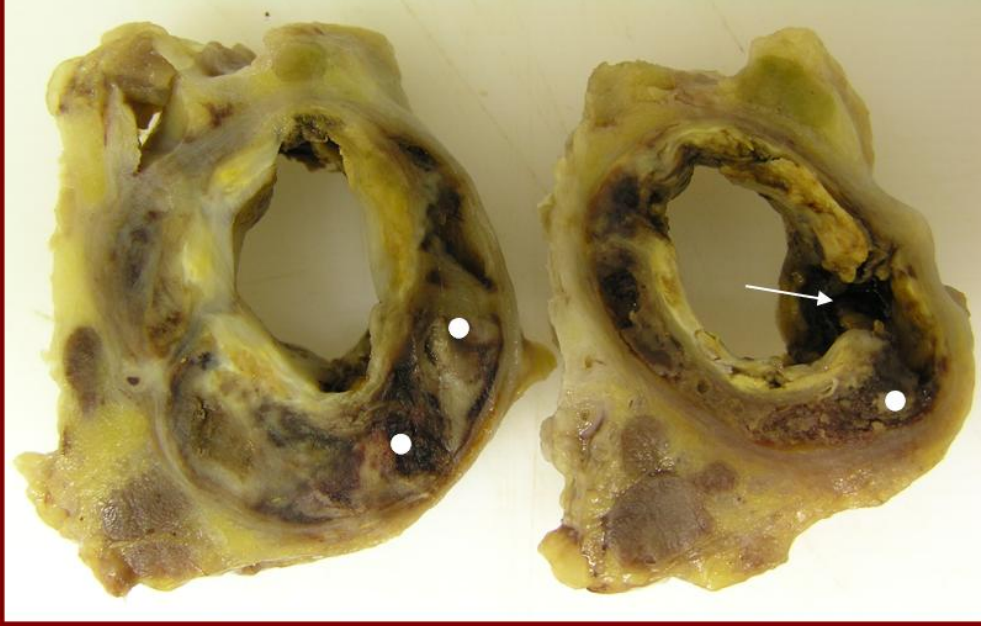
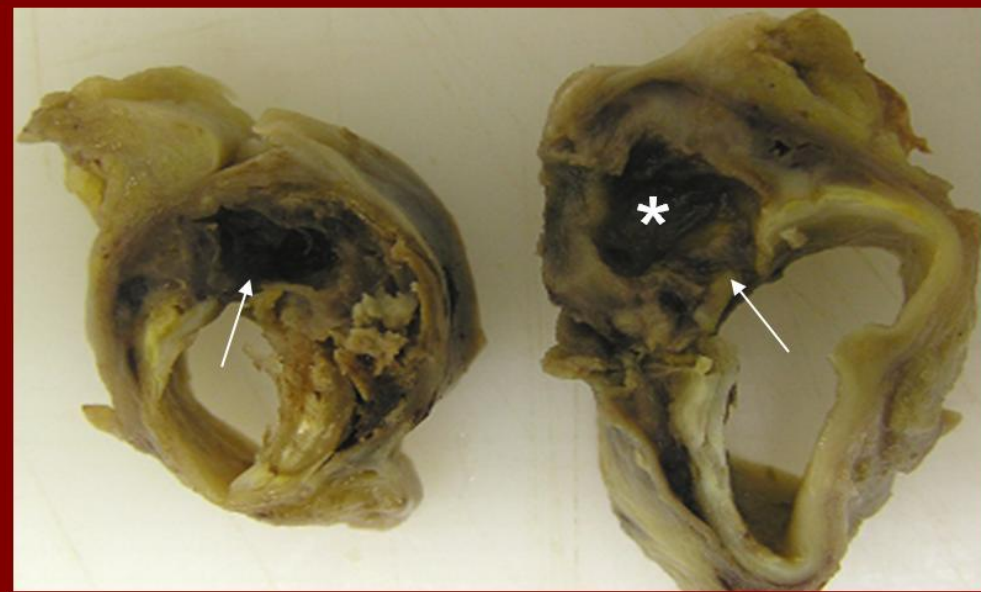


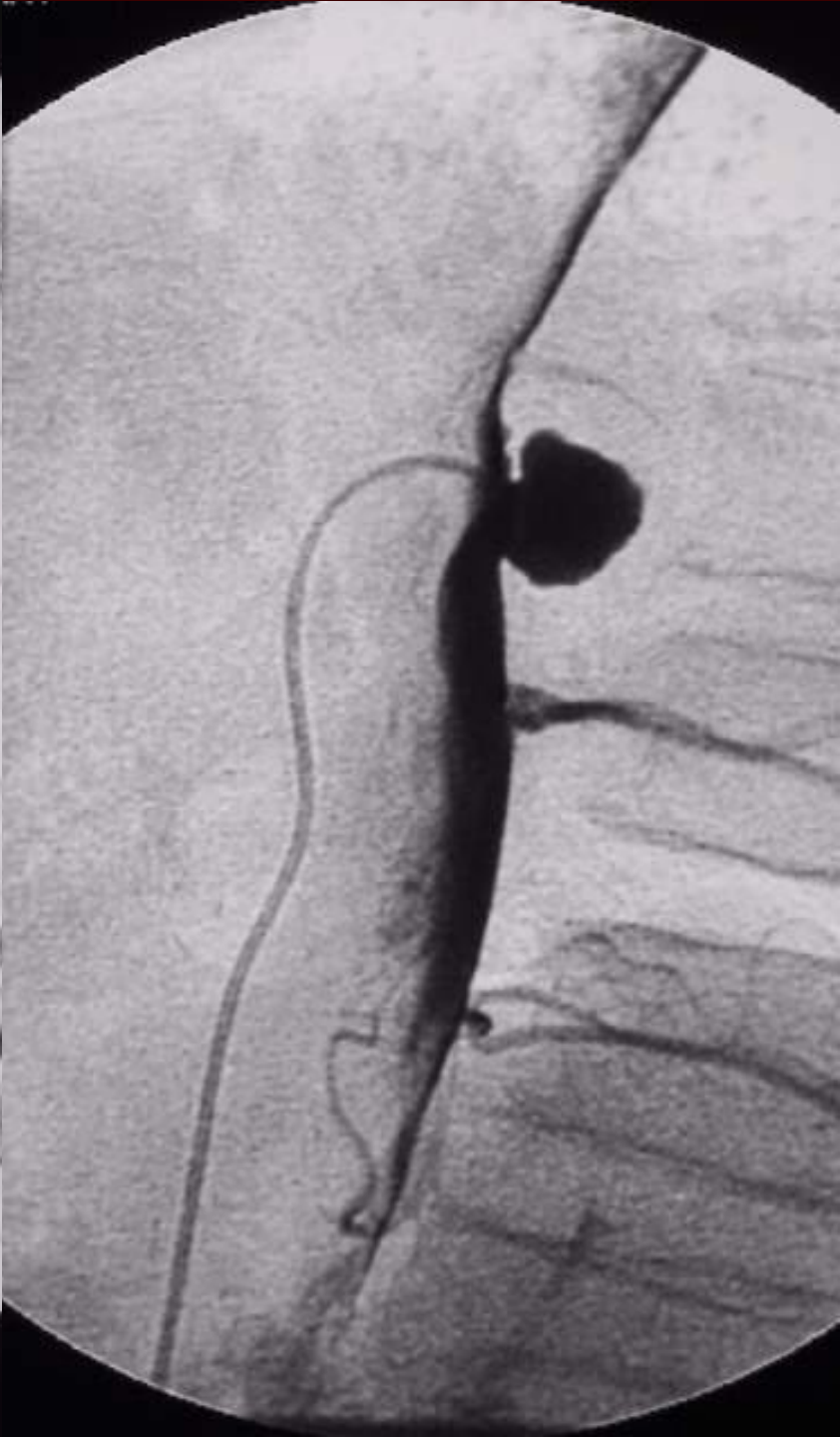


# Penetrating aortic ulcer



# Penetrating atherosclerotic aortic ulcer





# Clinical scenario-4

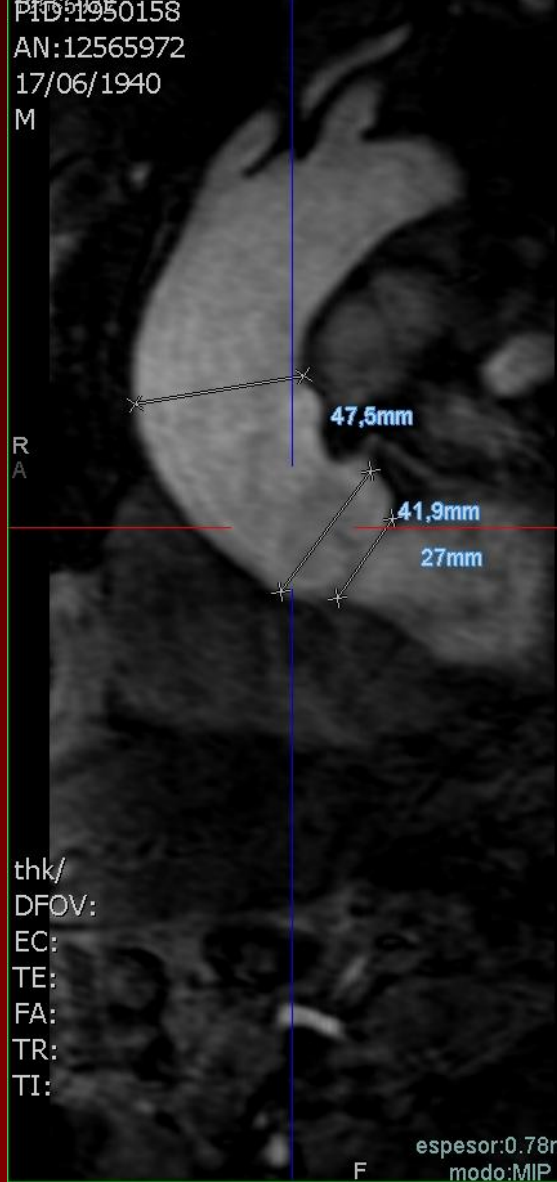
- 71year-old man with severe hypertension.
- **Acute chest pain** + dyspnea & sweat (17-10-10)
- During 2-3 days had mild thoracic pain.
- **H: Physical exam:** BP: 180/80 mmHg.
- **Chest-X ray:** prominent aortic Knob; no signs of heart failure.
- CT: aortic aneurysm. MR: aortic aneurysm.
- CAT: aortic aneurysm + discrete aortic bulging.

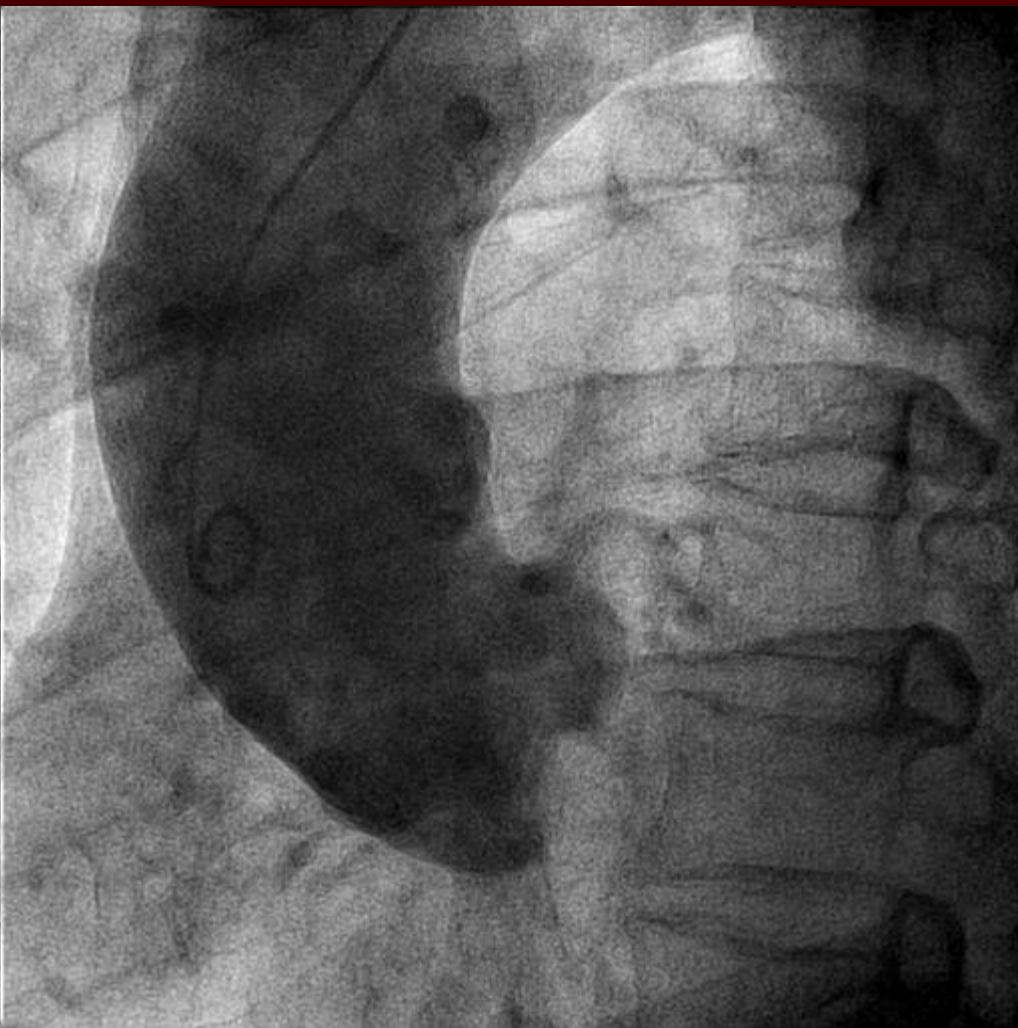
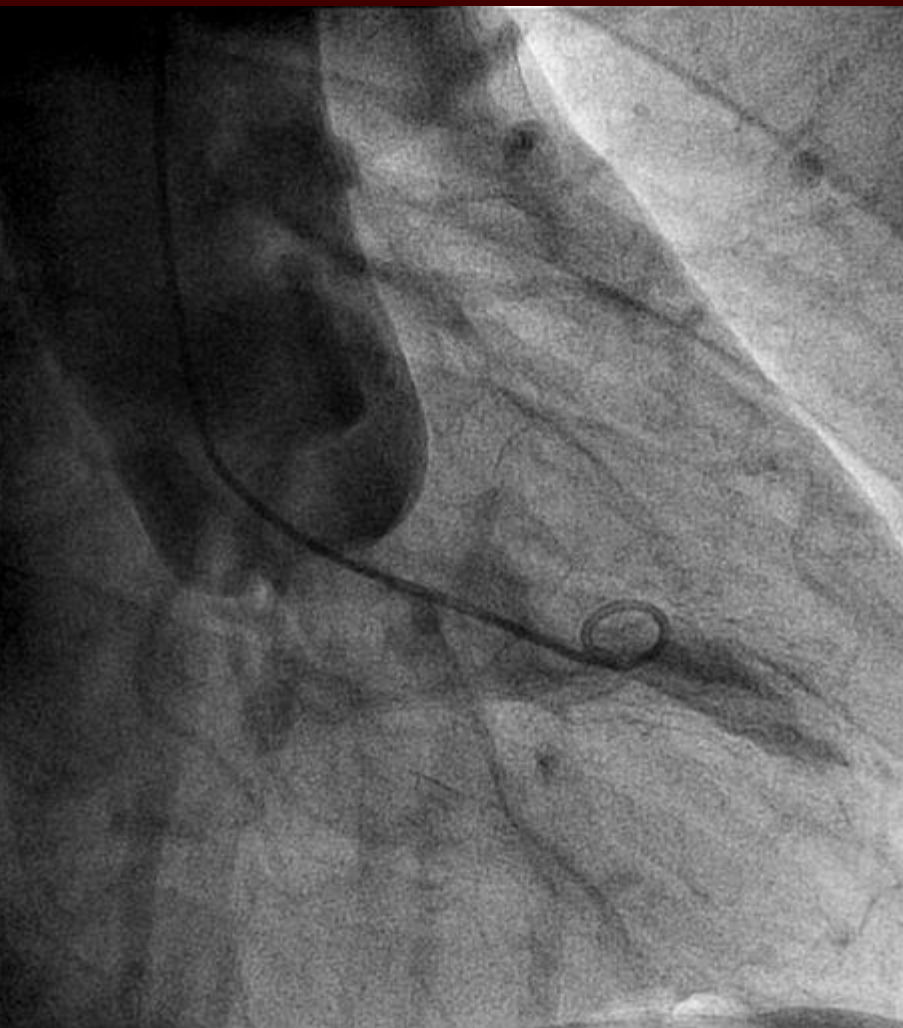
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PID:1950158  
AN:12565972  
17/06/1940  
M

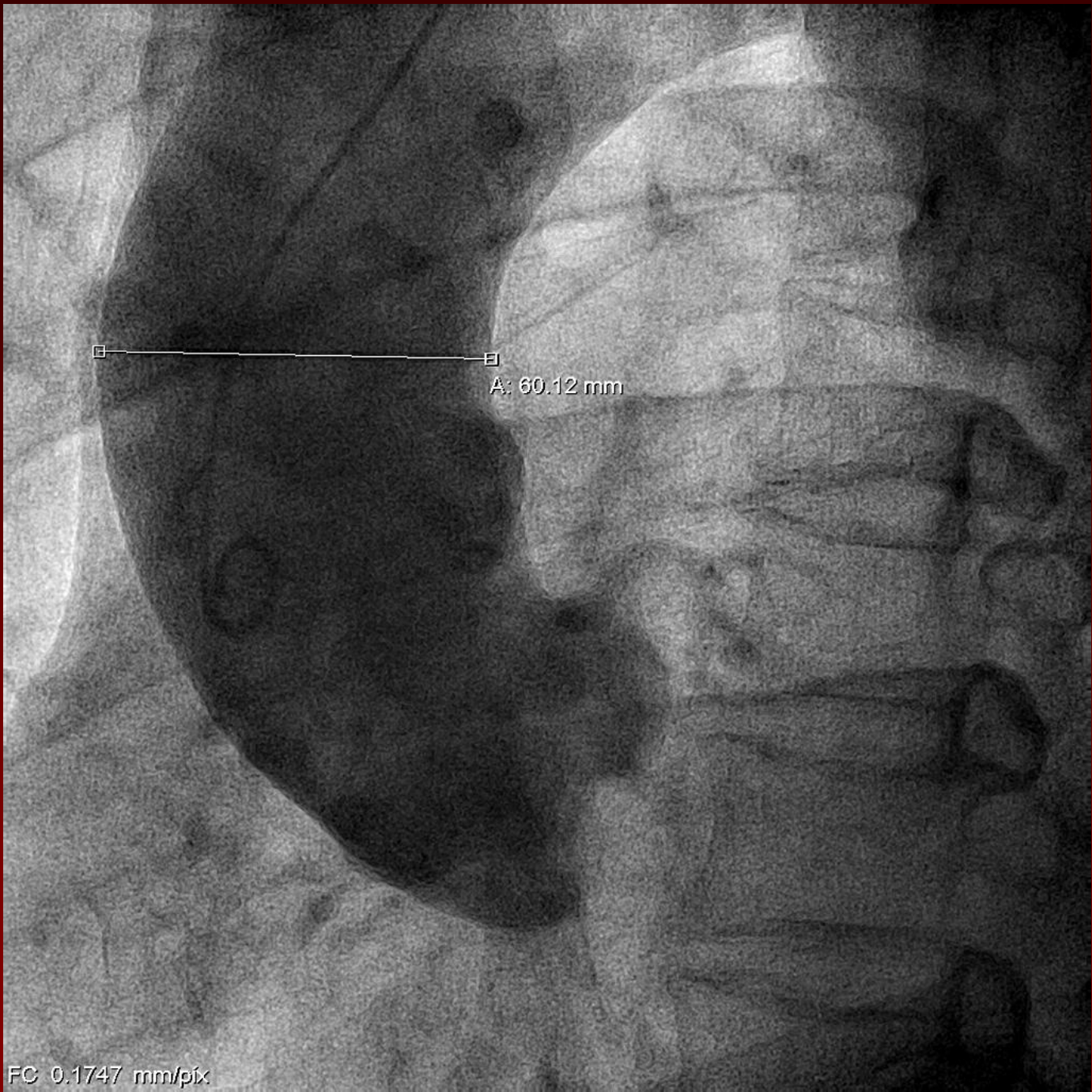
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HOSP CLINICO SAN CARLO  
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PID:1950158  
25/11/2013  
13:20:00  
M

CRUZ PALOMINO, MARCELO A  
CRUZ PALOMINO, MARCELO,  
PID:1950158  
AN:12565972  
17/06/1940  
M

ANGIO RM DE TORAX CON CONTRAS  
HOSP CLINICO SAN CARLO  
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25/11/2013  
13:23:40  
M



