Περιστατικό Πνευμονικής Υπέρτασης

30 Πανελλήνιο Συνέδριο Πνευμονικής Υπέρτασης

Αθήνα 8/6/2019

Μπούτσικου Μαρία MD, MSc, PhD
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Ιστορικό

- Ασθενής 86 ετών με ιστορικό ΠΥ σε έδαφος θρομβοεμβολικής νόσου
- Ιστορικό ΡΕ
- Παραπομπή στο τμήμα μας το 2011.
- Λοιπό ιστορικό: αρτηριακή υπέρταση υπό αγωγή.
- Φαρμακευτική αγωγή: Lasix, Lopressor, Sintrom

Κατά την πρώτη αξιολόγηση (2011)

- Ολιγο-συμπτωματικός (NHYA I-II)
- Κλινική εξέταση: αυξημένης έντασης-διχασμός S2
- Ήπια περιφερικά οιδήματα

- ΕΡΓΑΣΤΗΡΙΑΚΟΣ ΕΛΕΓΧΟΣ
- Echo:
  - Αμφικολπική διάταση
  - LV κφ ΚΕ 70%
  - IVS 11mm,
  - Διάταση RVEDD 37mm, ήπια RVH, TR 2/4 (Vmax 5.4m/s), RVSP 125mmHg,
  - TAPSE 14mm, Αμφικολπική διάταση, IVC 23mm με μέτρια αναπνευστική διακύμανση.
- ECG: SR 54bpm, IVCD V1-V2
- BNP 47pg/ml, INR 2.6, Hct 48%
- CT chest: παρουσία θρόμβων στους κατώτερους λοβιαίους κλάδους (RLL, LLL) , θρόμβοι και απόφραξη RLL
- 6MWT 421m (SpO2: 93%- >88%)

Πορεία νόσου

Ήπια επιδείνωση της δύσπνοιας.
Διενέργεια RHC και επανεκτίμηση

	RHC
mRA(mmHg)	6
RV (mmHg)	120/14
PA(mmHg)	120/26/60
PCWP(mmHg)	11
Ao(mmHg)	160/80/105
CO (l/min)	5.09
CI (l/min/m^2)	2.8
SVR (WU)	20
PVR(WU)	9.5
TPG (mmHg)	49

Έναρξη ERA
(Ambrisentan 5mg)

Πορεία νόσου (2014)

- NHYA I-II
- 6MWT 490m
- Echo
  - RVSP 105-110mmHg (TR Vmax 5.0m/s),
  - RVEDD 46mm,
  - TAPSE 18mm, LV 39/21mm KE 75%
  - IVC 2.2cm με >50% αναπνευστική διακύμανση
  - TR 2/4

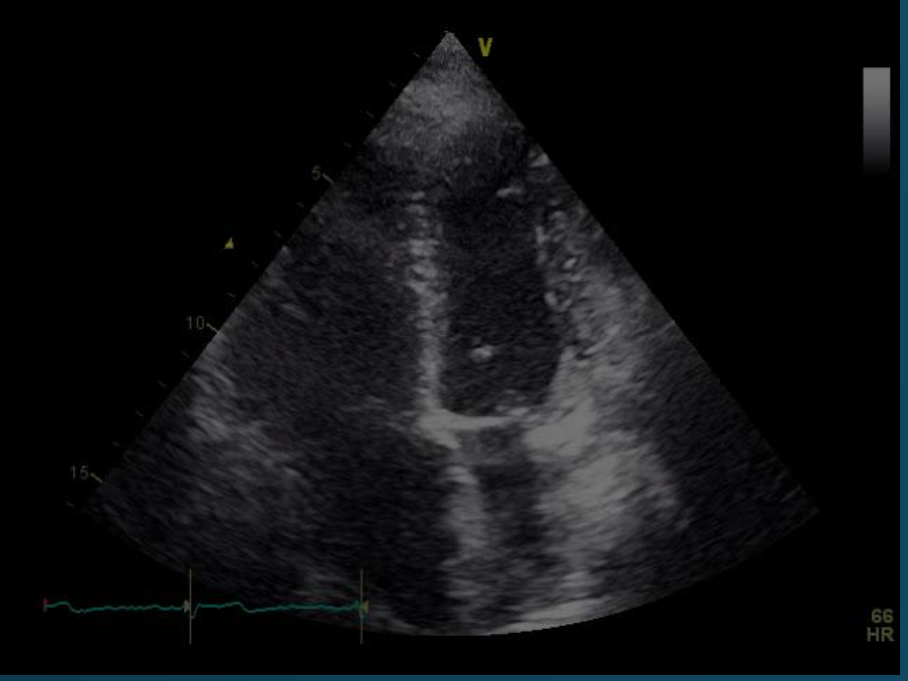
Πορεία νόσου (2014)