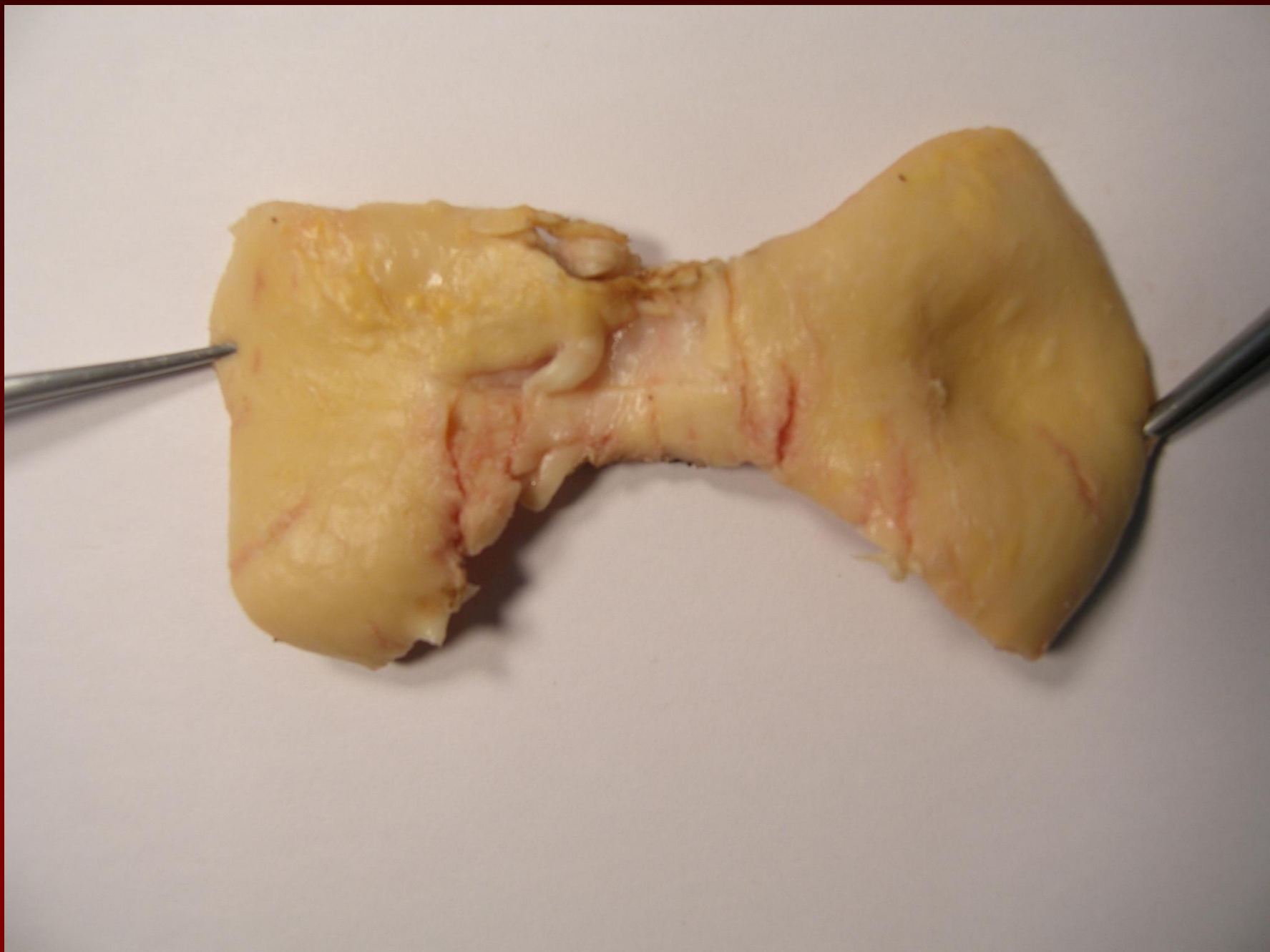


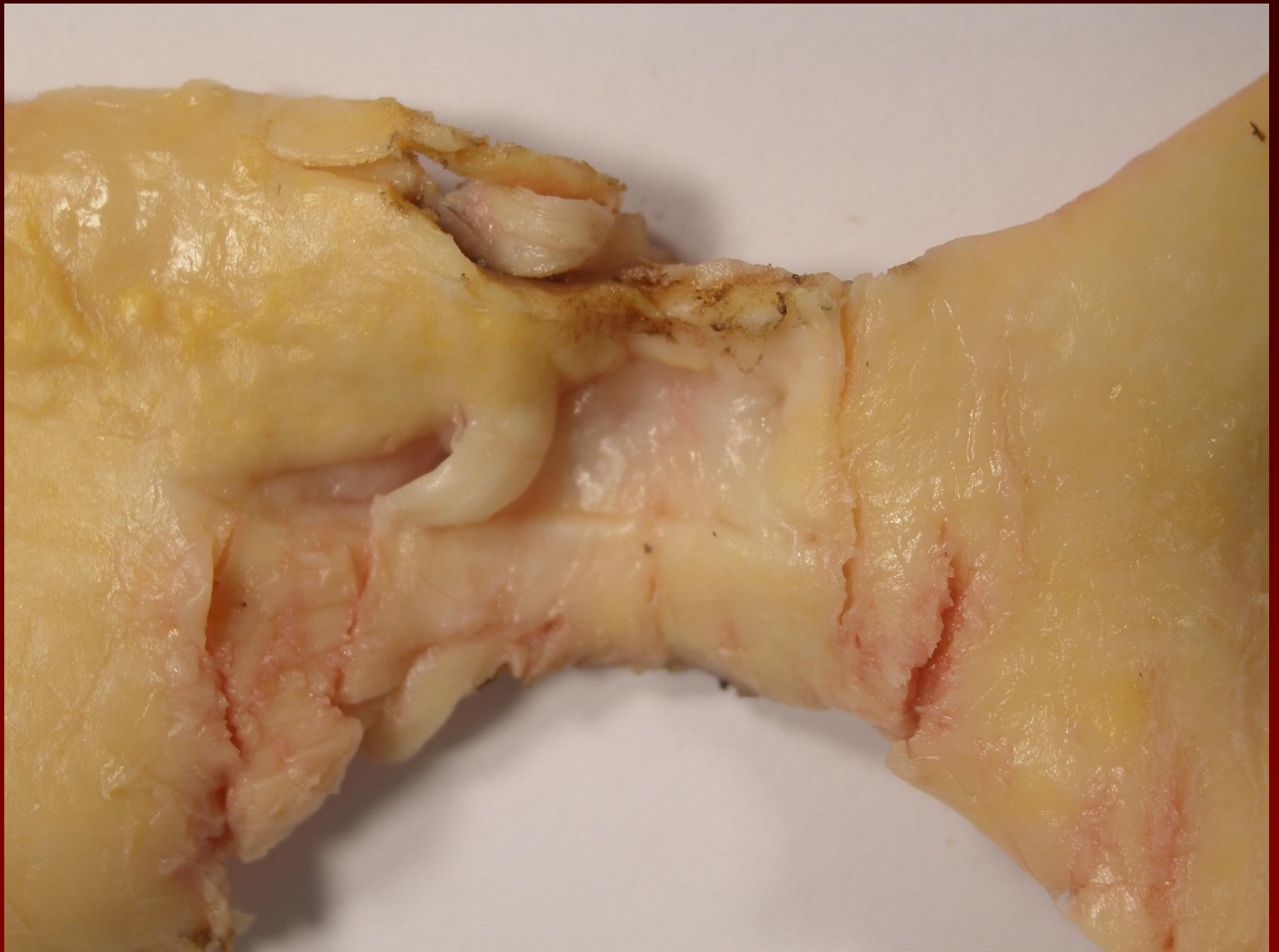


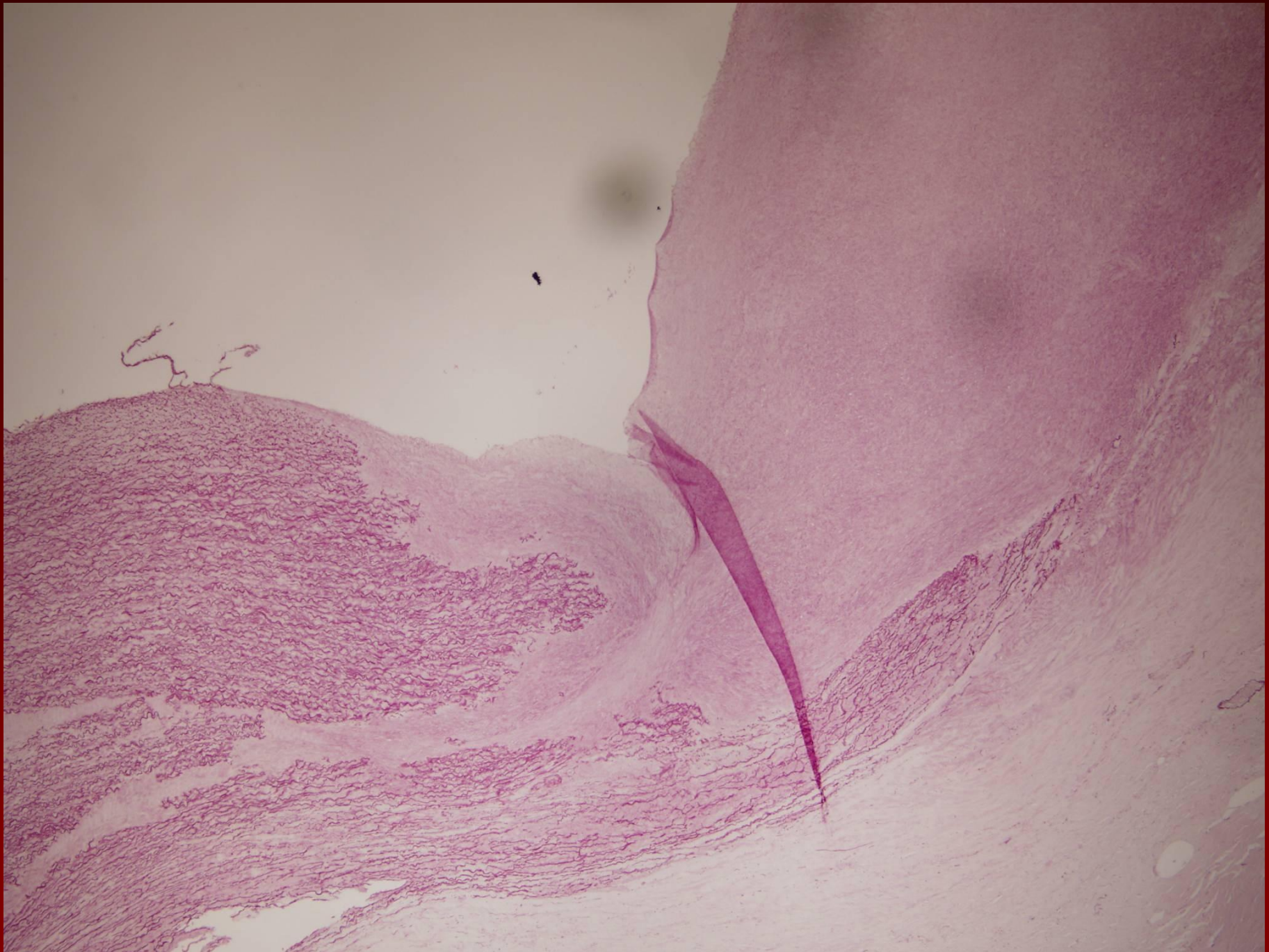
A: 60.12 mm

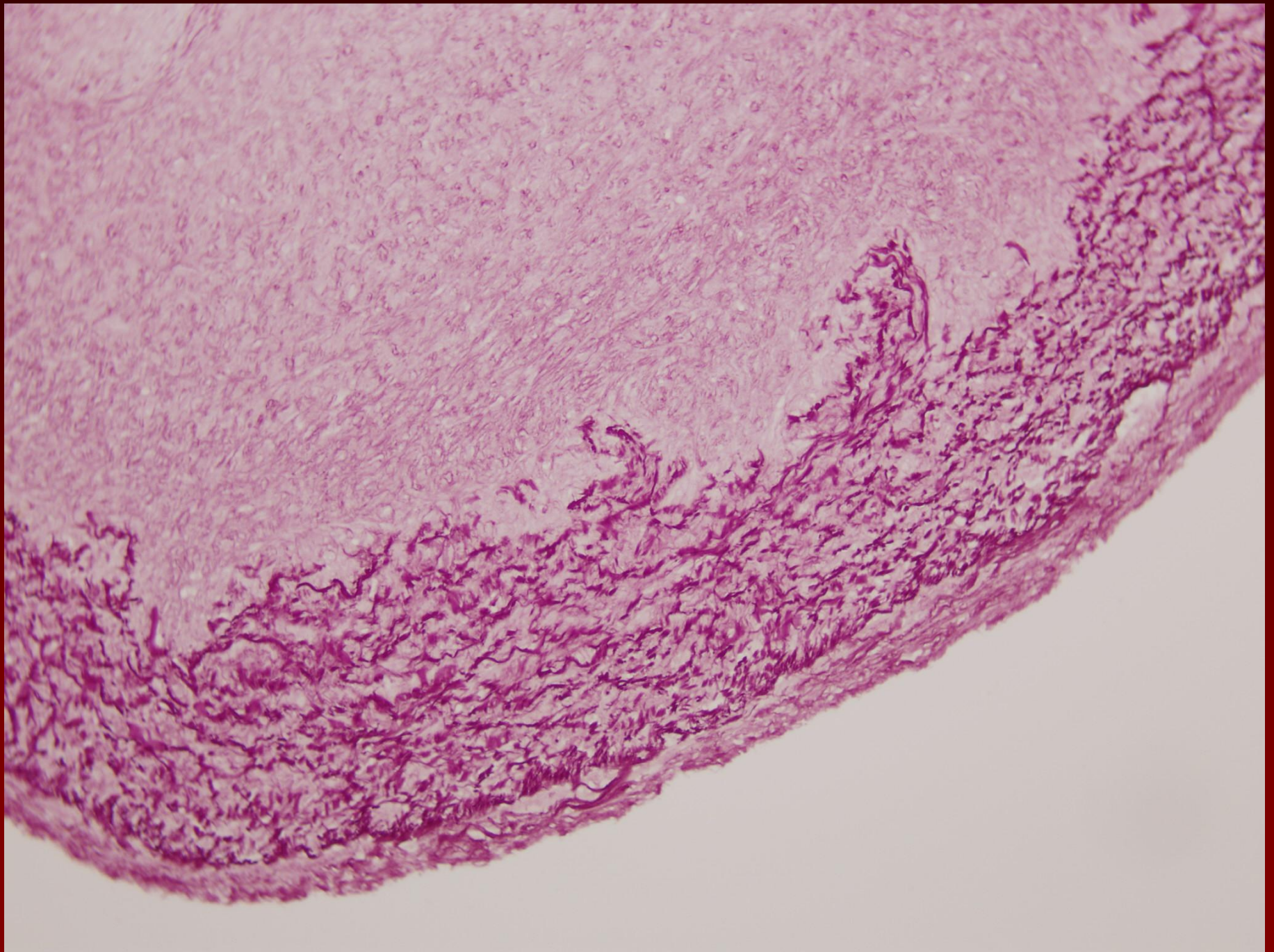
FC 0.1747 mm/pix



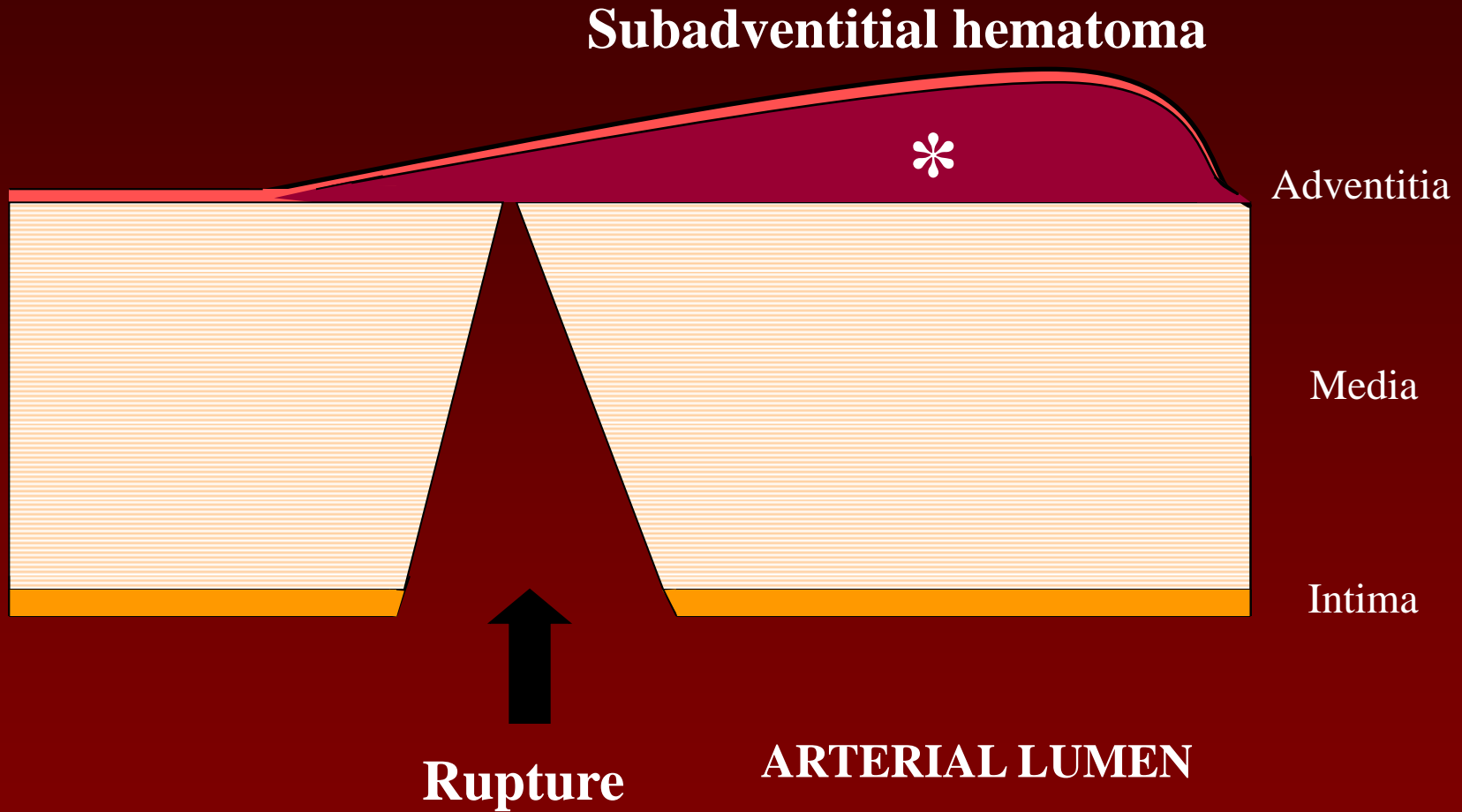






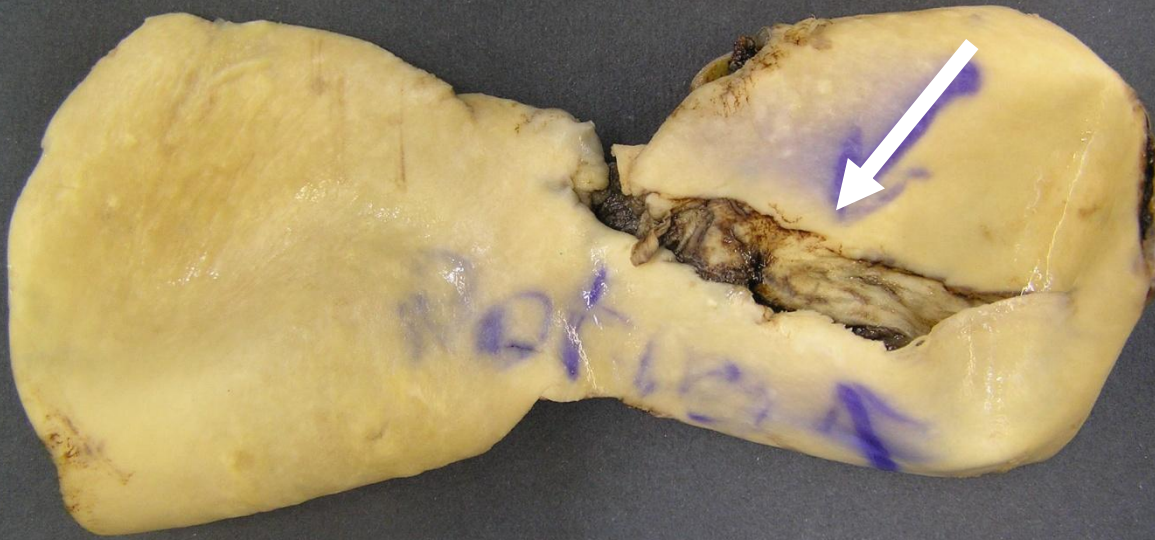


# INCOMPLETE DISSECTION

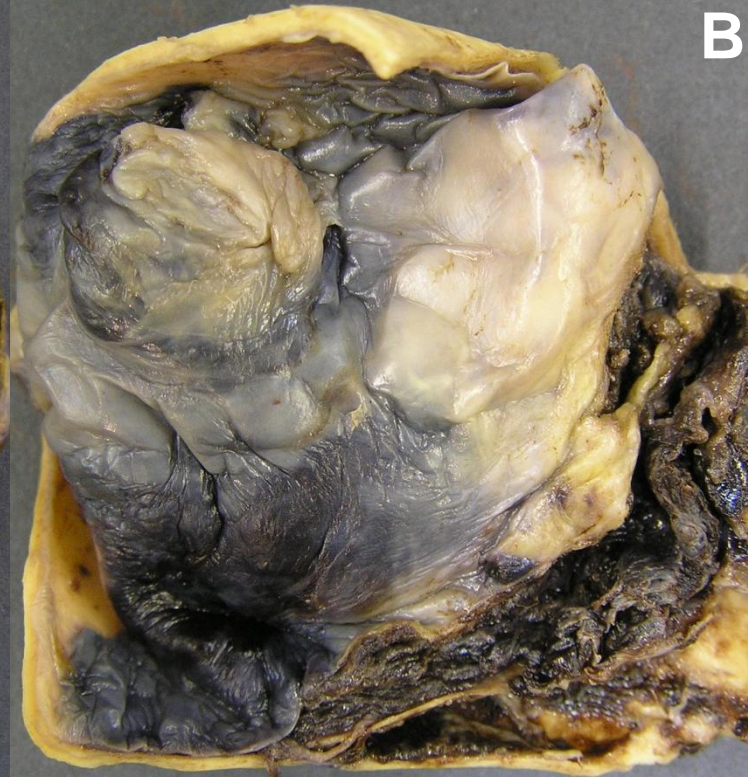


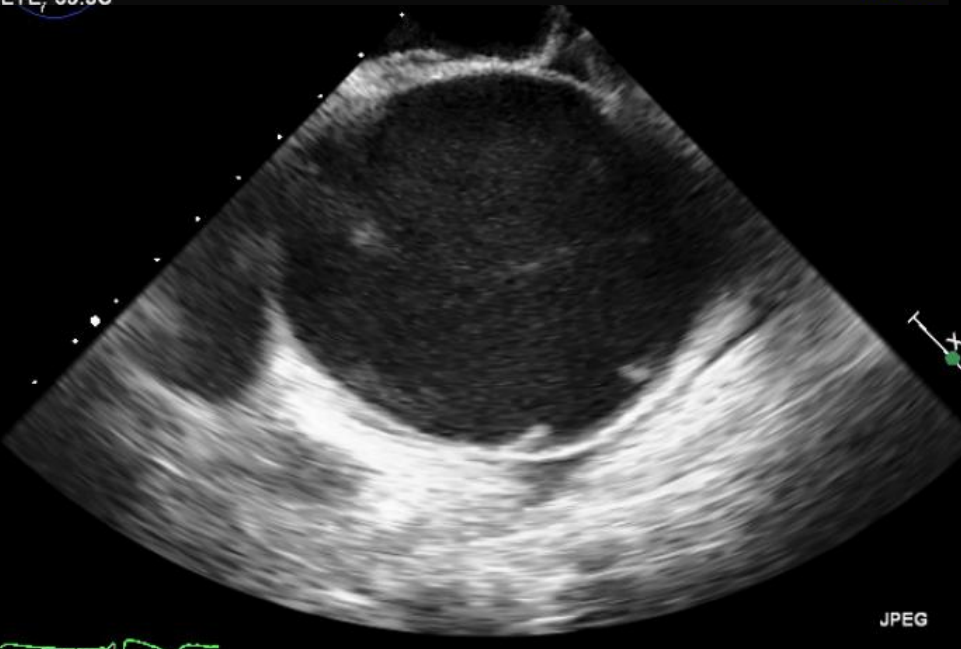
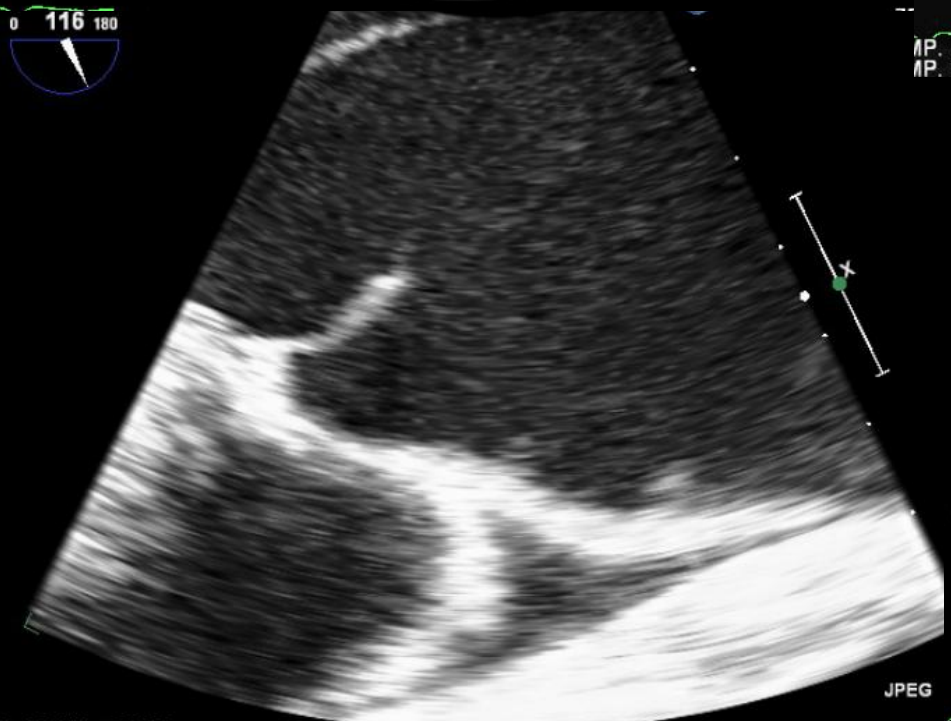
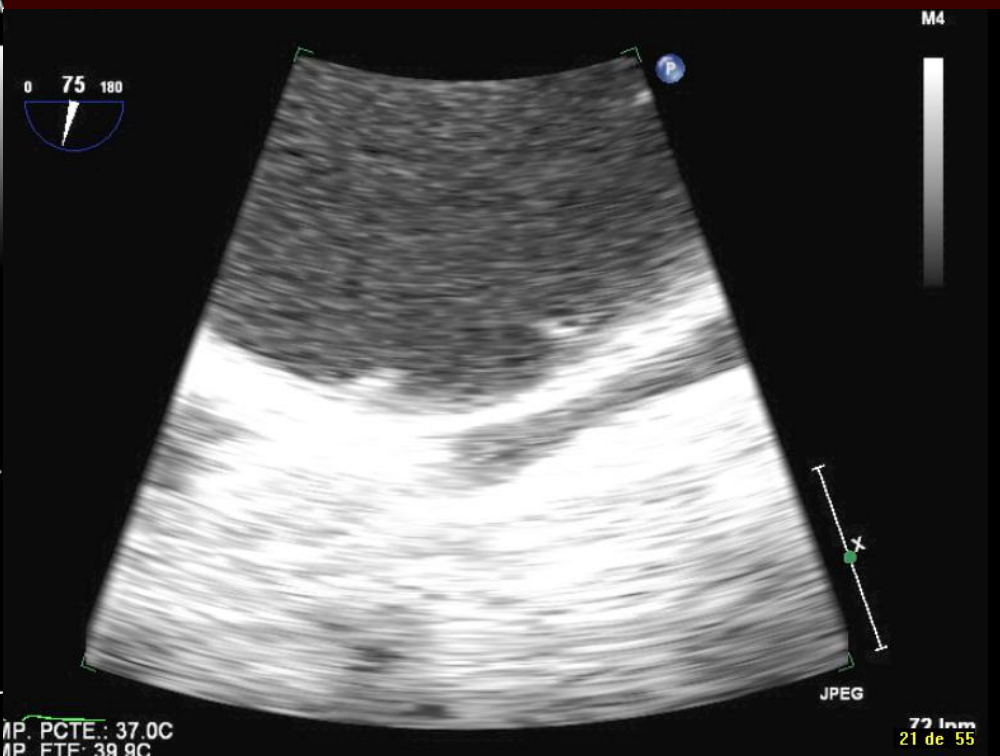
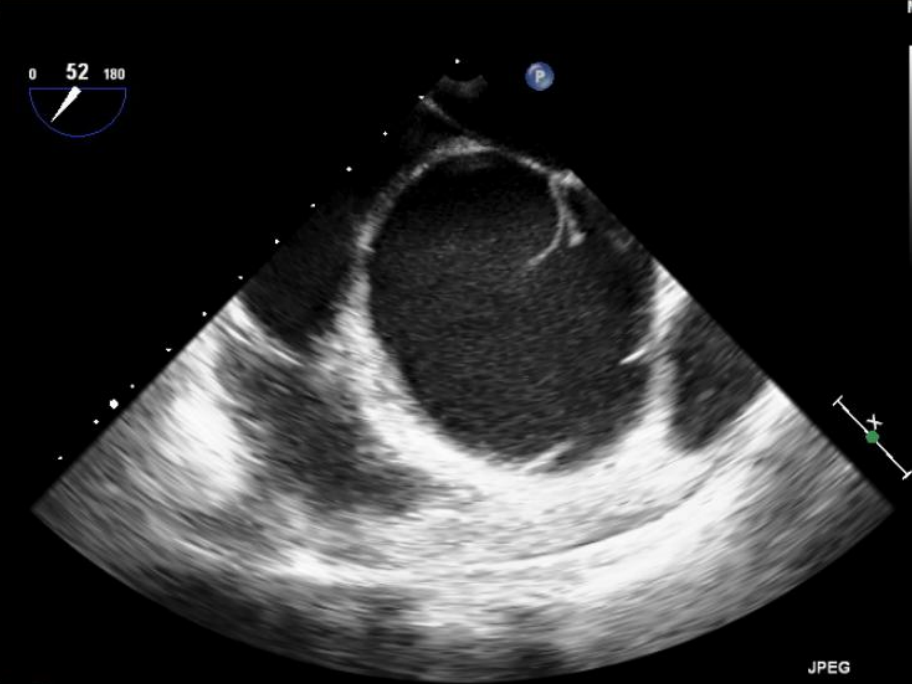
# INCOMPLETE DISSECTION

A

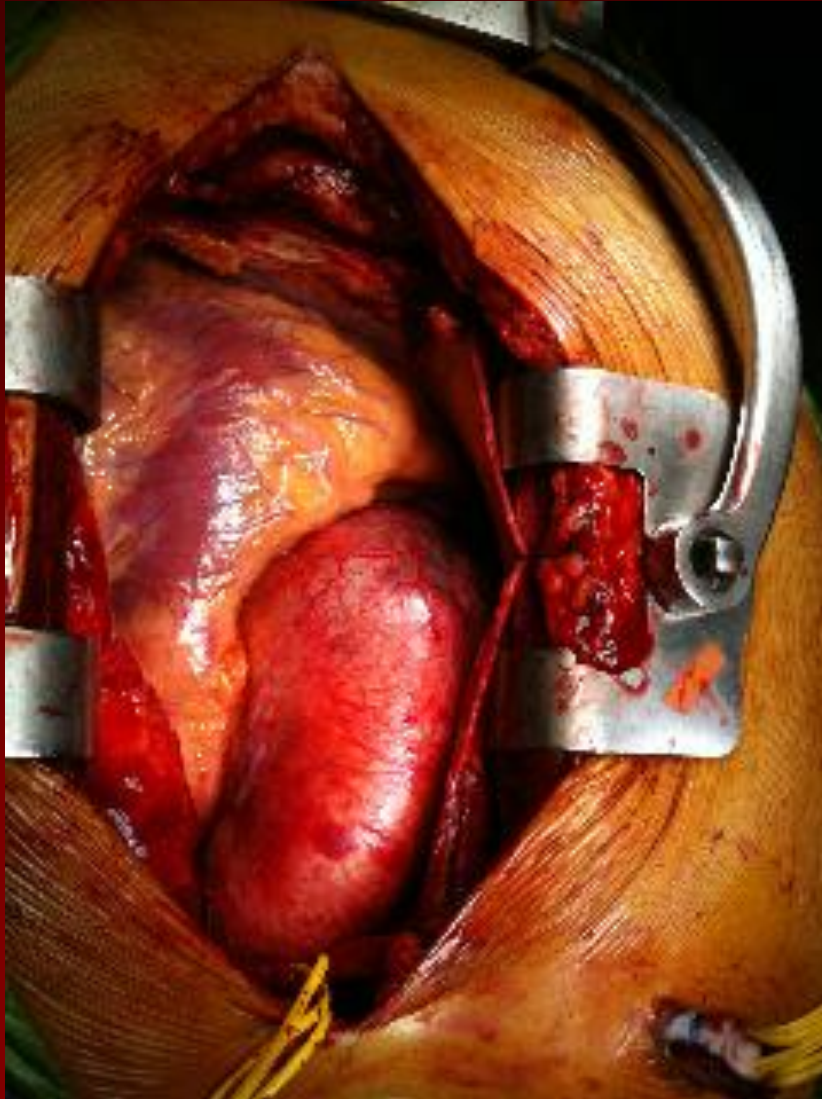


B





# SURGICAL PROCEDURE



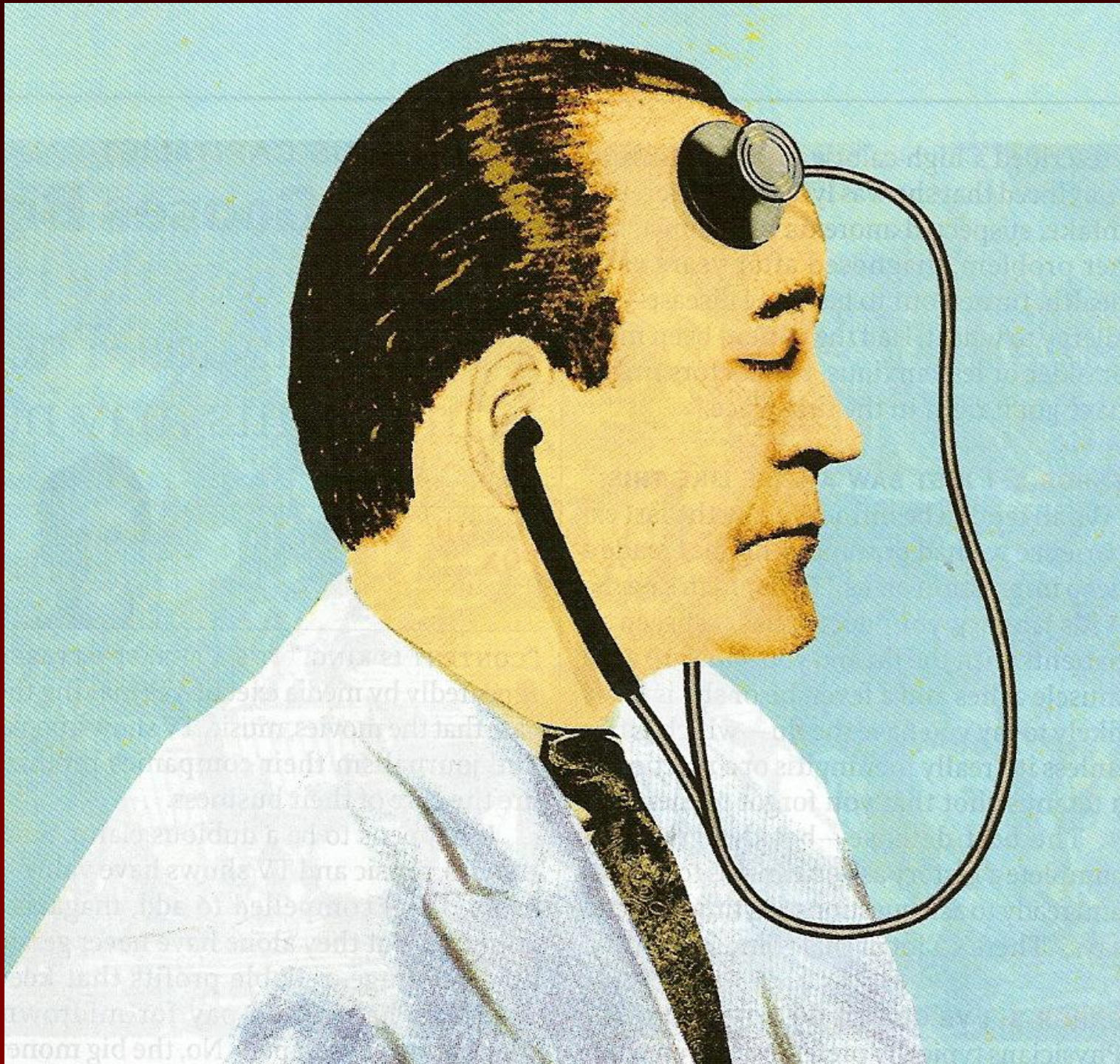


# Conclusion

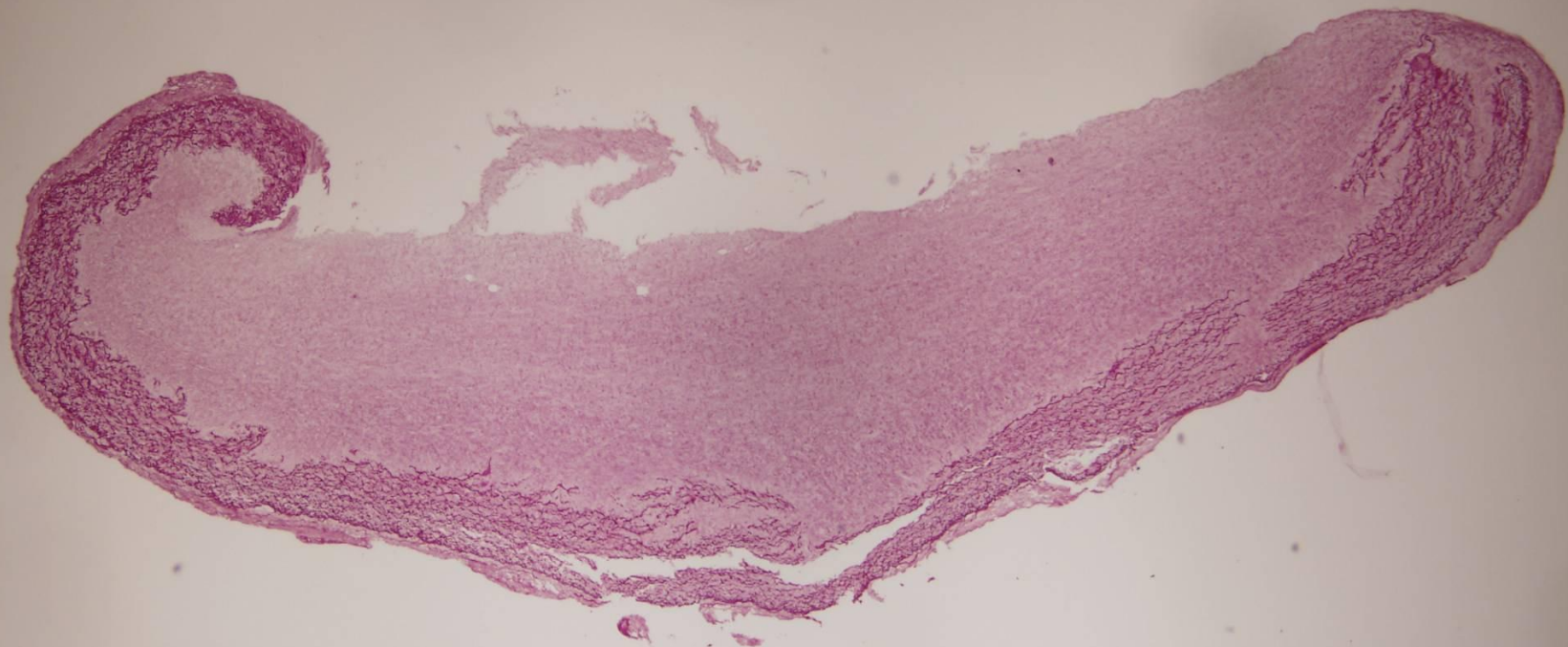
- Revision of the broad spectrum of acute aortic pathology, focusing on the distinctive findings of each entity and emphasizing the clues for their diagnosis.
- Increased attention to the different morphological aspects of this panoply of entities that make up the AAS will result in a more accurate diagnosis of acute aortic pathology, better knowledge of their natural history and the recognition of important prognostic predictors.

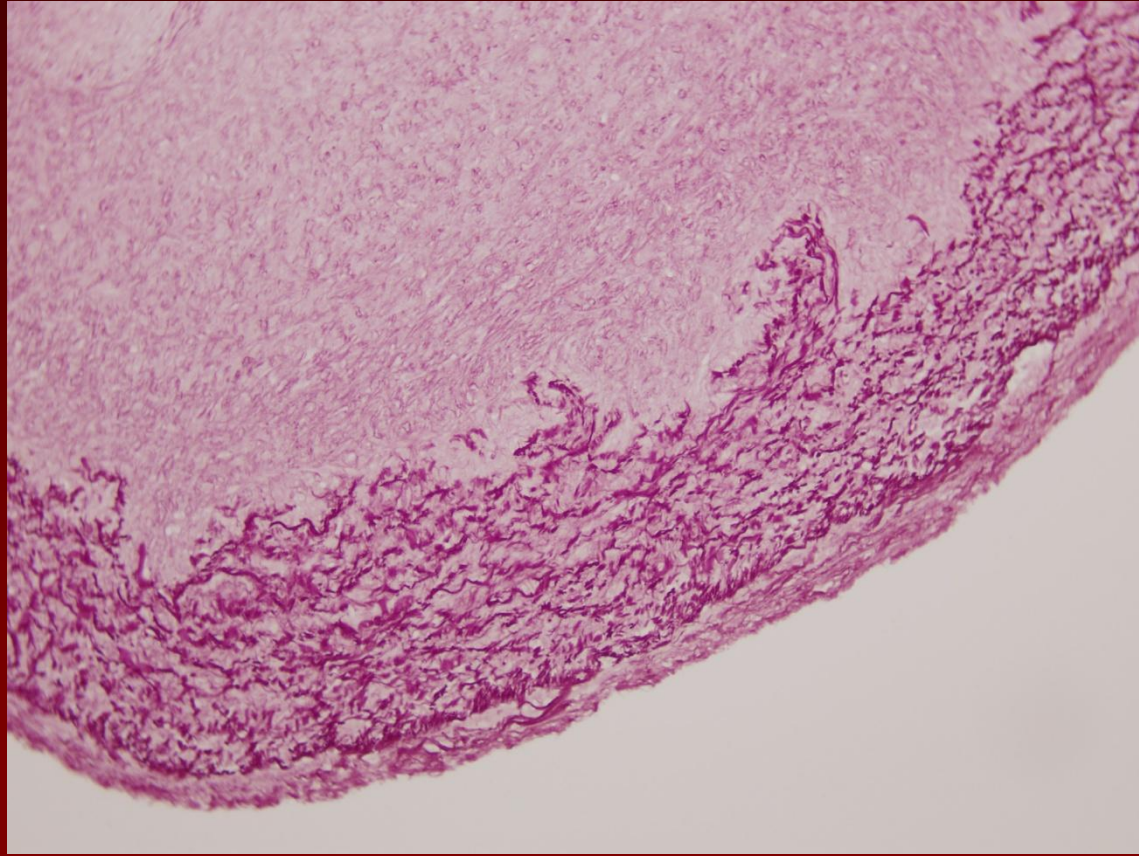
# Paradigm shift in AAS; conclusion

- 25 yr ago: 30% p with AD were not properly diagnosed.
- 15 yr ago: 35% of AD were not subjected to imaging.
- Nowadays, profuse imaging (CT, TEE) use in ER: (imaging equipment / doctors skill).
- **Success will mostly depend on an exquisite, conscientious and profound clinical assessment of ER clinician.**











# Diagnosis ?

- **PAU accompanied by some degree of intramural haemorrhage.**
- **IAH with an ulcer like projection.**
- **Type B aortic dissection with an almost completely thrombosed false lumen.**



# Diagnosis ?

- PAU accompanied by some degree of intramural haemorrhage.
- IAH with an ulcer like projection.
- Type B aortic dissection with an almost completely thrombosed false lumen.

**INTIMAL RUPTURE**

# Clinical presentation

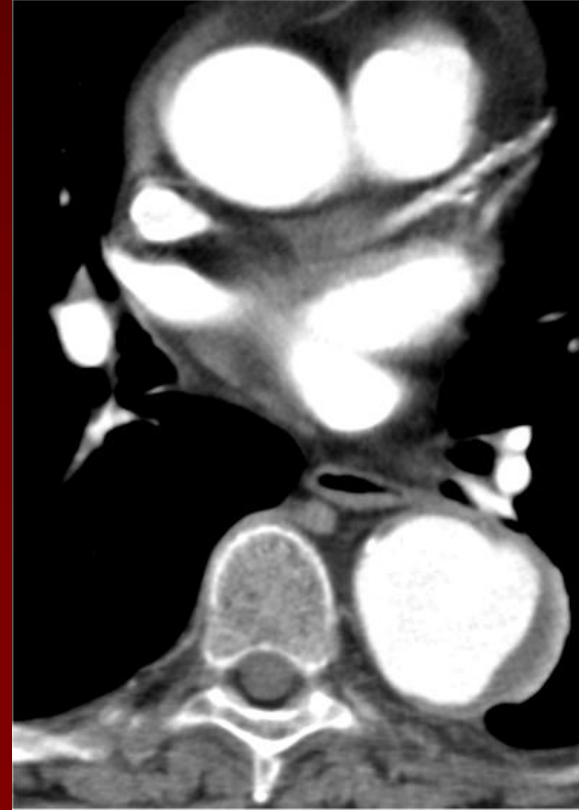
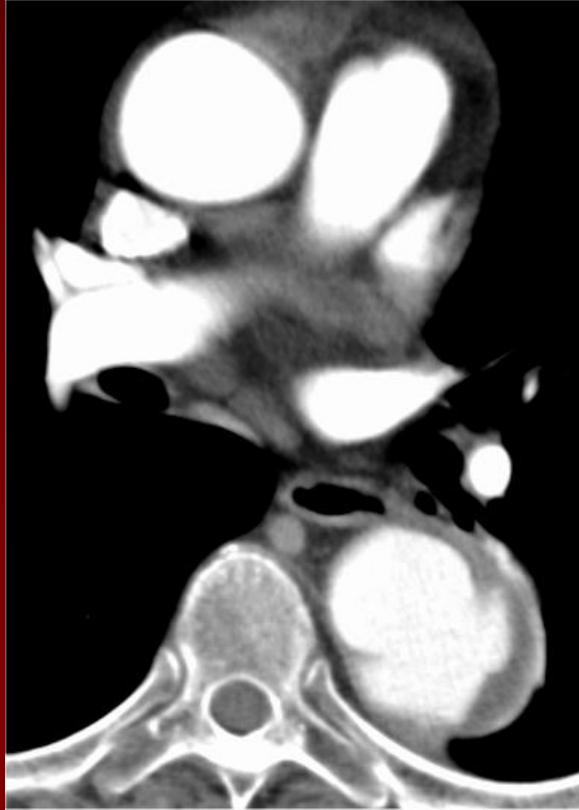
## Type B - PAU / IAH.