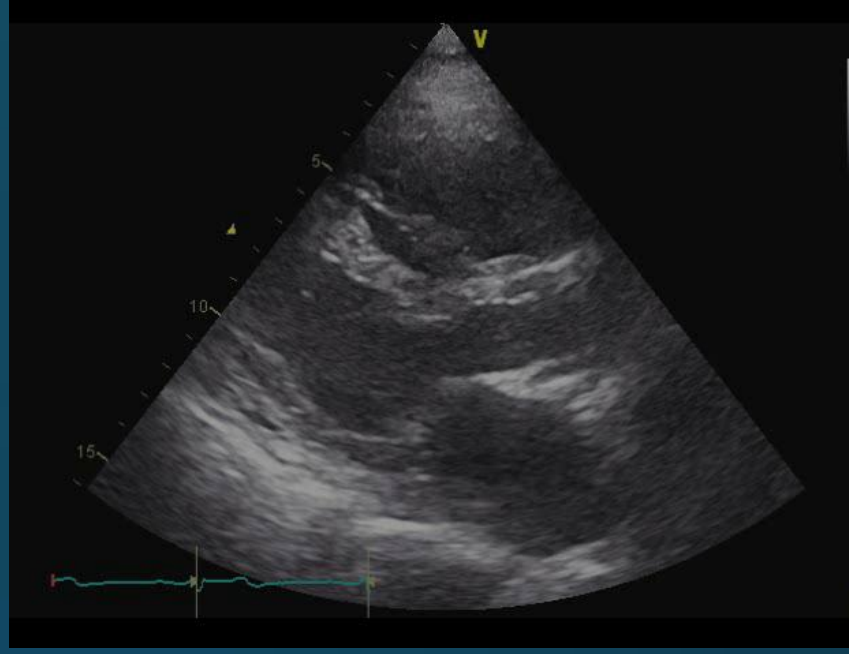
- Σταθερός ως το 11/2018 όπου
- Παρουσιάζει επιδείνωση της δύσπνοιας NHYA II-III