- **Symptomatic:**
  - **Stable.**
  - **Unstable:**
    - Persistent pain.
    - Lesion progression on serial imaging.
    - Signs of imminent rupture.
    - End organ ischemia.
- **Asymptomatic:**
  - **Stable.**
  - **Unstable:** lesion progression.

# Risk factors

- **Clinical risk factors:**
  - Difficulties in **blood pressure control**.
  - Persistent or recurrent **pain**.
  - **Hemodynamic instability**.
- **Imaging risk factors:**
  - Aortic **diameter** ( $\geq 50$  mm).
  - Hematoma **thickness** ( $\geq 10$ -12 mm).
  - **Presence** & size ( $> 20$  mm) of intimal tear.
  - Large or **increase** in size of pleural effusions.

# Type B AAS. Intimal rupture. Evolution.



# “Severity score”

- **Clinical risk factors:**
  - Persistent or recurrent pain 2
  - Hemodynamic instability 2
  - D-dimer reevaluation 1
- **Imaging risk factors:**
  - Large intimal tear 1
  - Aortic  $\varnothing \geq 50$  mm or increase in size 1
  - Increase of IAH thickness or PAU depth 1
  - Increasing pleural effusion 2

# Goals of treatment

- **Eliminate or reduce symptoms.**
- **Prevent lesion progression.**
- **Prevent aortic rupture.**
- **Prevent short & long-term death.**

# Therapeutic options

- **Medical treatment:**
  - Control of systolic **blood pressure: antihypertensives.**
  - Administration of **beta blockers.**
  - Patient **monitoring** (BP <120 mmHg, D-dimers).
  - Strict **bed rest**, iv line.
  - Control pain **analgesia** (morphine).
  - Strict imaging follow-up.

# Surgical treatment

- **Basic principle:**
  - Replacement of the pathologic segment.
  - Local excision and graft interposition.
  - Prosthetic-aortic sutures in areas of normal wall.
  - Teflon banding reinforcing.
  - Biological glues for a complete sealing.
- **Main considerations:**
  - Patient risk: age, comorbidities, overall physical condition, and anticipated life expectancy.
  - Aortic morphology and tissue quality.



# Endovascular treatment

- Completely **covered stent-grafts**.
- **Landing zone**: should include proximal and distal area of **normal aortic wall**.
- Careful **imaging protocol**: positioning, landing and selecting the appropriate stent graft (diameter and length).
- Check feasibility of **endovascular access**.
- **Avoid** using a modeling **balloon** to complete stent-graft deployment.

# Endovascular treatment

- **High surgical risk** for open surgical repair.
- **Favourable anatomic target**: localized lesion with normal-sized, minimally angulated, cylindrical proximal and distal landing zones of adequate length.
- **Adequate vascular access** (arterial size, lack of excessive tortuosity and occlusive disease).

# EVAR vs surgery for descending thoracic aortic disease

- Metaanalysis of 42 studies; 5888 patients.
- Similar characteristics between TEVAR and open groups excepting age ( $54 \pm 13$  vs  $51 \pm 13$  years).
- **All cause mortality** (1, 2-3 yr) did not differ.
- **Overall risk of stroke** similar except for MC.
- **Paraplegia or paraparesis** was significantly reduced for TEVAR vs open surgery.

# EVAR vs surgery for descending thoracic aortic disease

- **TEVAR significantly reduced:**
  - The risk of renal dysfunction.
  - Reoperation for bleeding.
  - The need of transfusion.
  - Respiratory complications.
  - Hospital length of stay (- 7 days).
  - Total ICU length of stay (- 4 days).
  - Procedure time (mean of 140 min).

# Therapeutic strategy

- Evaluate clinical & imaging risk factors:
  - Severity score  $< 3$ :
    - Medical treatment is the rule.
    - Clinical & imaging follow-up is mandatory.
  - Severity score  $\geq 3$ :
    - Active treatment (TEVAR or open surgery).

# Therapeutic strategy

- **Intimal rupture without IAH:**
  - **Endografts.**
- **Intimal rupture with IAH:**
  - **Endografts:** anchor the endograft in nondissected wall.
- **IAH without intimal rupture:**
  - **Normal caliber Ao: endografts** covering descending Ao. ?
  - **Aneurysmal aorta: surgery ?.**

# Conclusions

- **Use severity score for clinical decision making.**
- **Medical treatment is the currently accepted treatment when the patient is stable.**
- **Use EVAR in most patients with this type of distal aortic lesions & high severity score.**
- **In the absence of definitive randomized controlled trials, the value of EVAR relative to conventional open surgical approaches will continue to be debated.**

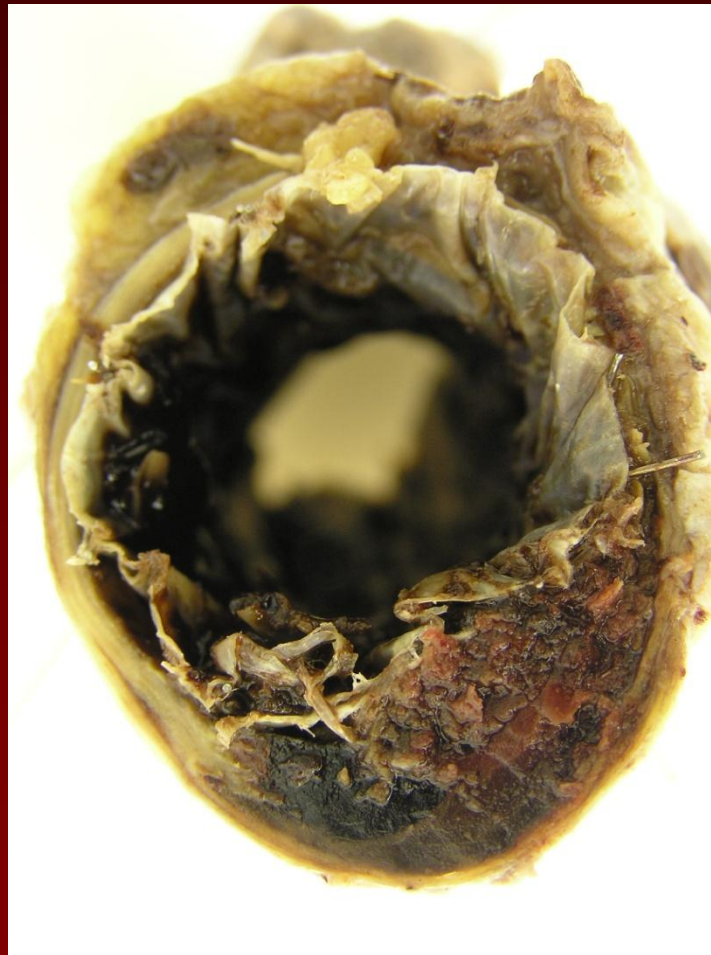
OV: 26.4cm  
FT/+



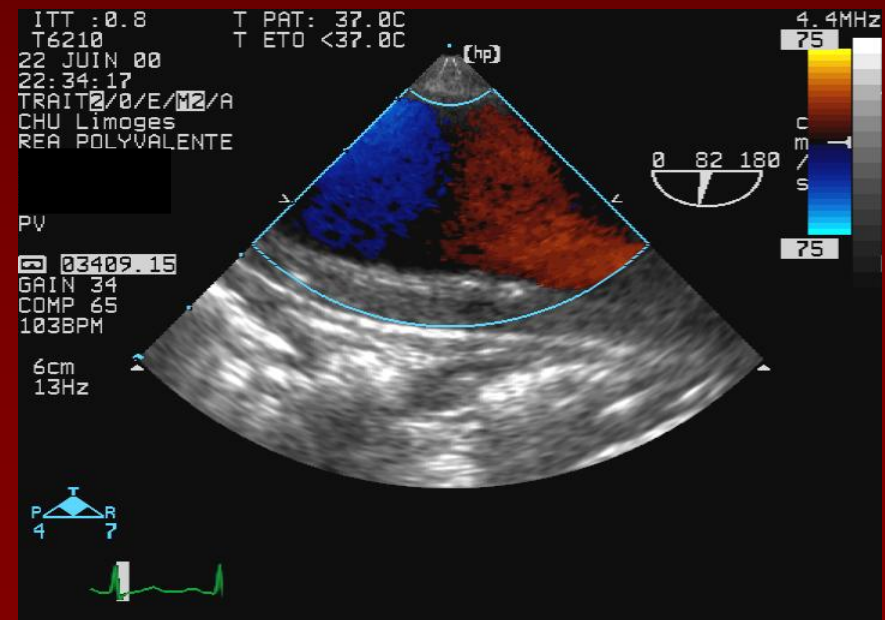
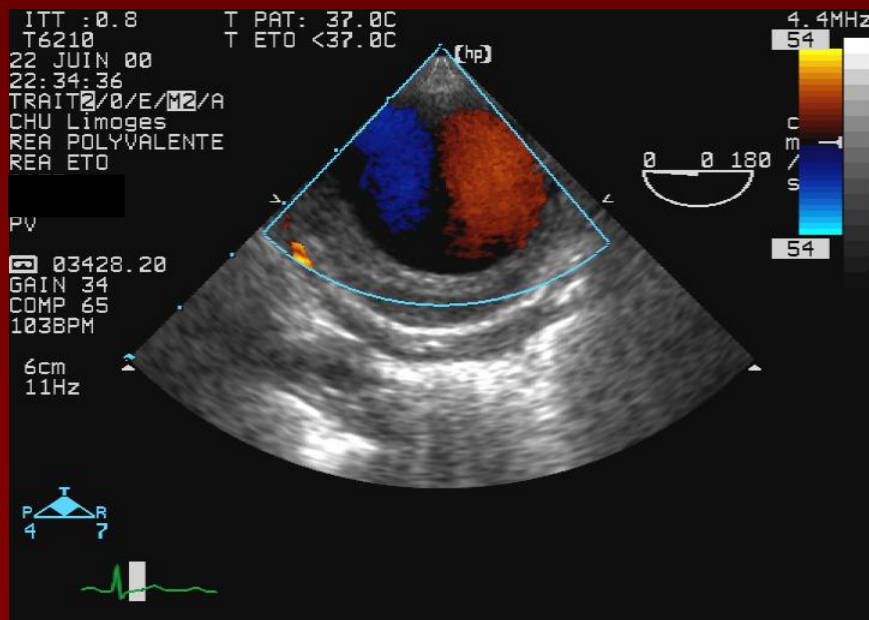
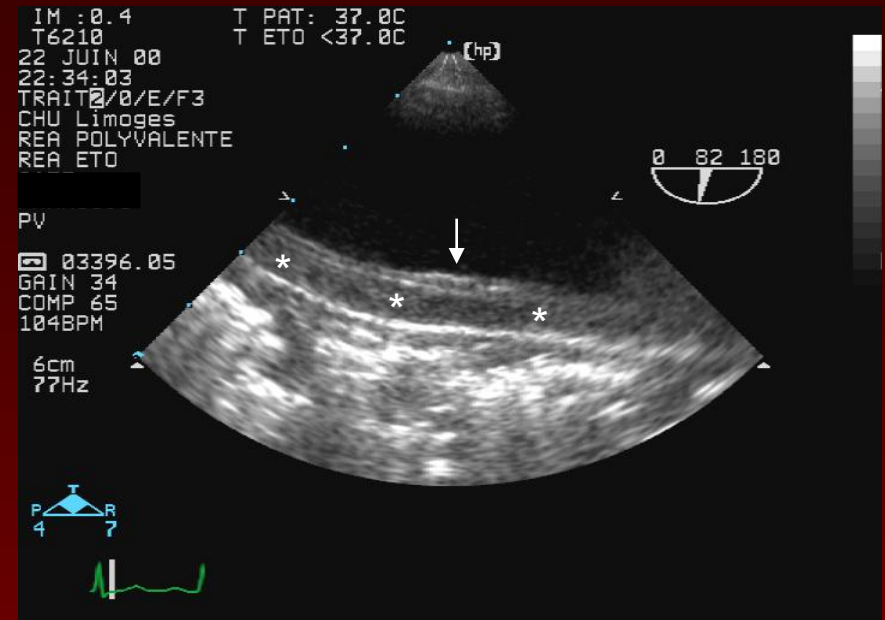
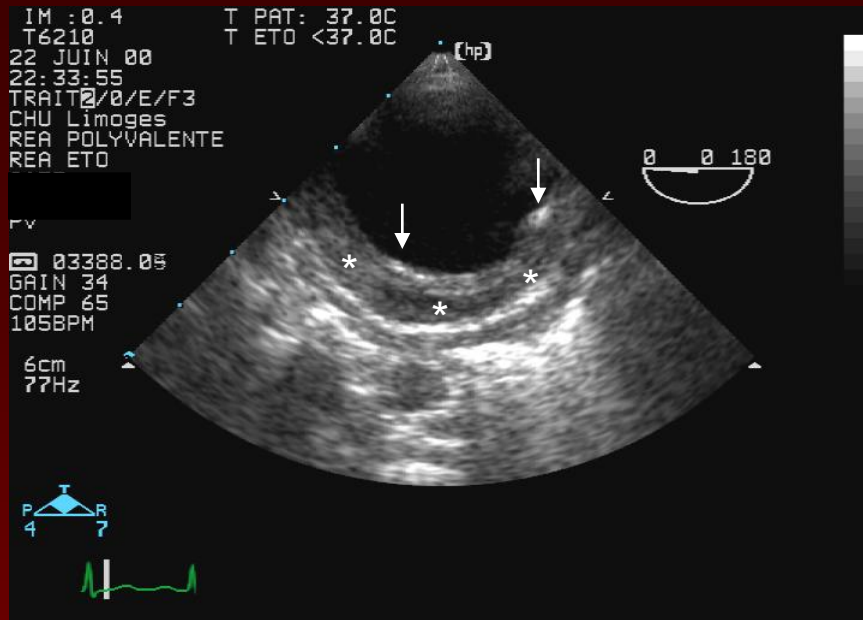
P  
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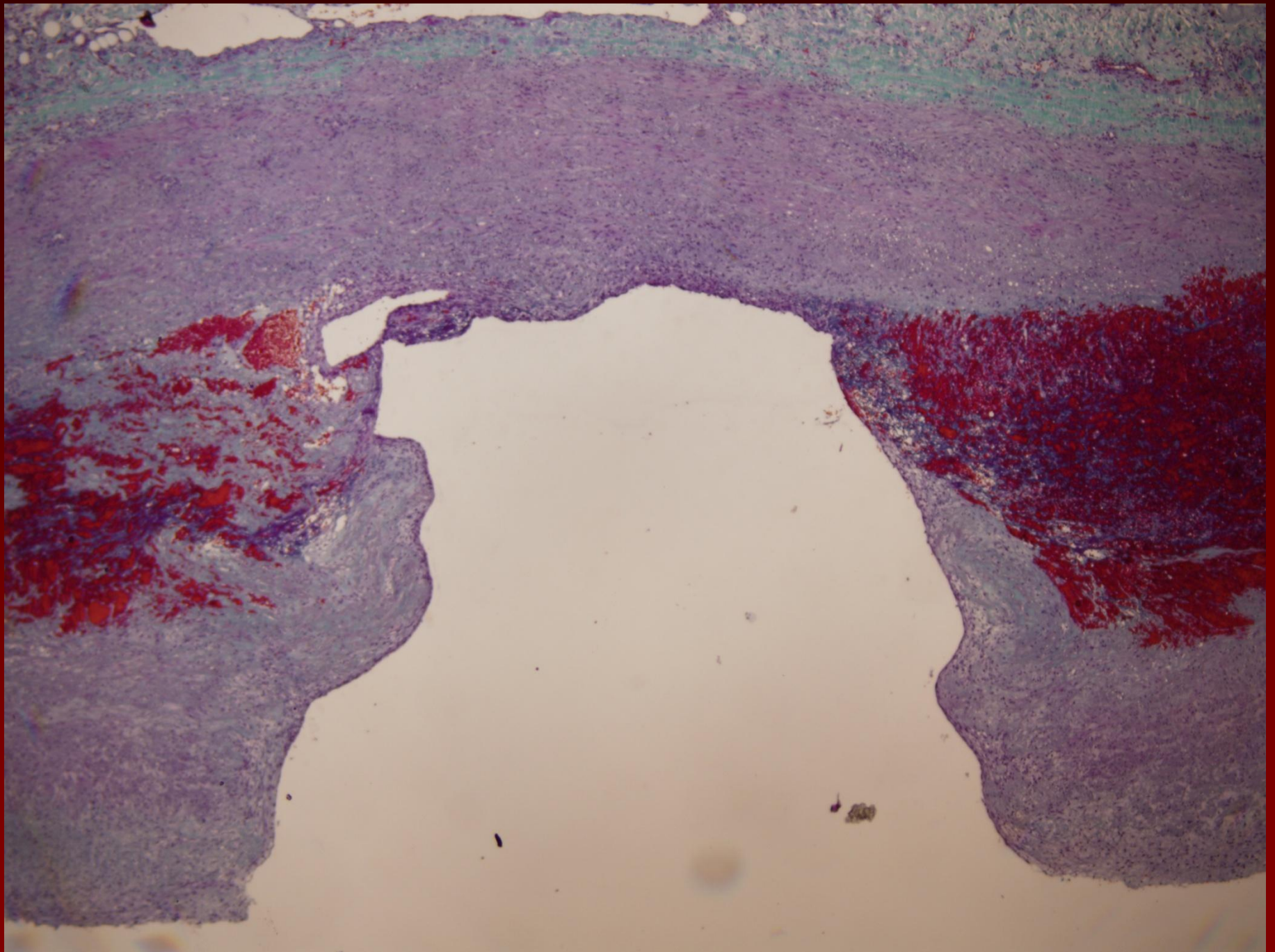
VOI  
120  
Mod.  
1.0.50s/HE+ 39.4mm/rot





# Intramural aortic hematoma





# UPA asintomática

- Definición-Anatomía patológica.
- Diagnóstico-Técnicas de imagen.
- Aspectos clínicos:
  - Sintomática (SAA, embolias, etc).
  - **Asintomática.**
- Diagnóstico diferencial.
- Factores predictores de progresión.
- Tratamiento.

# PAU / IAH.

## Therapeutic strategy

- **Acute Aortic Syndrome:**
  - **Type A:**
    - **Early surgery.**
  - **Type B:**
    - **Medical treatment (BP control &  $\beta$ -blockers) is the currently accepted treatment when the lesion is stable.**

# Why do we need an algorithm?

- Correct suspicion:  $< 50\%$  of presentations.
- Diagnostic delay of more than 24 h after hospitalization occurs in up to 39% of cases.
- Incidental discovery during an imaging procedure to assess for other diagnoses.
- Autopsies: the diagnosis is still missed in  $>10\%$ .
- Indiscriminate use of diagnostic imaging in poorly chosen patients with very low pretest probability of having AAS yields up to an 85% rate of false-positive results.

# Úlcera penetrante aórtica

- Complicación de algunas placas ateroscleróticas aórticas.
- **Rasgo distintivo:** erosión de la superficie que rompe la lámina elástica interna y penetra en la capa media.
- Hematoma intramural segmentario acompañante.
- Varones, edad avanzada, aorta “enferma”, aorta descendente, localizada-múltiples.

# Diagnóstico

- **Detección del cráter ulceroso.**
  - Imagen en forma de sacabocados que horada y deforma la pared aórtica y se rellena de contraste o Doppler color.
  - TC, RM, ETE, aortografía.

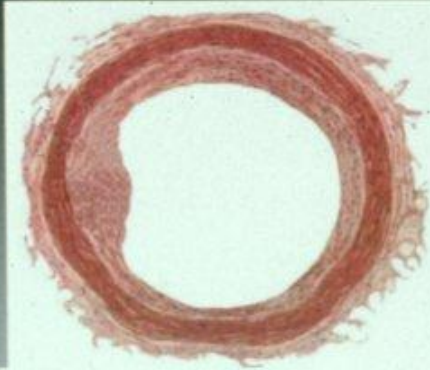


# Diagnóstico

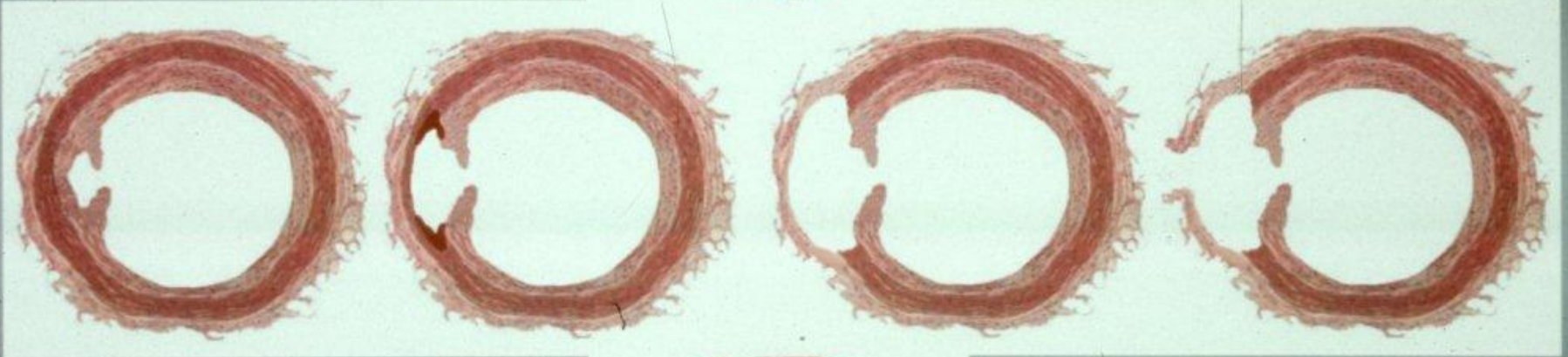


# ÚLCERA PENETRANTE AÓRTICA

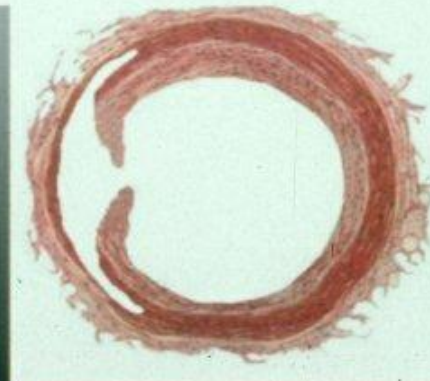
**HEMATOMA**



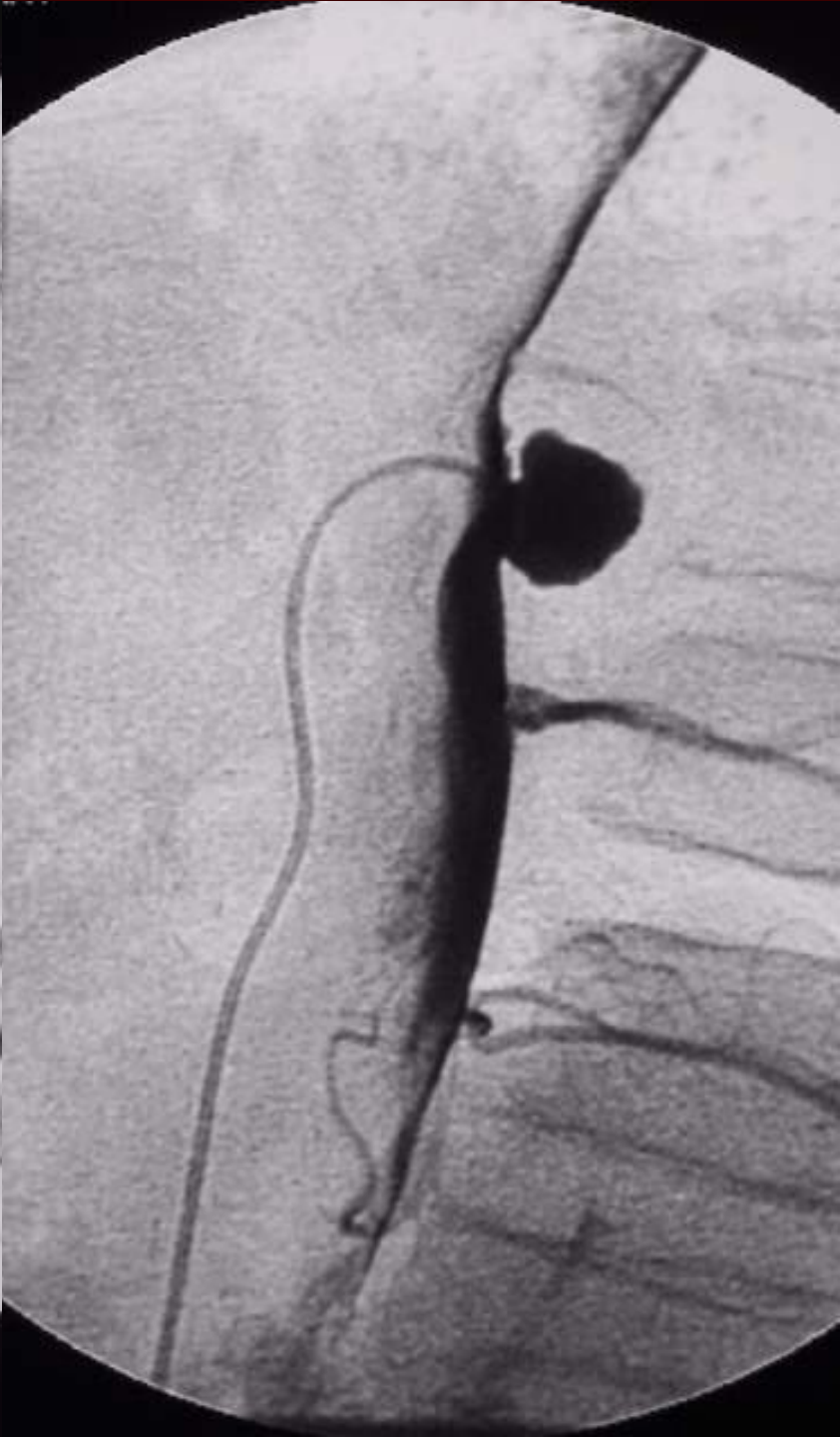
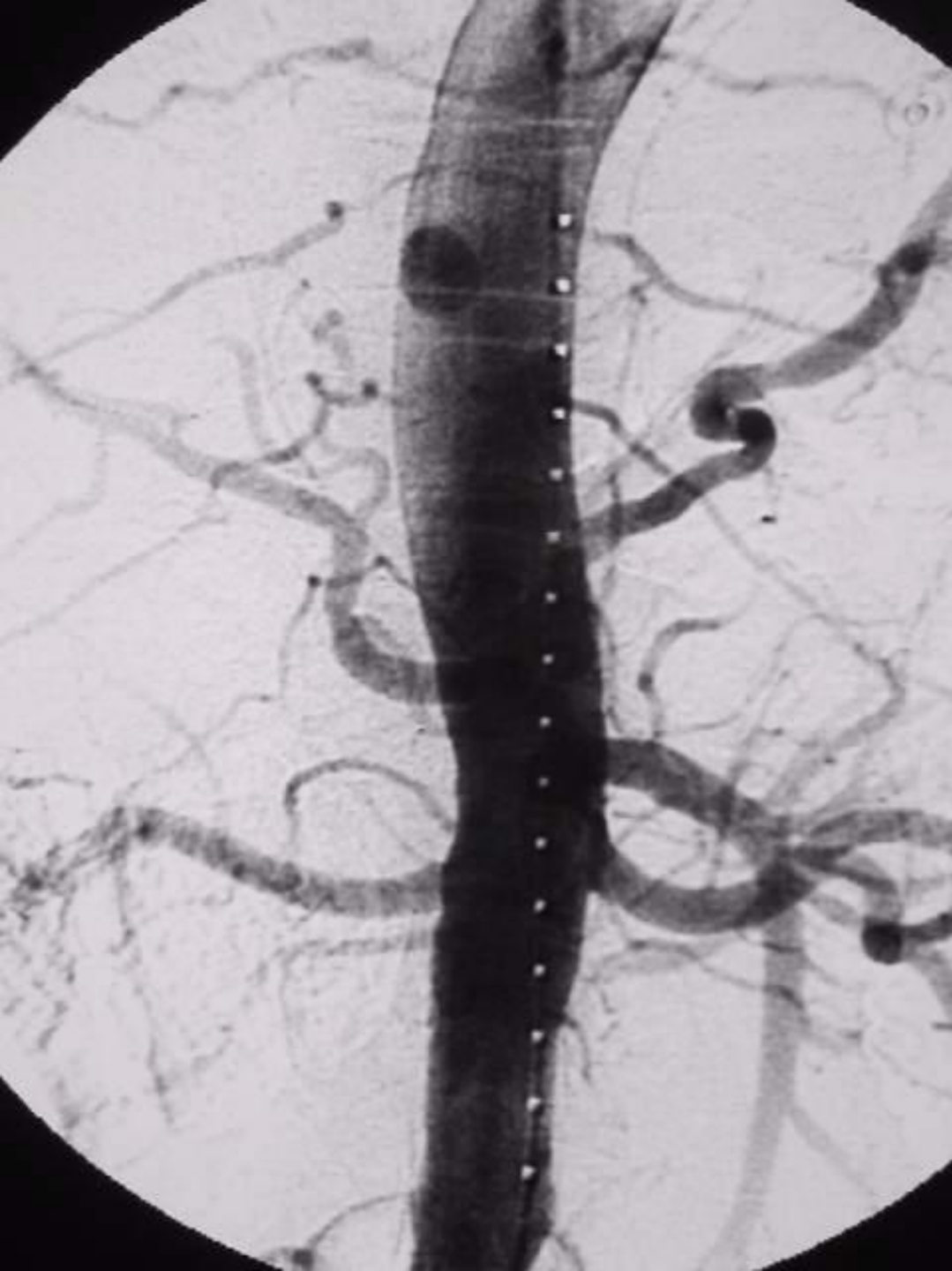
**ROTURA**



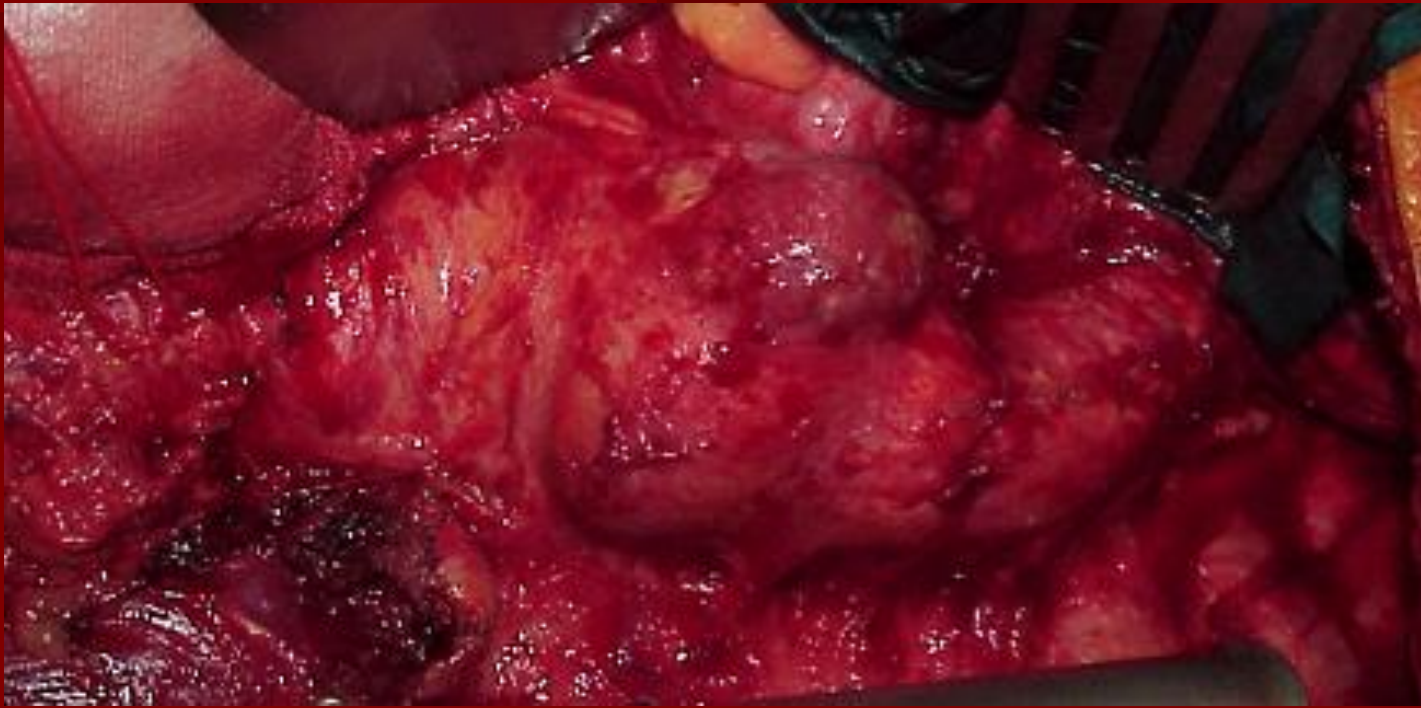
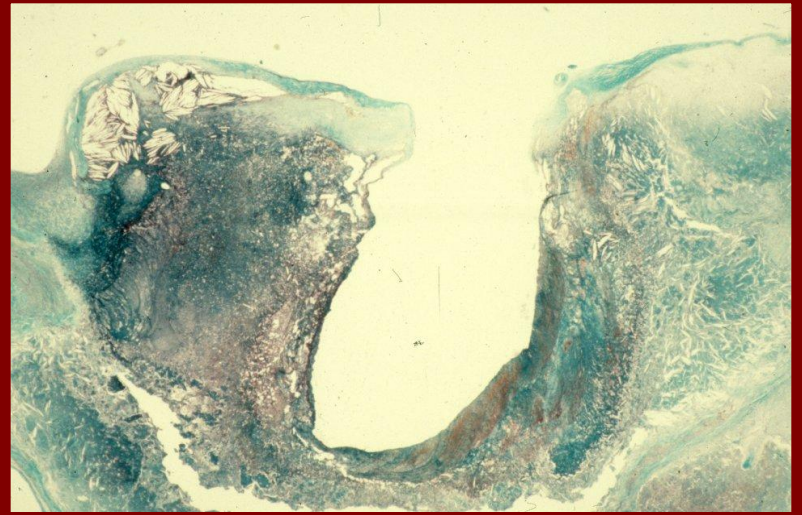
**PSA**



**DISECCIÓN**



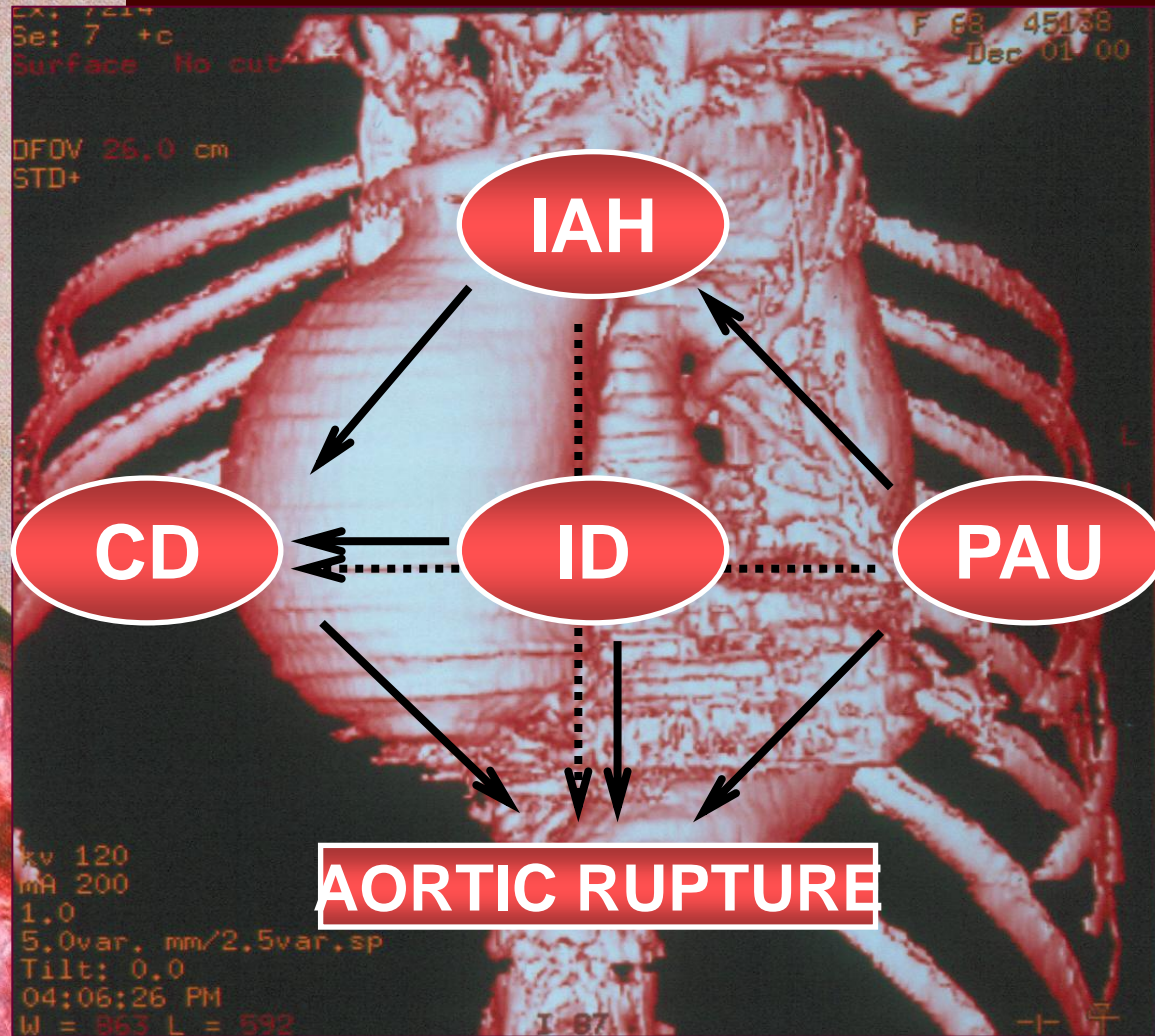
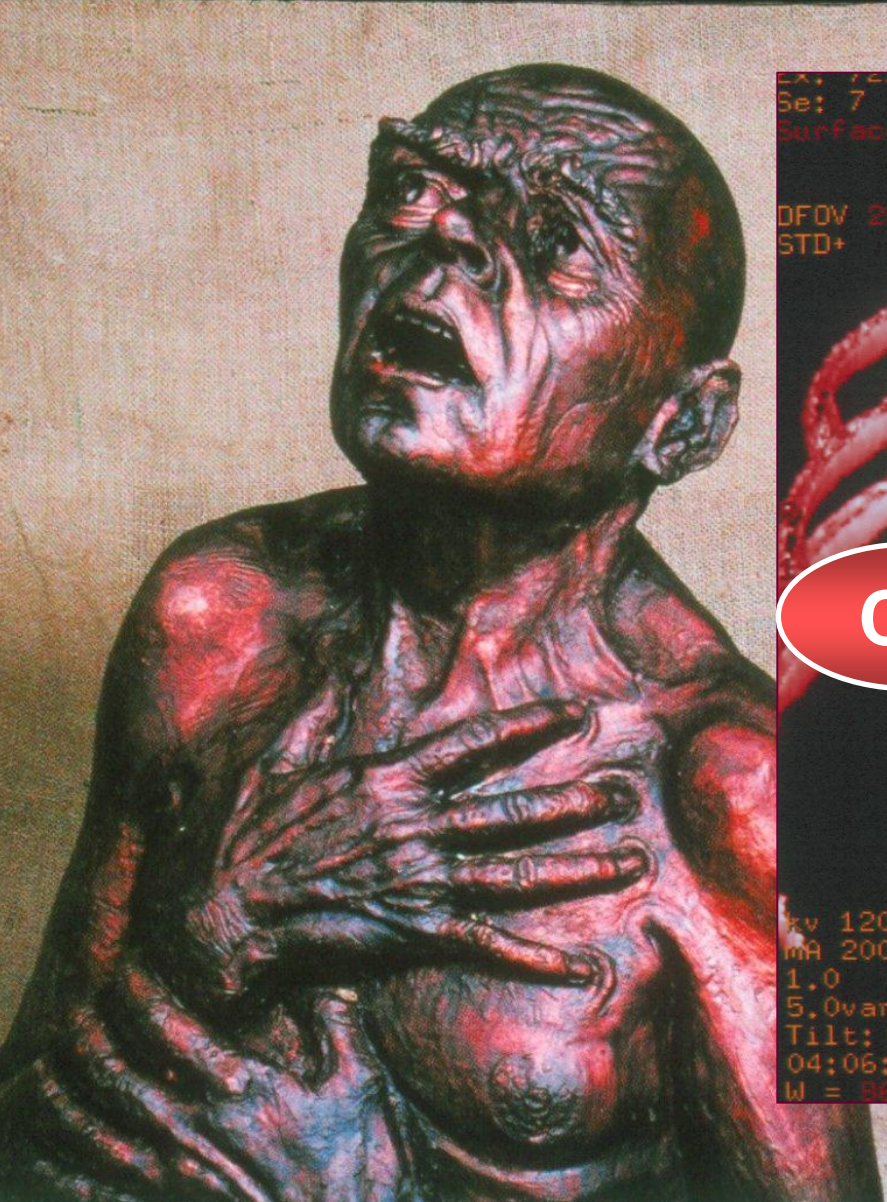
# Penetrating aortic ulcer



# Úlcera penetrante aórtica

- **Formas de presentación clínica:**
  - **Sintomática:**
    - Síndrome aórtico agudo.
    - Embolias.
    - Dolor abdominal.
  - **Asintomática.**

# Síndrome aórtico agudo



# Caso clínico (1-3-2008)

- Mujer de 80 años de edad, **hipertensa**.
- **Dolor súbito** en toda la caja torácica y en el piso abdominal superior, como si se tratara de un latigazo y acompañado de sensación de muerte.
- **Exploración:** PA: 170/80 mmHg, PVY: normal, PC: regular. AC: soplo sistólico de eyección. Abdomen: blando, depresible, sin signos de peritonismo, ligeramente doloroso de forma difusa. Pulsos presentes y simétricos.

# Caso clínico (1-3-2008)

- **ECG:** R/S, normal.
- **Rx tórax:** botón aórtico prominente, aorta elongada y sin signos de ICI.
- **Analítica:** Hb: 13,8; leucocitos: 9600; Cr: 0,97; CPK: 40; CPK-MB: 1,27; Tr. I: 0,2; plaquetas: 115000; GOT: 146; GPT: 127; LDH: 2870; Dímero D: 2251; Amilasa: 72; pH: 7,5; pO<sub>2</sub>: 70; pCO<sub>2</sub>: 29.



# UPA asintomático

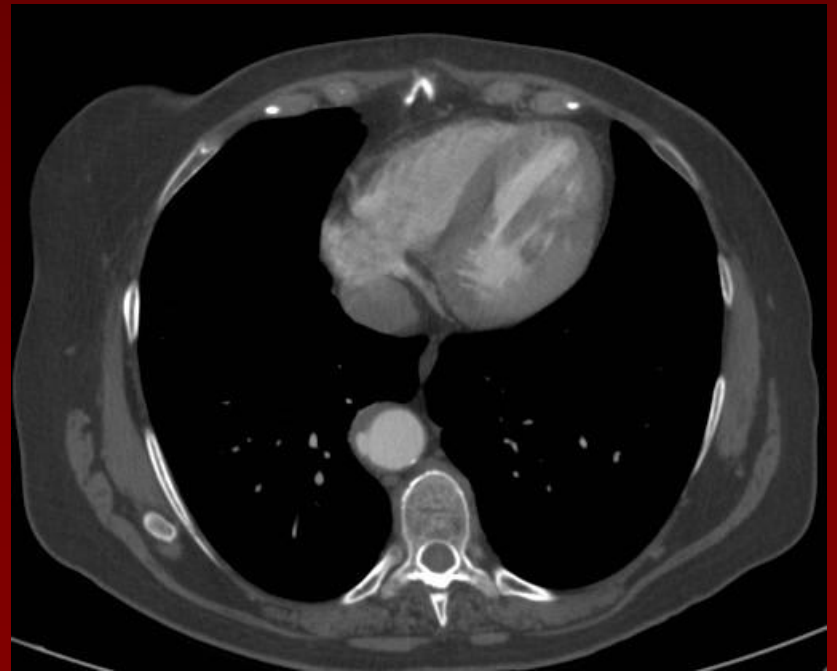
- Paciente de 58 años con hipertensión severa conocida desde hace 3 años. Ingresa en otro centro por una crisis hipertensiva (cefalea intensa, alteraciones de la visión, 240/140 mmHg).
- Exploración: masa abdominal.
- TC toracoabdominal:
  - Teratoma.
  - Disección aórtica.