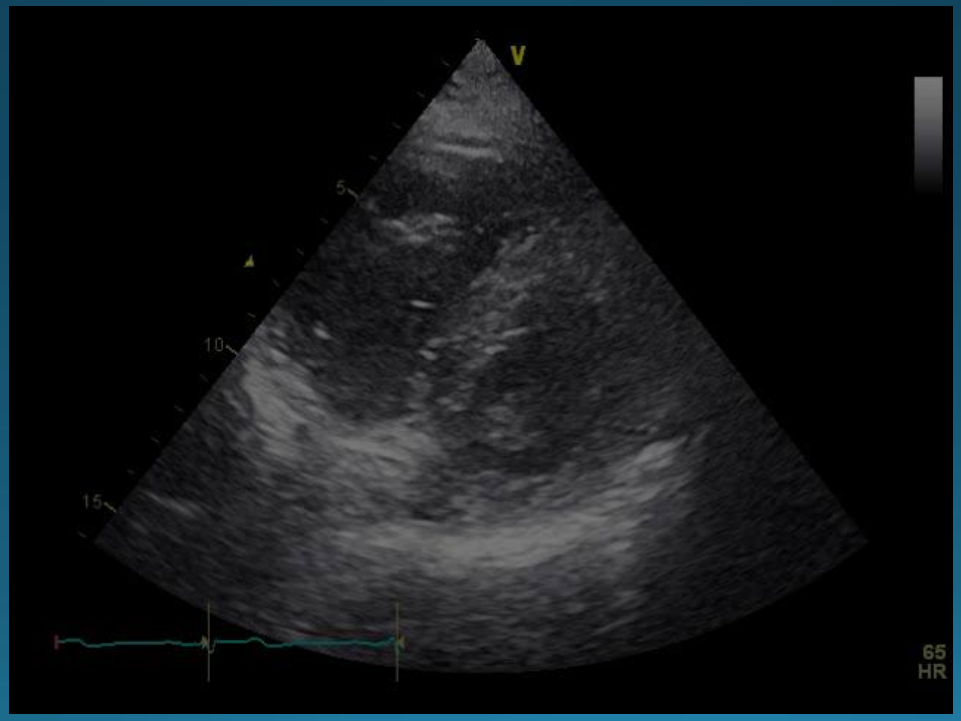
Και επιδείνωση της νεφρικής του λειτουργίας (Cr 1.7mg/dl)

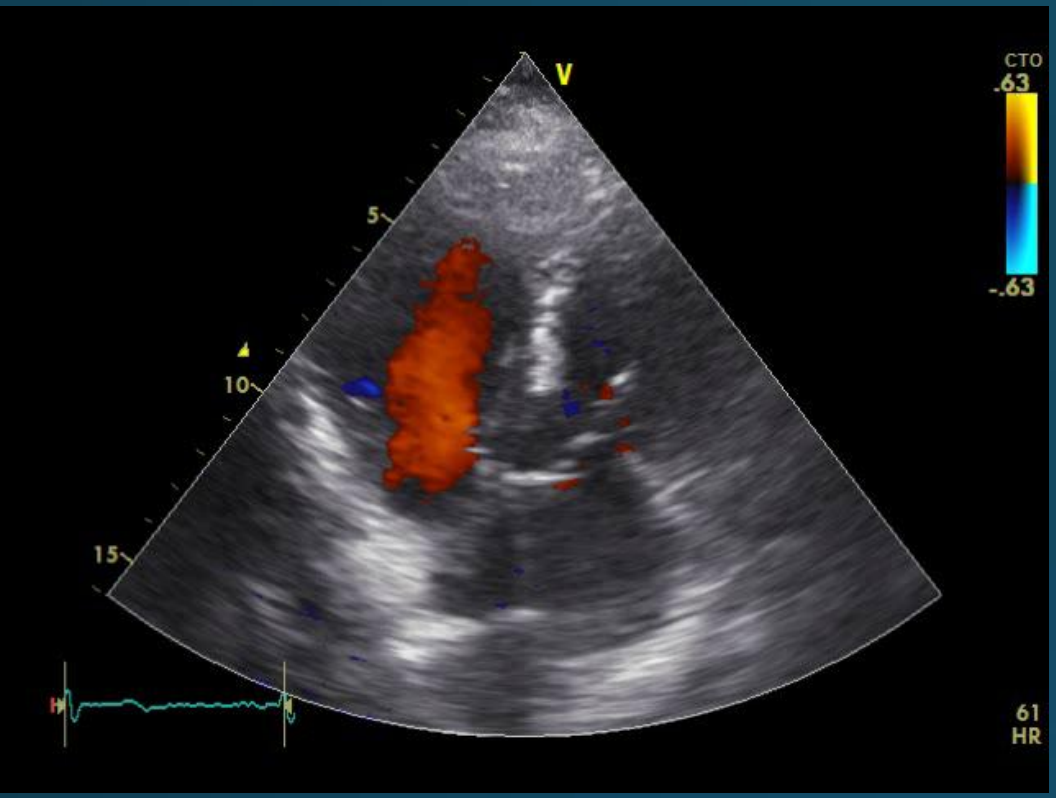
- Κλινικά
  - ΑΠ 130/80mmHg
  - Καρδιά: S1/S2+ 2/6 αριστερά παραστερνικά
  - Αναπνευστικό: ρεκχάζοντες άμφω
- ECG SR 62bpm, RVH
- 6MWT 380m 90%-88%
- Ntpro-BNP 637pg/ml
- Echo:
  - LA 44mm, IVS10mm, LV 46/31mm, KE 60% TR 5.5m/s, RVSP 131mmHg
  - Επιπέδωση IVS (D-shaped RV), RVH , Οριακή IVC με 50% αναπνευστική διακύμανση ,TP 3/4