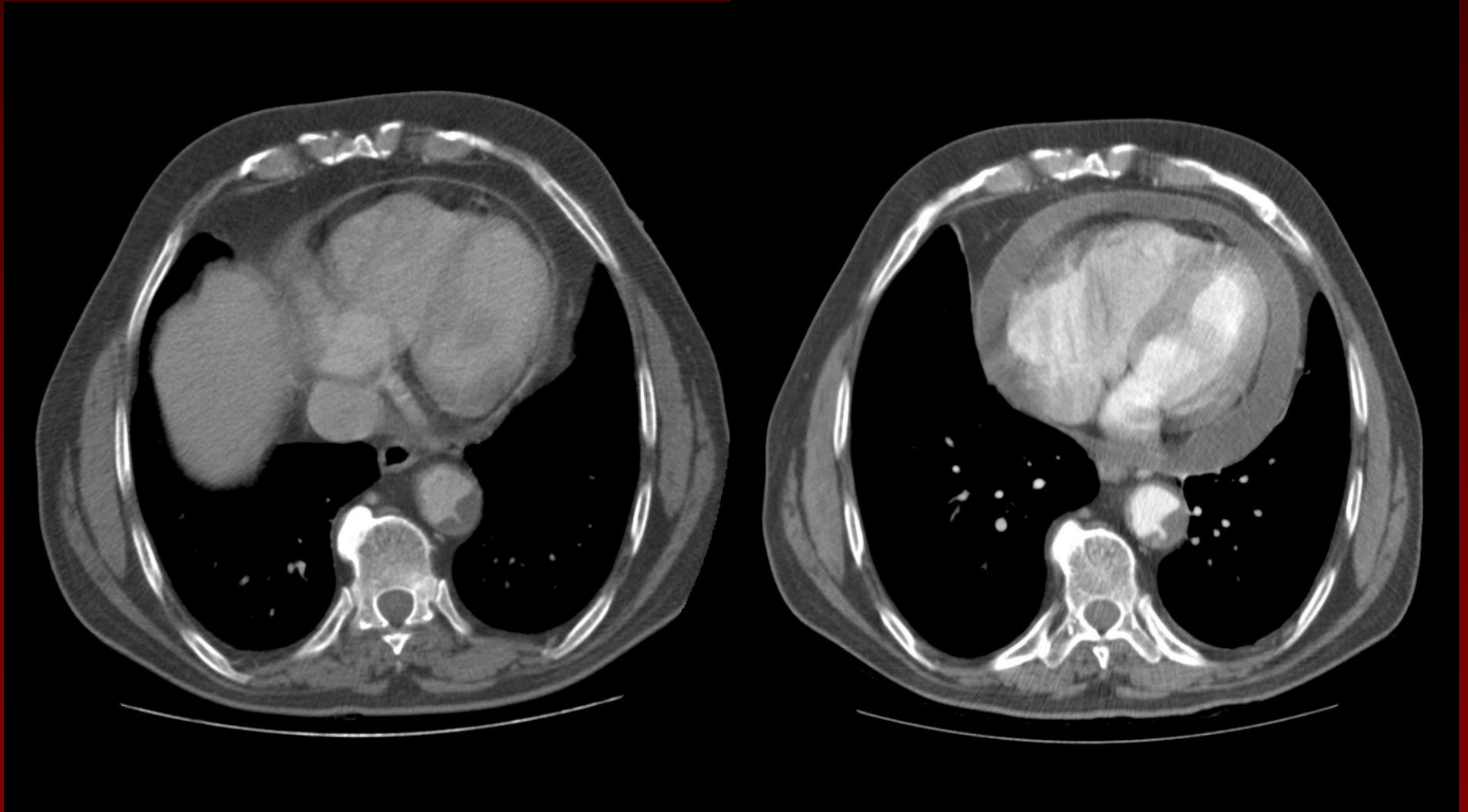


# UPA asintomático

- Varón de 76 años de edad, hipertenso, dislipémico y con un AAA en seguimiento.
- Asintomático.
- En un TC toraco-abdominal se detectan dos lesiones compatibles con UPA.

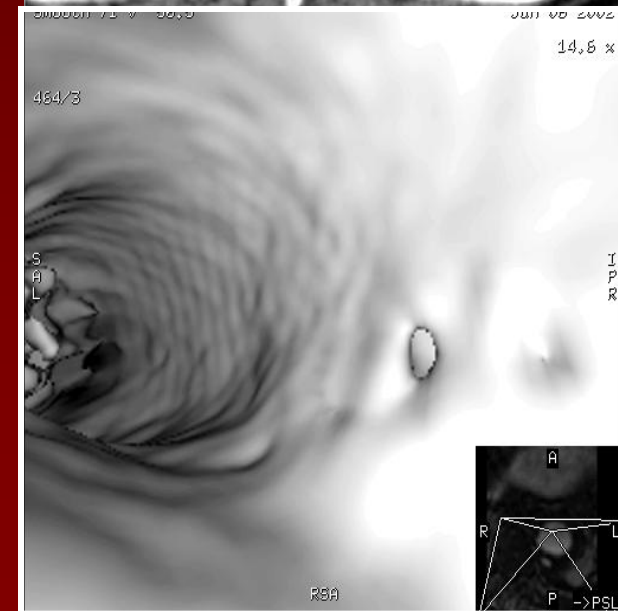


# Diagnóstico diferencial



# “Ulcer-like” projections

- Eyer WR, Clark MD. Radiology 1965.
- “localized blood-filled pouch protruding from the true lumen into the thrombosed false lumen of the aorta”.
- Correlación anatómica: No
  - Desgarro intimal.
  - UPA.



# “Ulcerlike projections”

- Detección al ingreso o durante la evolución del paciente con HIA.
  - A lo largo de toda la aorta. Ausencia de calcificación y márgenes finos.
  - Frecuencia: 15-33% de los pacientes.
  - HIA: 15 d - 2 m tras el evento clínico agudo.
  - Complicaciones: 31% (11/36)<sup>1</sup> – 70% (12/17)<sup>3</sup> (1-6 meses). Predictor de progresión: aorta asc. y arco.
    - Aumento de tamaño.
    - Disección.
    - Rotura.
  - **SEGUIMIENTO: HIA +ULP: peor pronóstico.**

*1. Bosma MS, et al. AJR 2009, 2. Jang YM, et al. Clin Radiol 2008, 3. Sueyoshi E, et al. Radiology 2002. 4. Quint LE, et al. Radiology 2001.*

# Úlcera penetrante aórtica

- **Dificultades diagnósticas.**
- **Historia natural UPA: Desconocida.**
  - *Quint LE, et al. Radiology 2001.*
  - *Bosma MS, et al. AJR 2009.*
- **Heterogeneidad.**
- **Perfil clínico del paciente:** edad avanzada, comorbilidad.
- **Rotura aórtica:** complicación más temida, alta mortalidad.



# Objetivos del tratamiento

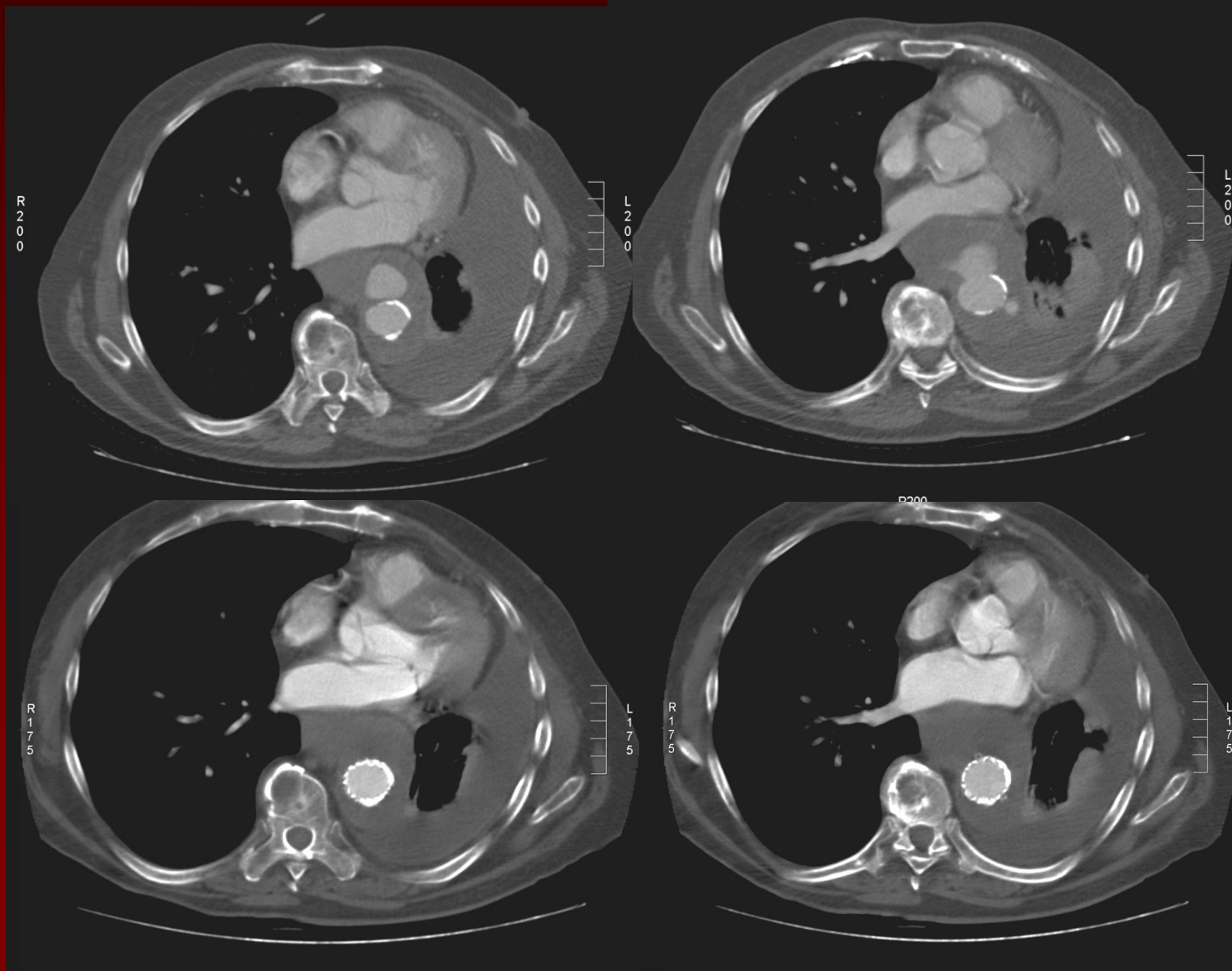


**Seguimiento estrecho con pruebas de imagen**

# ÚLCERA PENETRANTE AÓRTICA



# UPA-ROTURA CONTENIDA ENDOPRÓTESIS



# Predict. de progresión. UPA-ULP

- Afectación de la aorta ascendente:
  - Complicaciones frecuentes.
  - *Coady MA, et al. J Vasc Surg 1998.*
  - *Sueyoshi E, et al. Radiology 2002.*
  - *Bosma MS, et al. AJR 2009.*
  - *Jang YM, et al. Clin Radiol 2008.*
  - *Sueyoshi E, et al. Radiology 2002.*
  - *Quint LE, et al. Radiology 2001.*

# Predict. de progresión. UPA-ULP

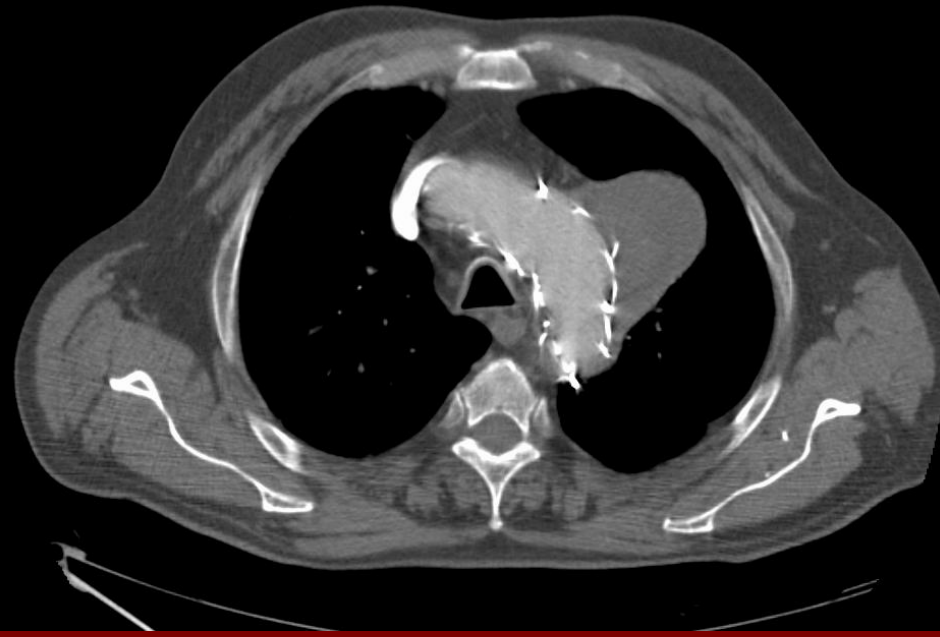
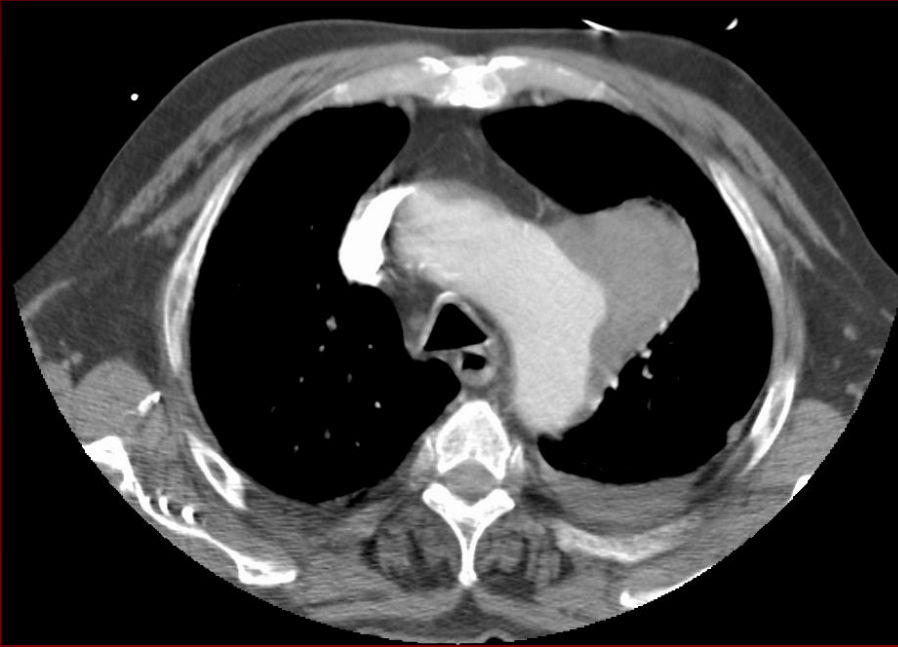
- Afectación de aorta descendente:
  - Diámetro aórtico  $> 50$  mm.
  - Dilatación aórtica progresiva.
  - Incremento del HIA.
  - Gran erosión intimal (diámetro del cráter).
  - Asimetría.
  - Derrame pleural importante o progresivo, hemopericardio, hemomediastino.
  - Dolor persistente o recurrente
  - Inestabilidad hemodinámica.



# Predictores de progresión de la UPA

- Afectación de la aorta ascendente.
- Diámetro aórtico  $> 50$  mm.
- Dilatación aórtica progresiva.
- Gran erosión intimal (diámetro del cráter).
- Asimetría.
- Derrame pleural importante o progresivo.
- Dolor persistente / inestabilidad clínica.

# ÚLCERA PENETRANTE AÓRTICA ENDOPRÓTESIS





# ENDOPRÓTESIS - DISECCIÓN

## CUESTIONES SIN RESOLVER

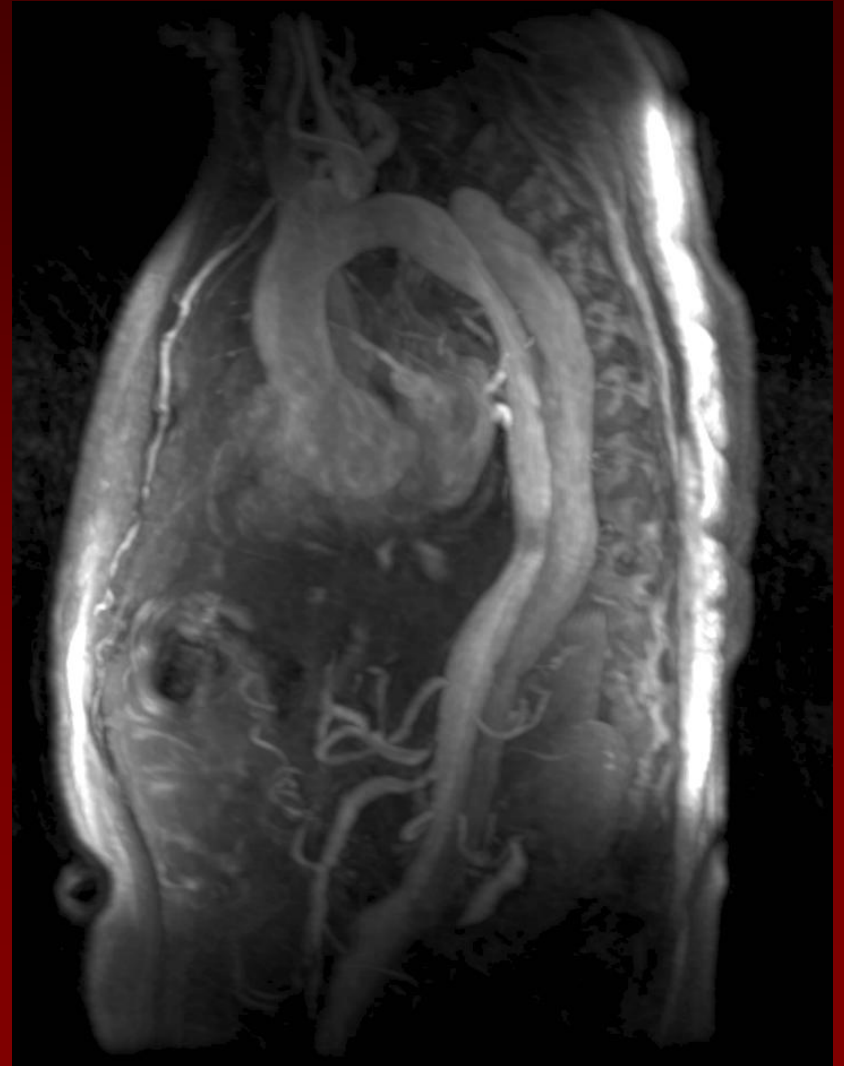
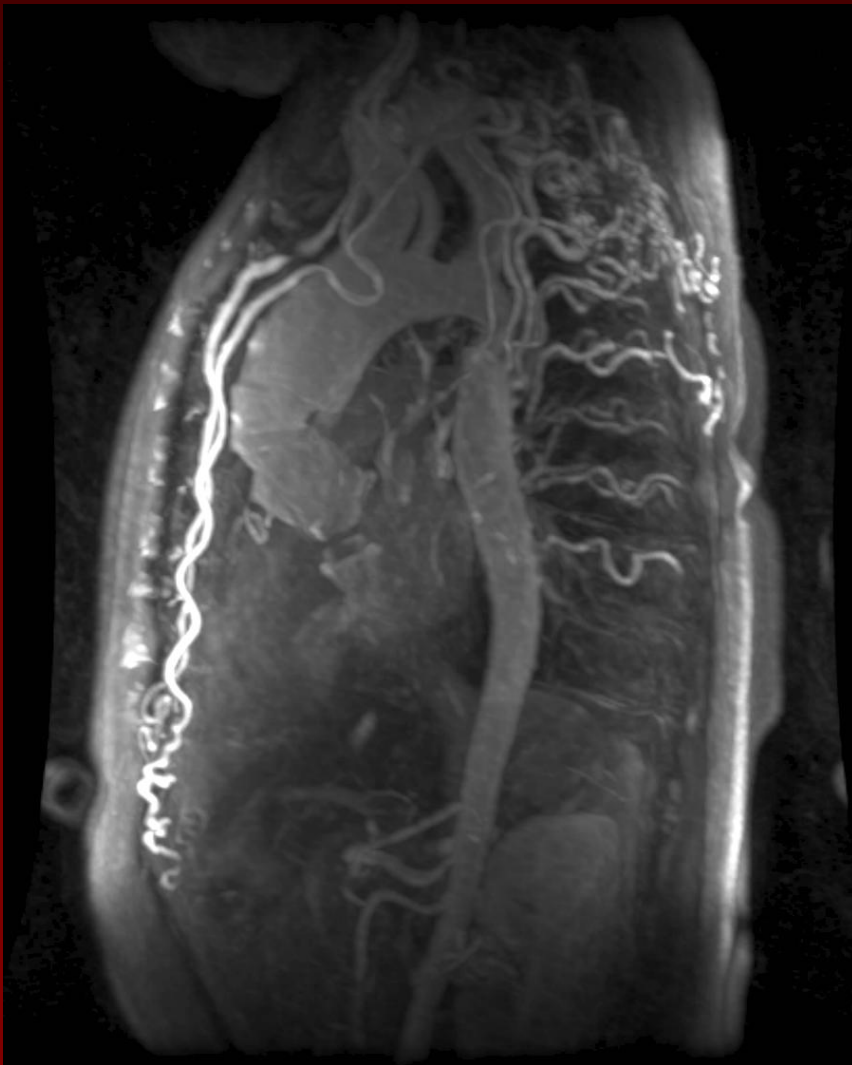
- ¿Cuál es el candidato ideal?
- ¿Es suficiente con sellar la puerta de entrada?
- ¿Es conveniente utilizar stents adicionales con objeto de expandir la LV?
- ¿Cuál es el momento óptimo para implantar la endoprótesis?
- ¿Es conveniente centralizar esta patología?
- ¿Cuál es la durabilidad del stent endovascular?

# ENDOPRÓTESIS - DISECCIÓN

El éxito técnico es prerequisite para el éxito clínico, pero no son sinónimos:

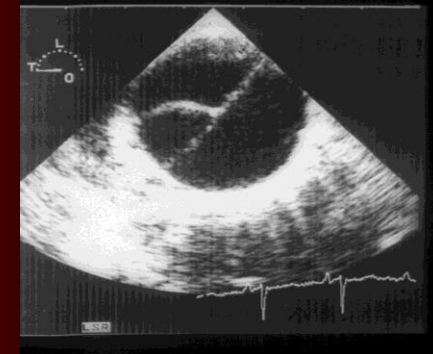
- Estado de la LF: 40%; trombosis LF: 75%
- Complicaciones mayores: 11%
- Reintervención: 12%

# PATOLOGÍA AÓRTICA – RM.

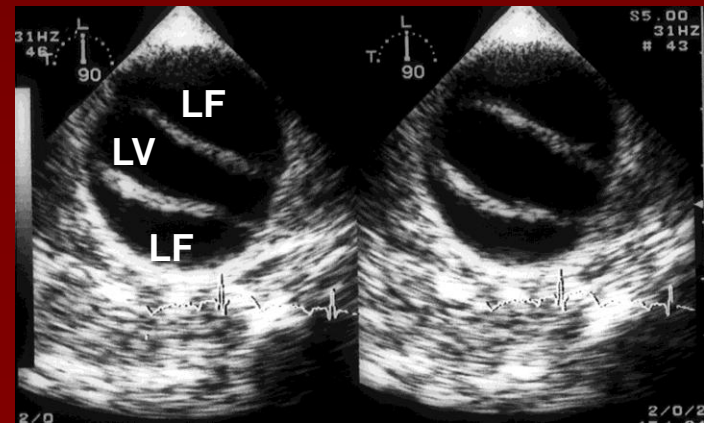




# DIFERENCIACIÓN LV / LF



	LF	LV
Tamaño en sístole	Disminuye	Aumenta
Autocontraste	Sí	No
Trombosis	Sí	No
Tamaño total	Grande	Pequeño
Pared externa	Delgada	Gruesa
Flujo	Lento, de remolino	Laminar, sist. precoz
Signo de la telaraña	Sí	No
Signo de las tres luces	Periférica	Central



# TRATAMIENTO DEL SAA

- Ingreso en la UCC.
- Monitorización continua de la PA (iv.).
- Tratamiento hipotensor. PAs:< 120 mmHg.
- Control estricto de la diuresis.
- Betabloqueantes ( $\downarrow dP/dt$ ).
- Control del dolor.