Echo 2018

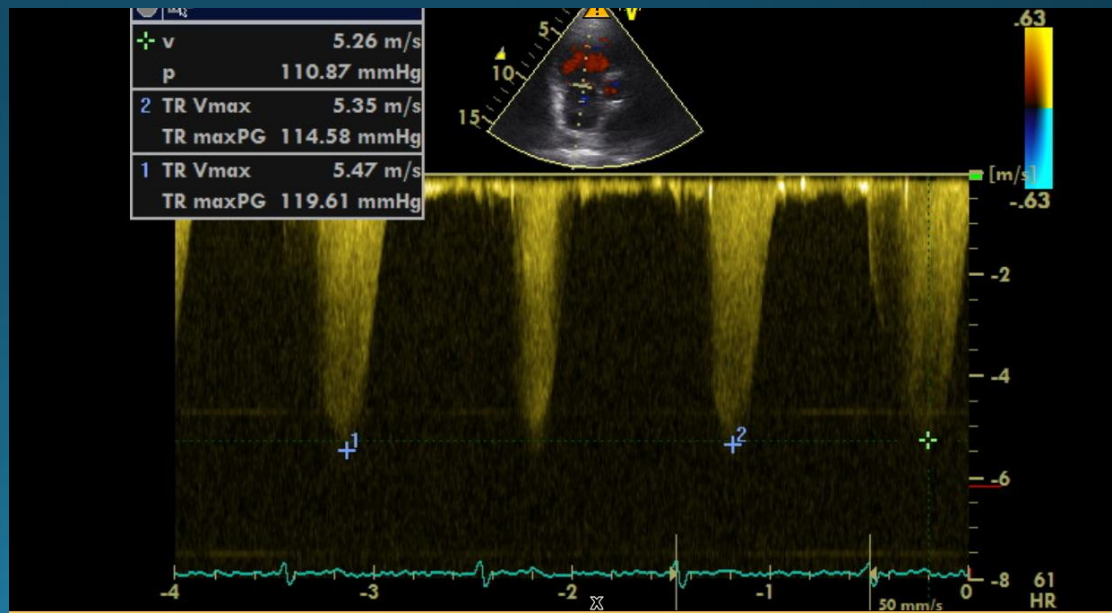




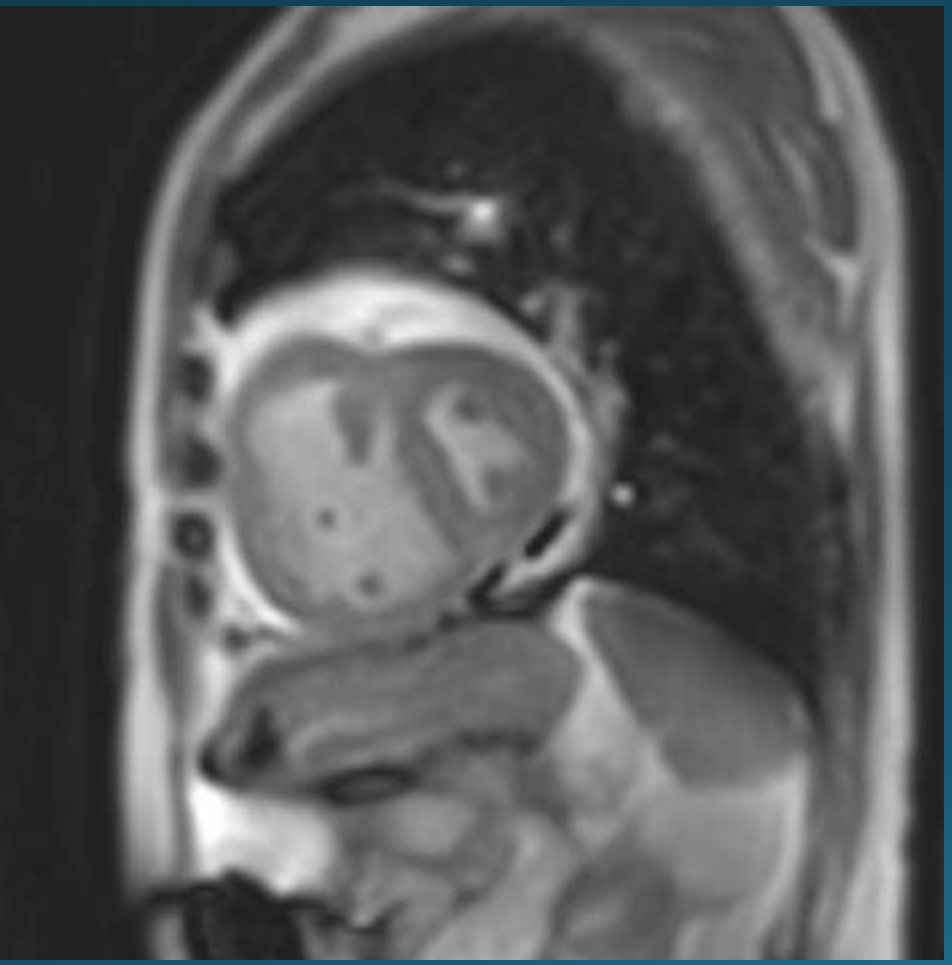


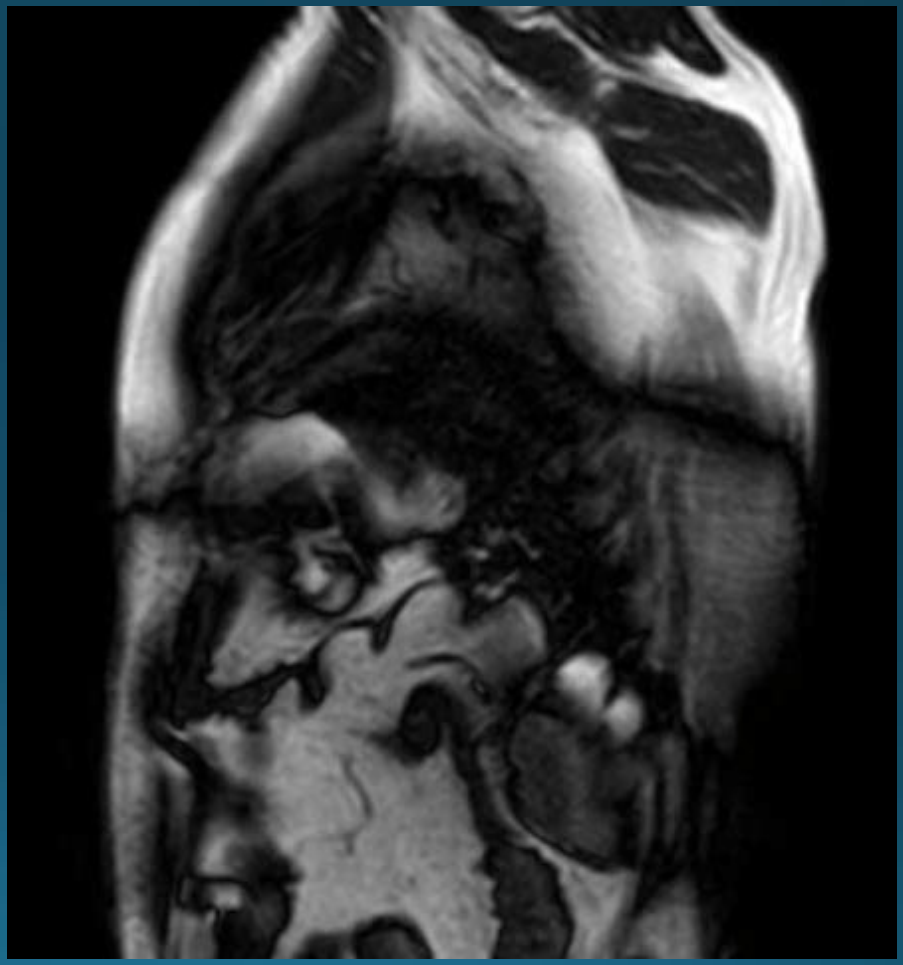

CTO
.63
-.63
V
5
10
15
61
HR

Echo 2018

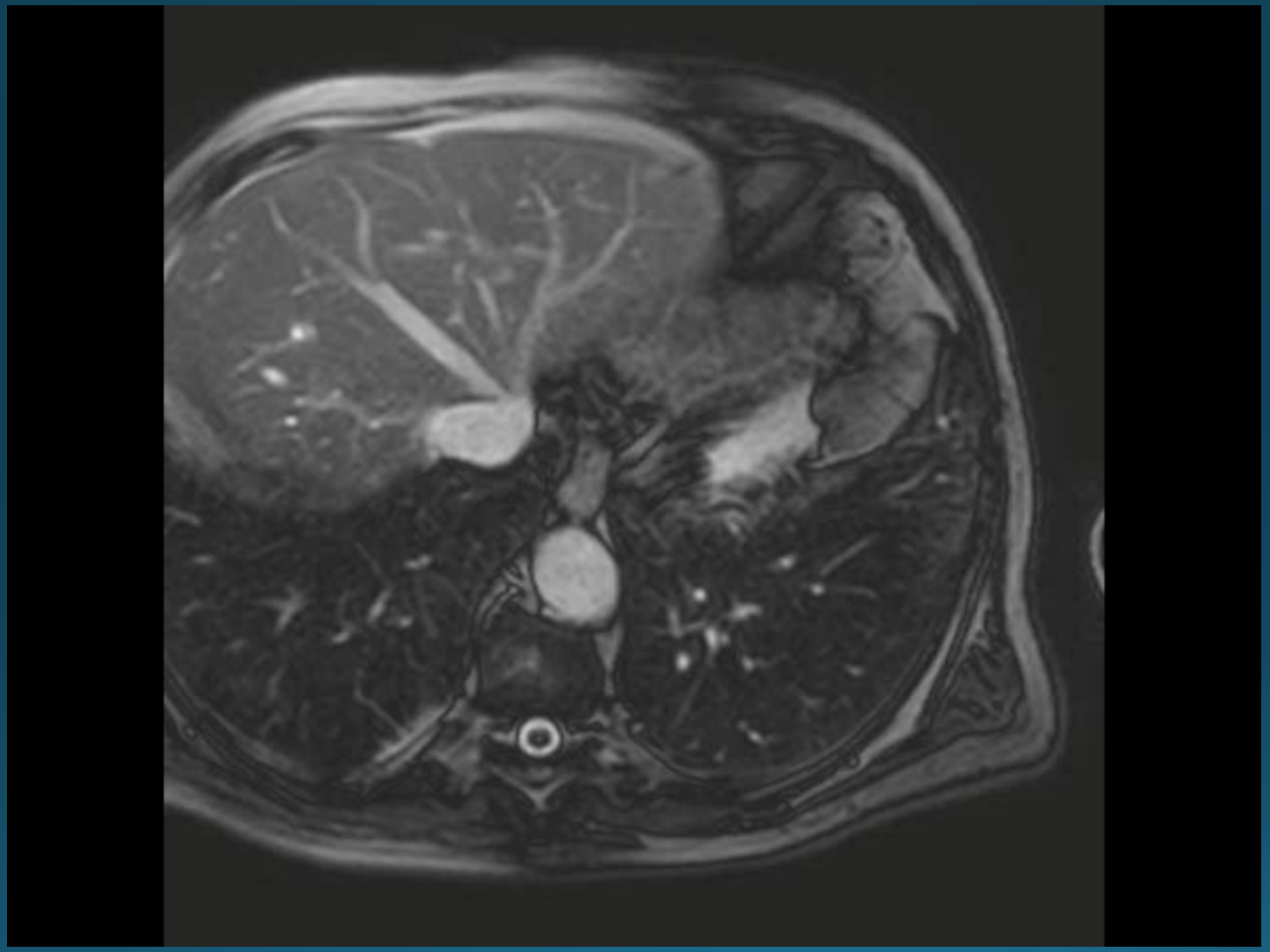