# EVALUACIÓN DIAGNÓSTICA

ETE, CT, RM.

- Puerta de entrada
- Puerta de reentrada
- Puntos de comunicación
- Relación y diámetros de LV/LF
- Relación LV/LF con vasos viscerales y femorales
- Extensión de la disección

# DISECCIÓN TIPO A + B



# Factores determinantes

Afectación de aorta ascendente

Diámetro aórtico > 50 mm

Dilatación progresiva

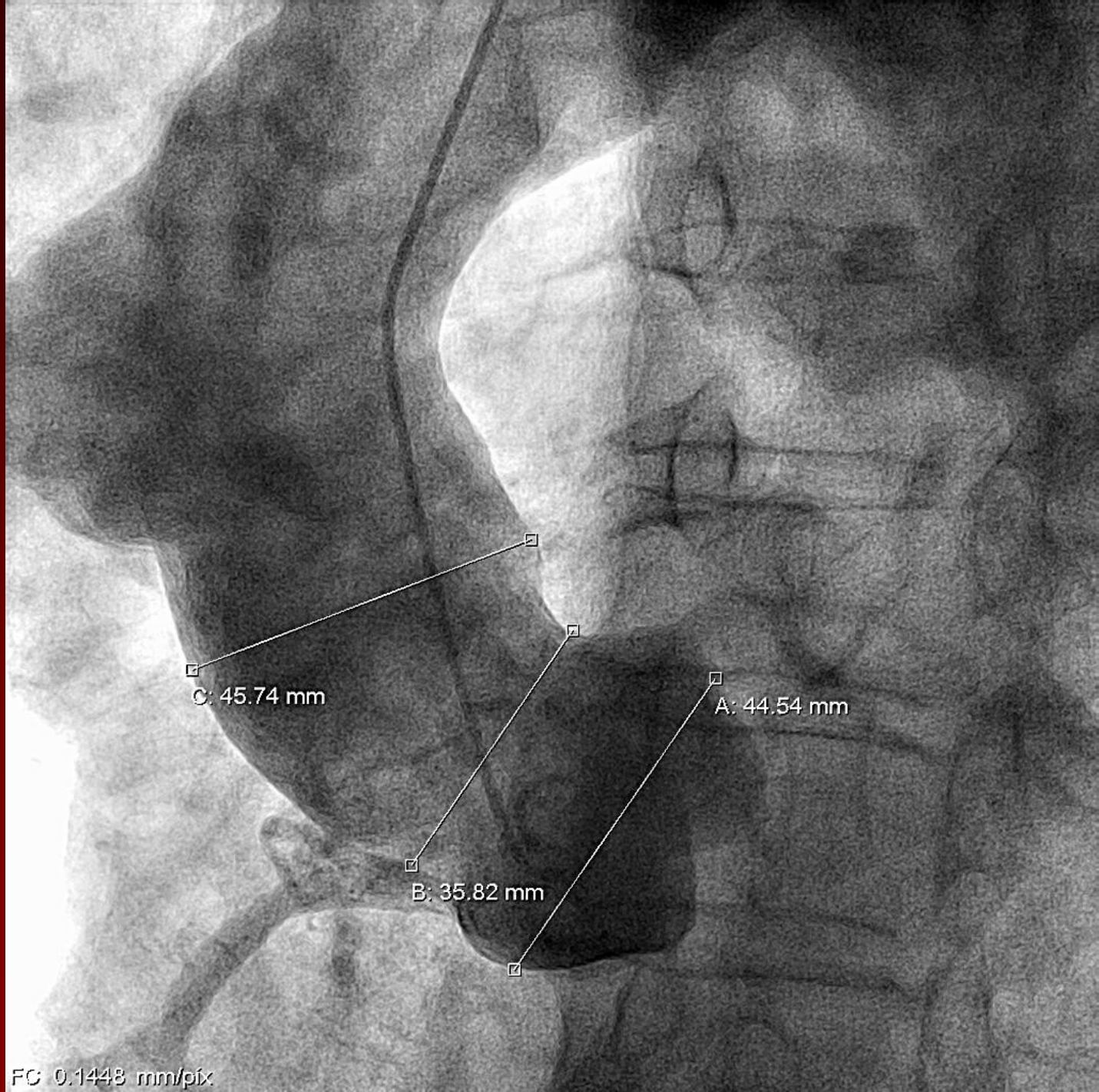
Gran erosión intimal (diámetro cráter)

Asimetría

Derrame pleural importante o progresivo

Dolor persistente/ inestabilidad



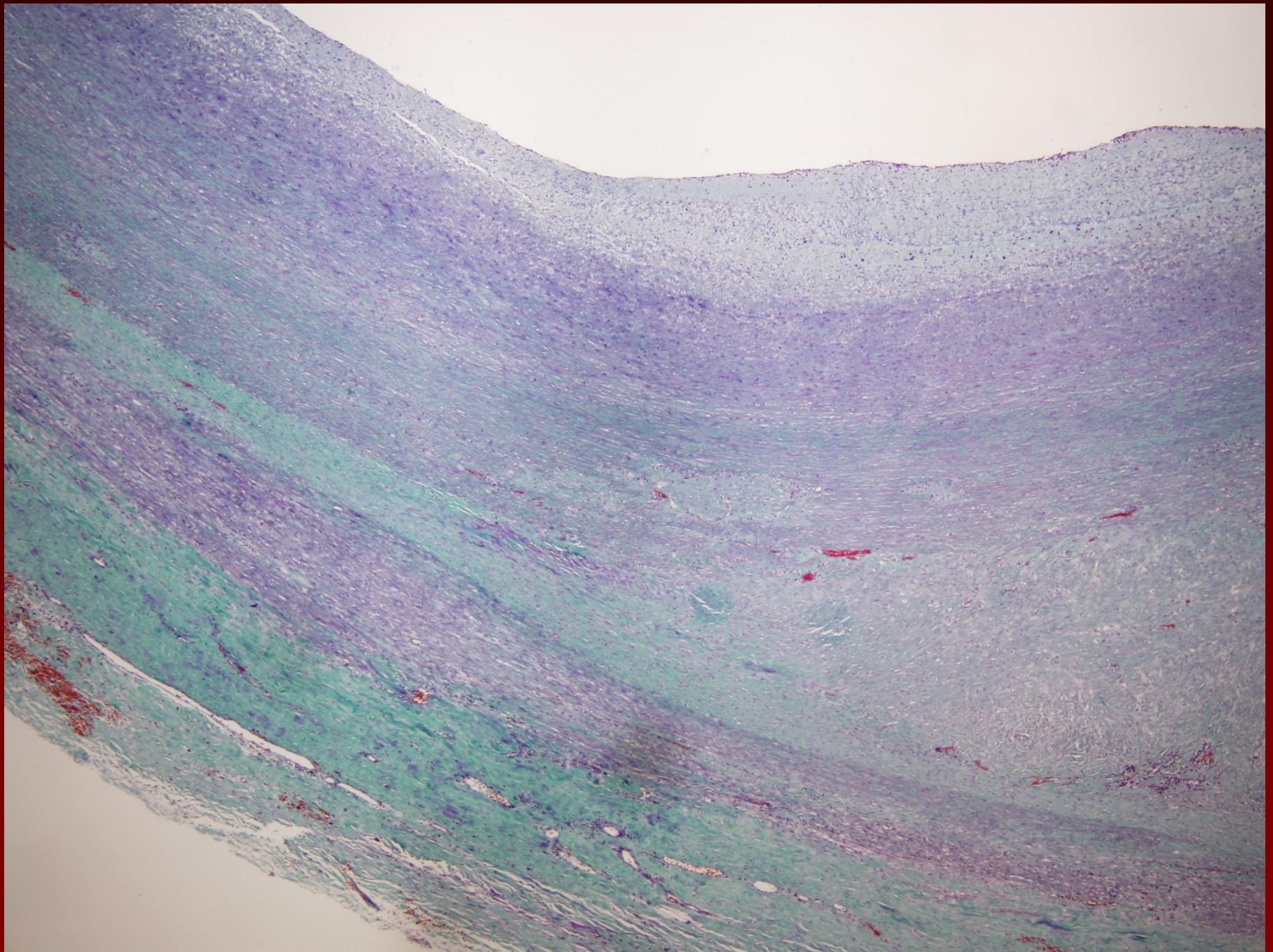


C: 45.74 mm

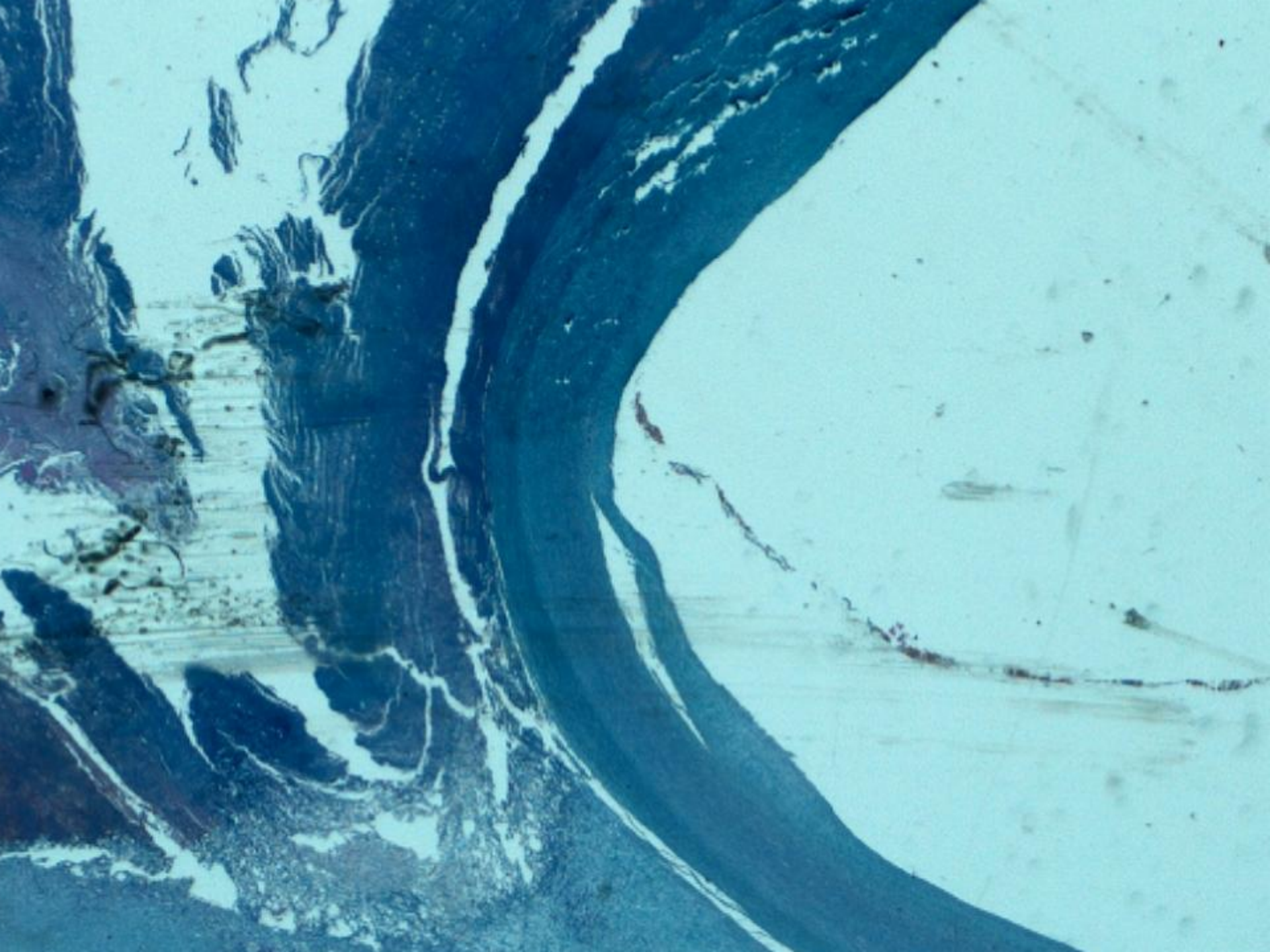
B: 35.82 mm

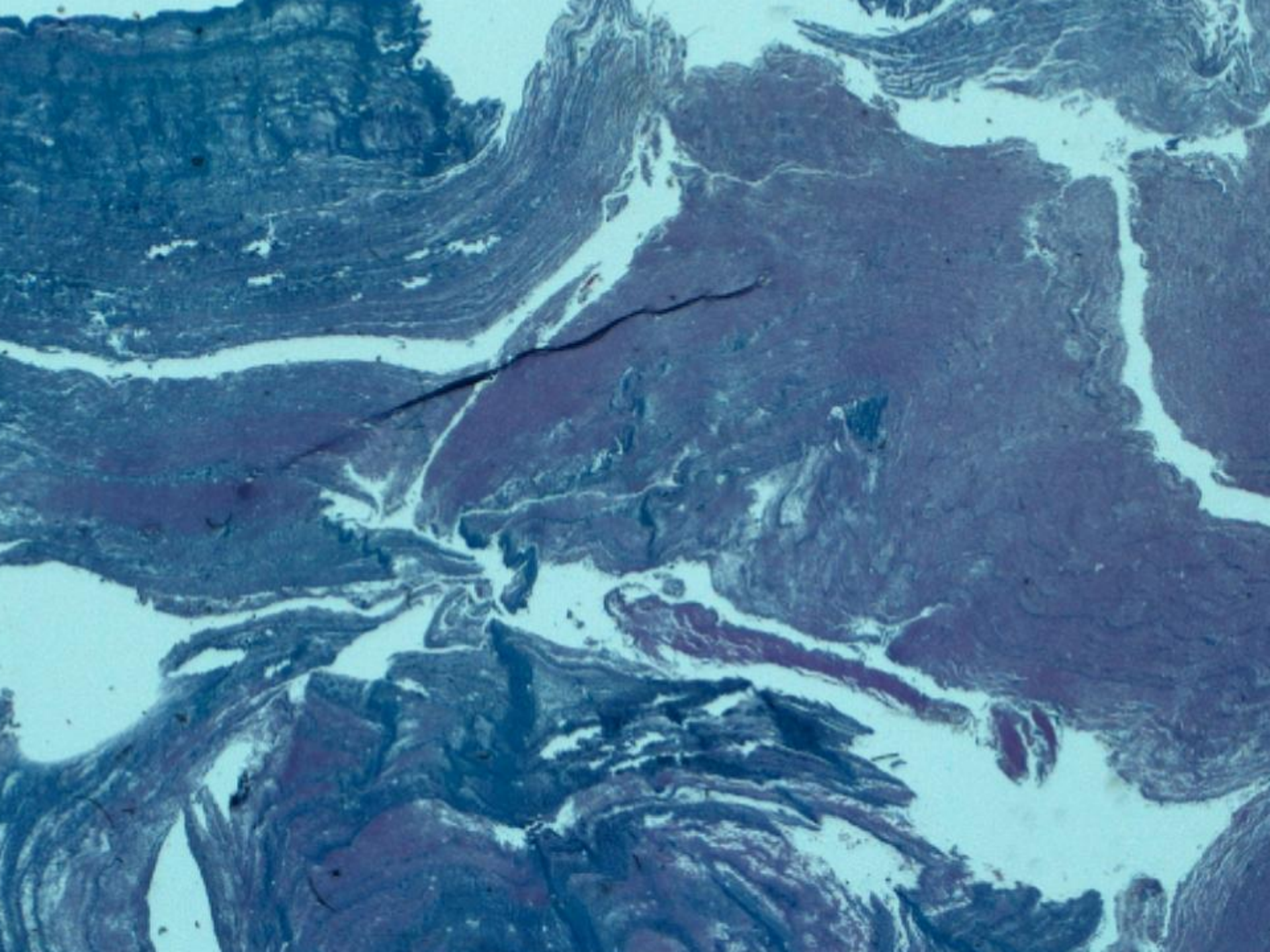
A: 44.54 mm

FC 0.1448 mm/pix

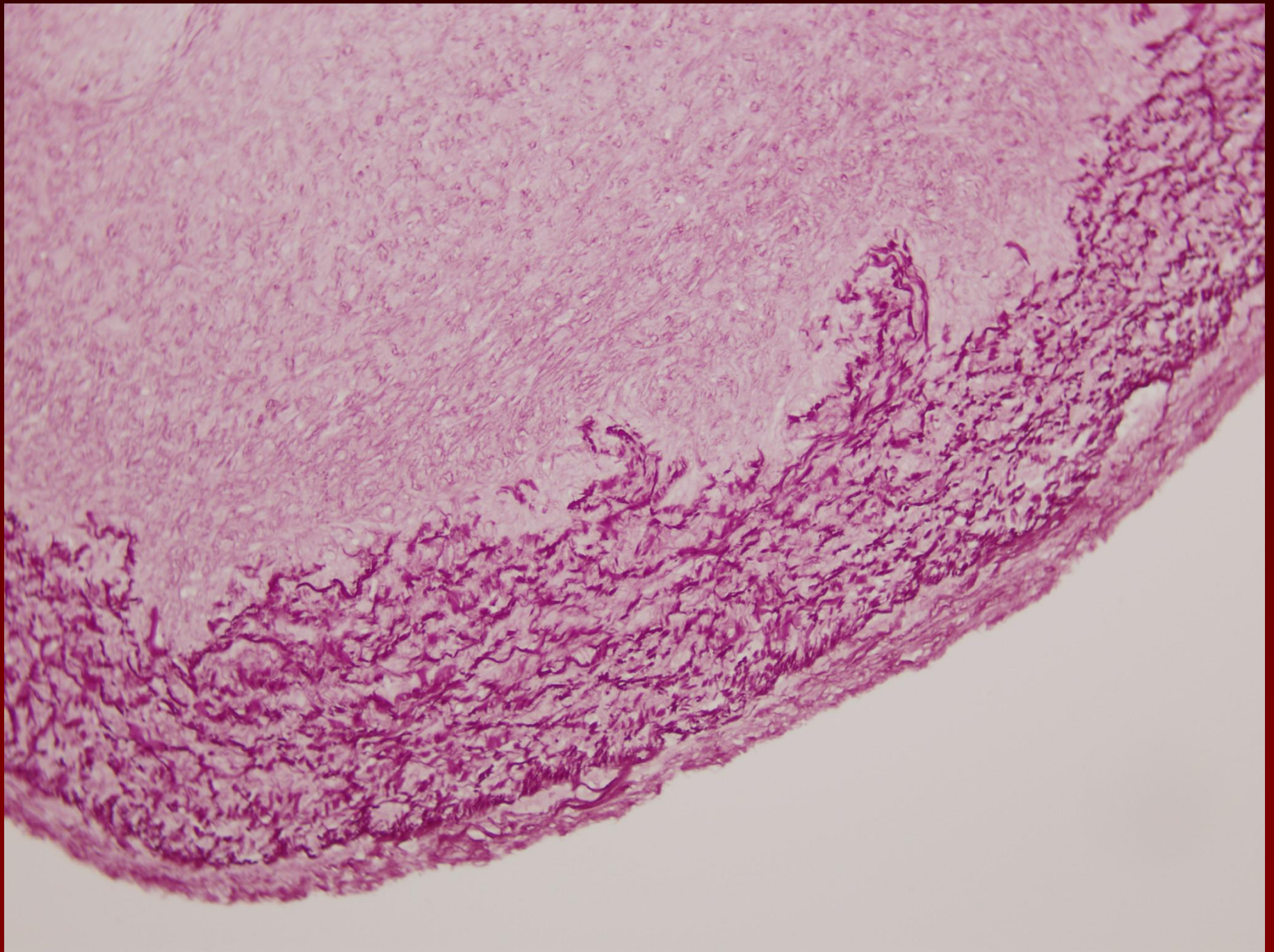


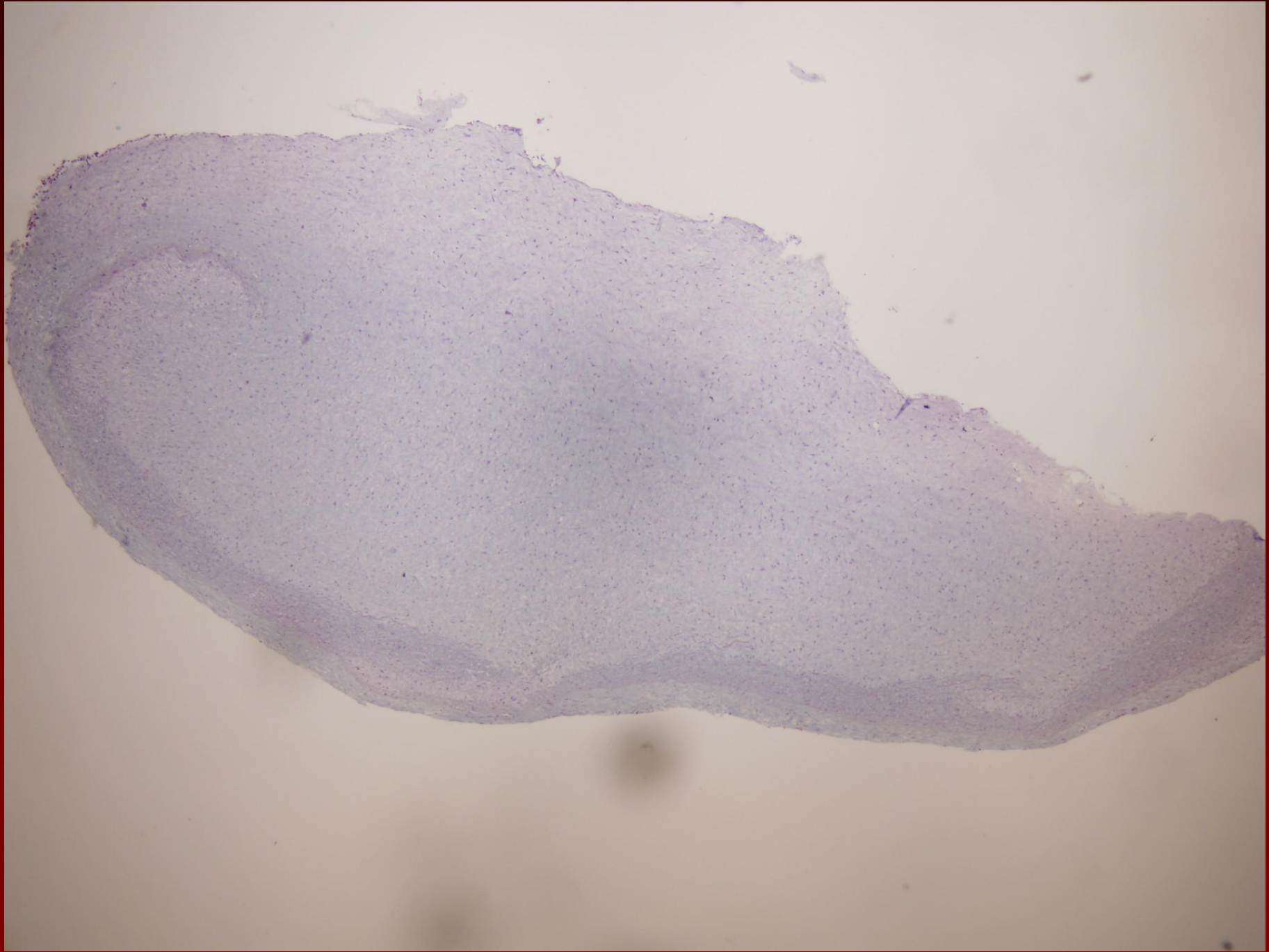




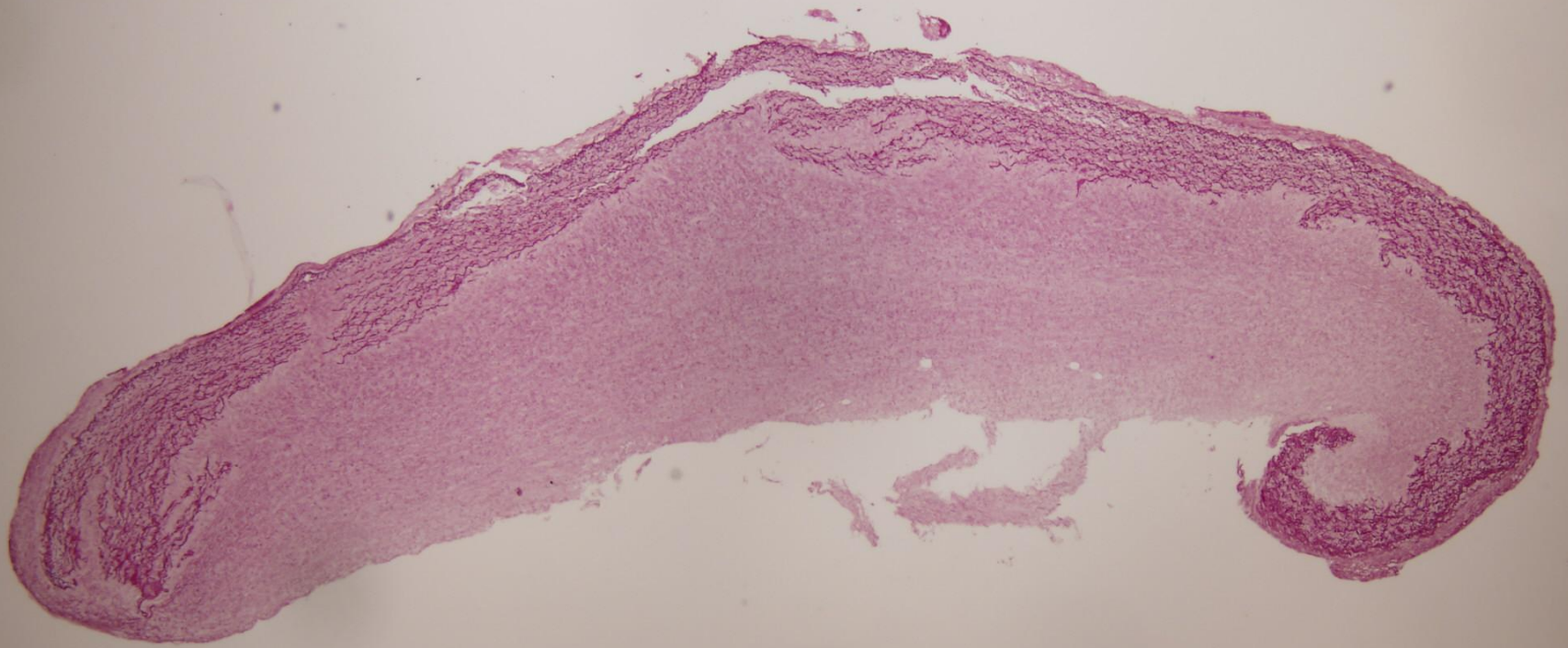


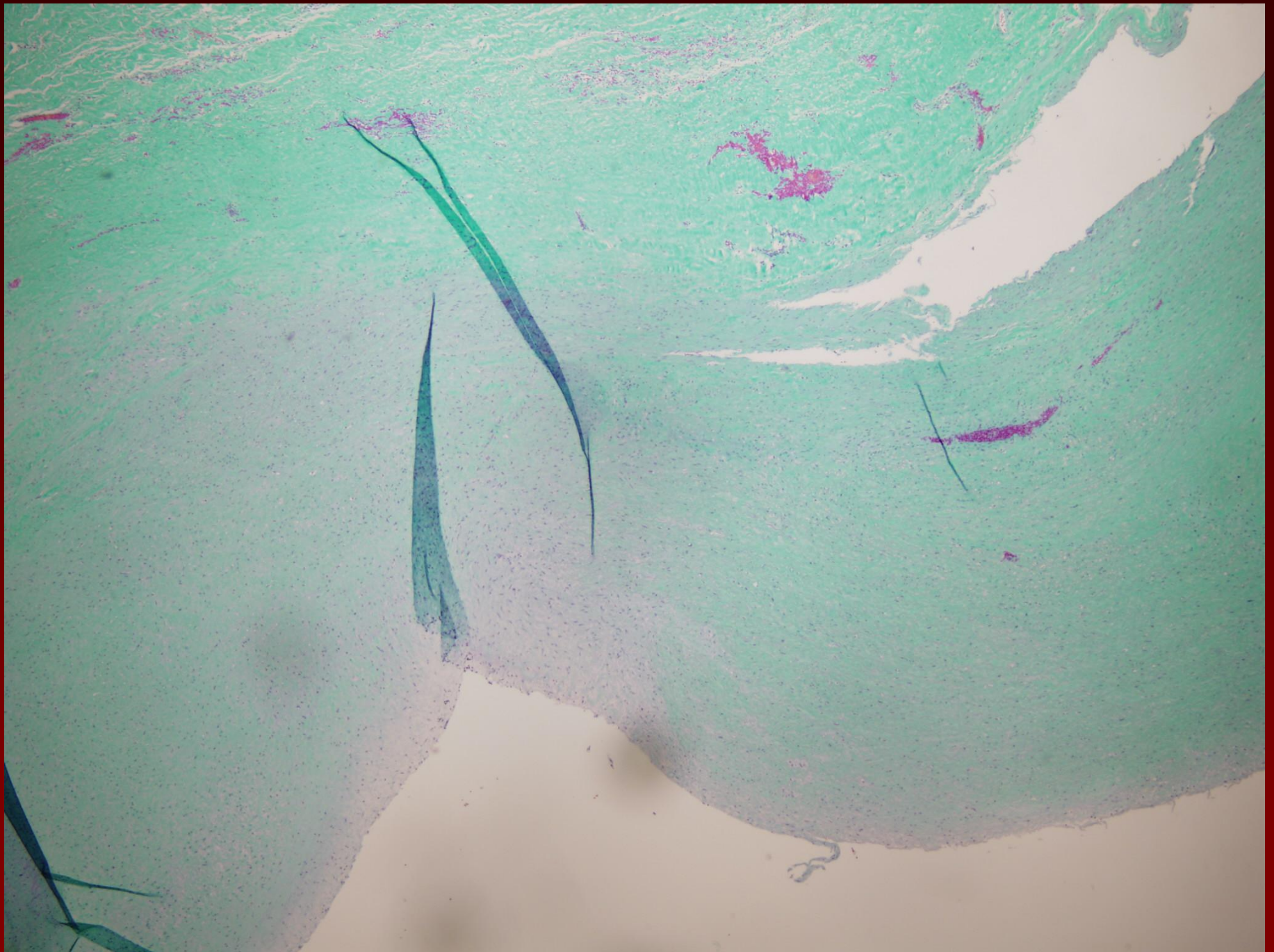


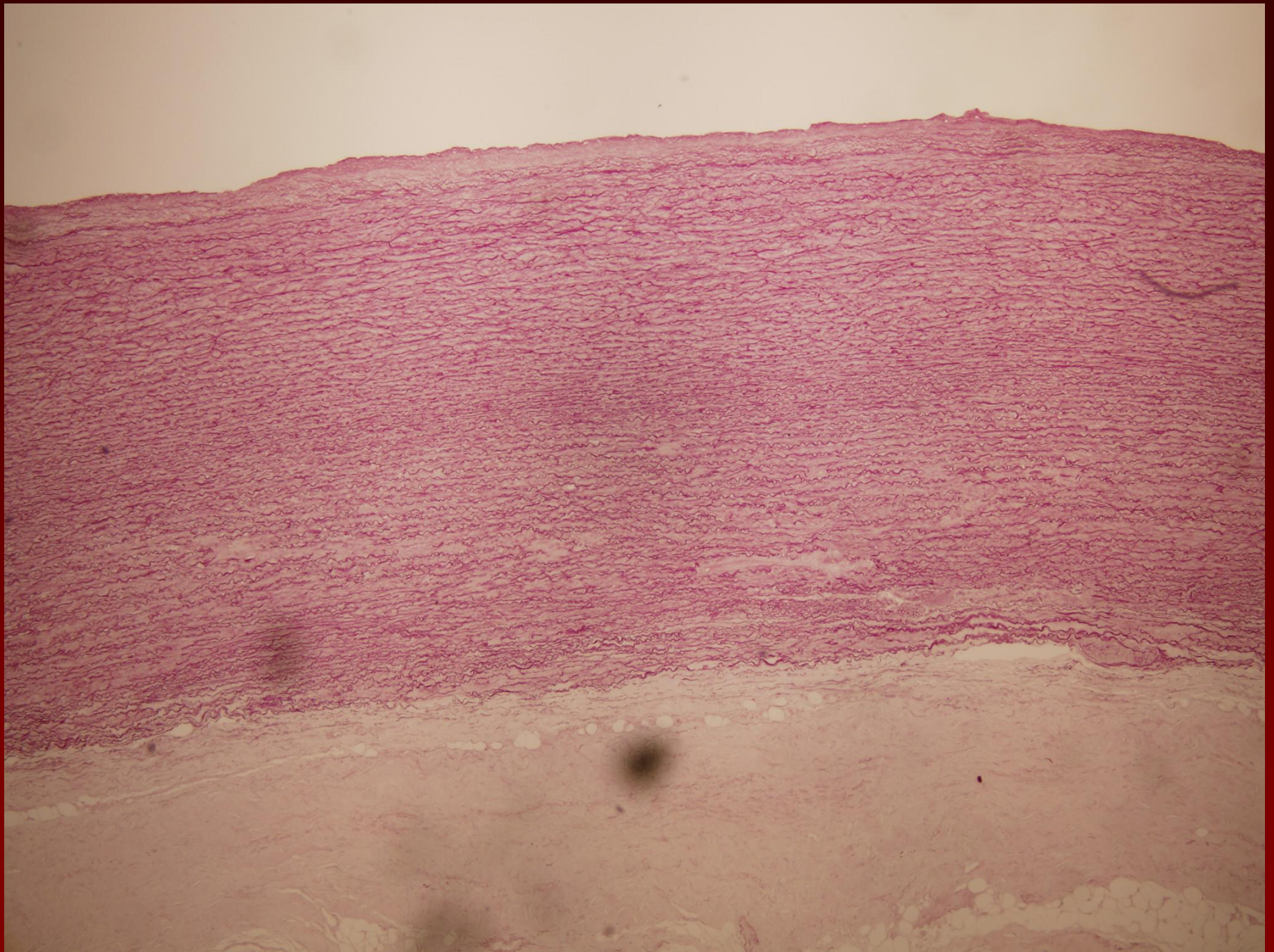


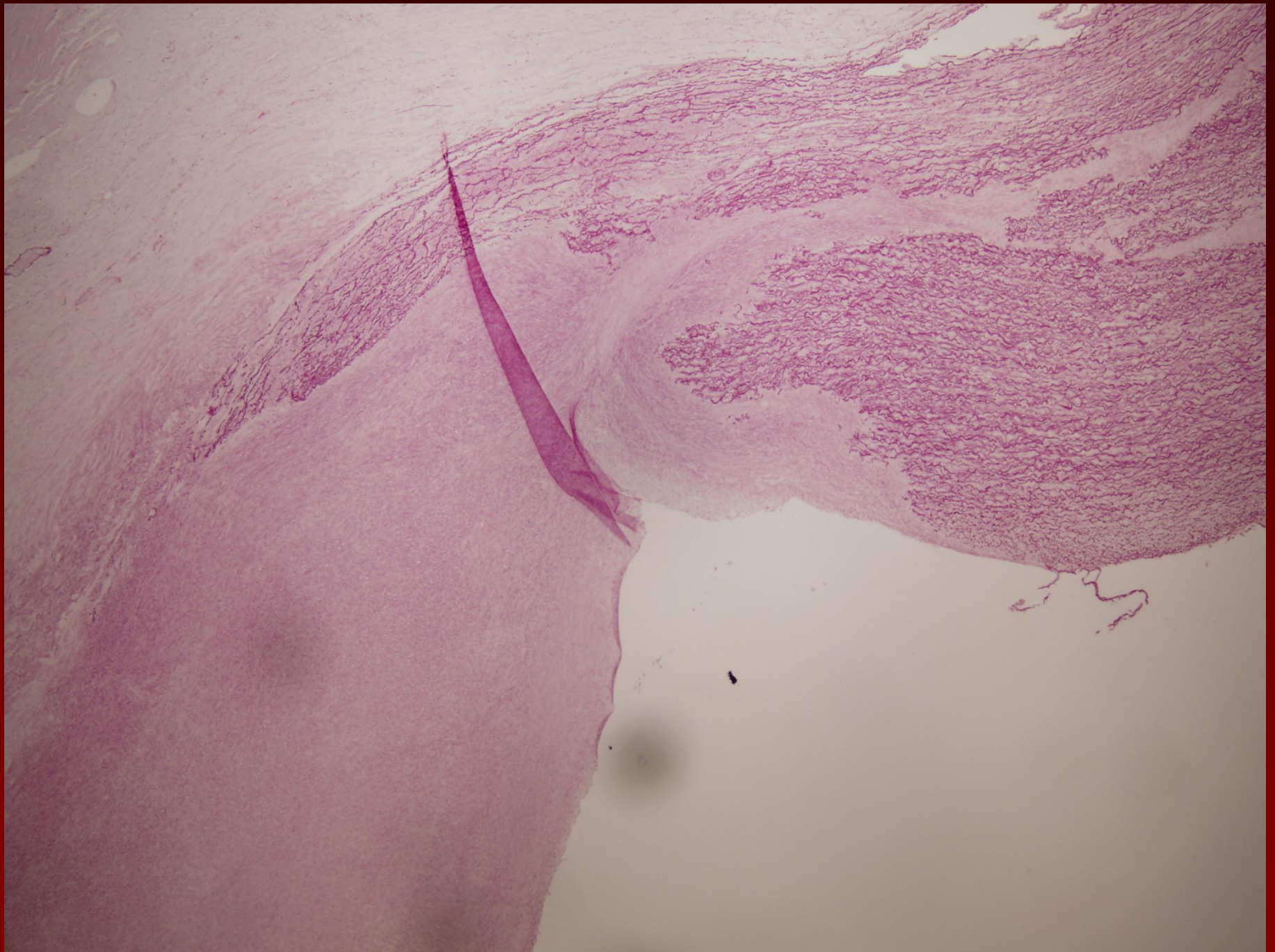


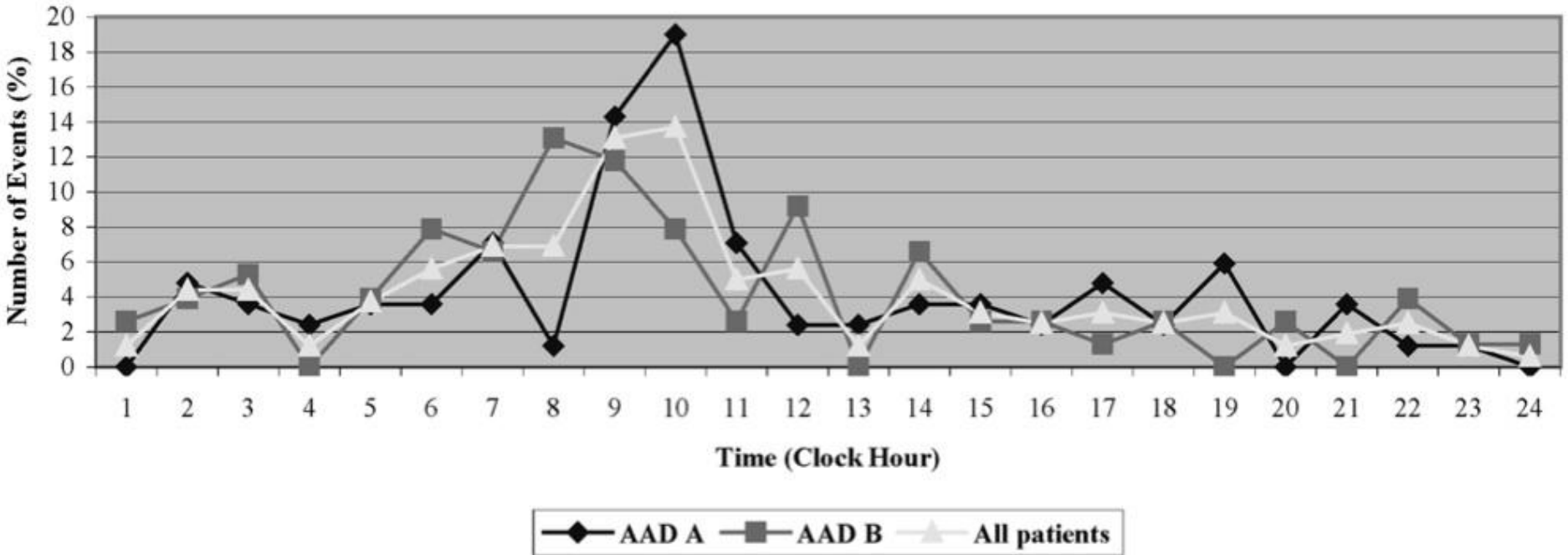




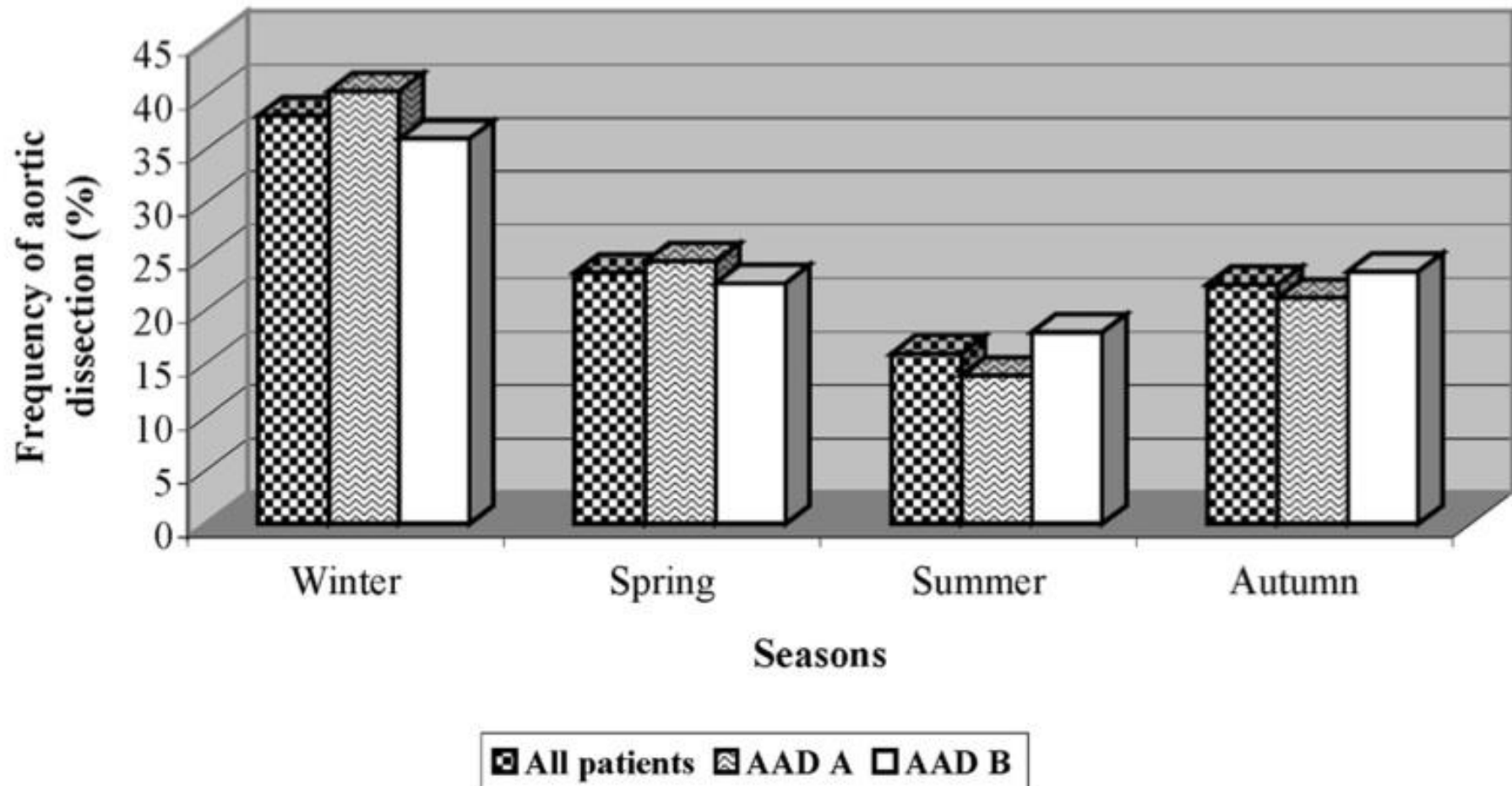








**Figure 2.** Circadian variation of AAD with regard to initial pain onset and to dissection type.



**Figure 4.** Seasonal variation of AAD with regard to type.

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