v 5.26 m/s
p 110.87 mmHg
2 TR Vmax 5.35 m/s
TR maxPG 114.58 mmHg
1 TR Vmax 5.47 m/s
TR maxPG 119.61 mmHg
V
5
10
15
.63
-.63
[m/s]
-2
-4
-6
-8
-4
-3
-2
-1
0
50 mm/s
61
HR

MRI 2019

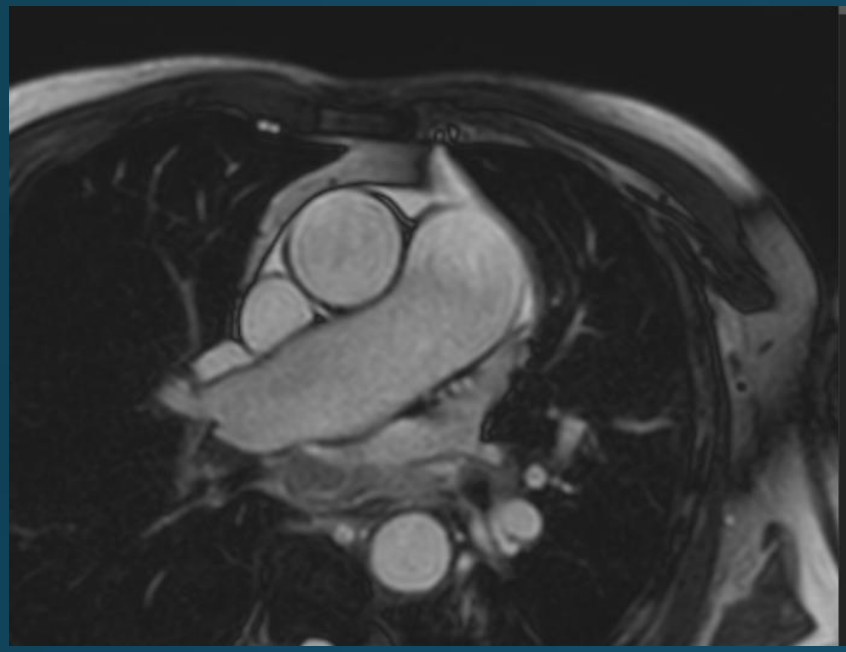


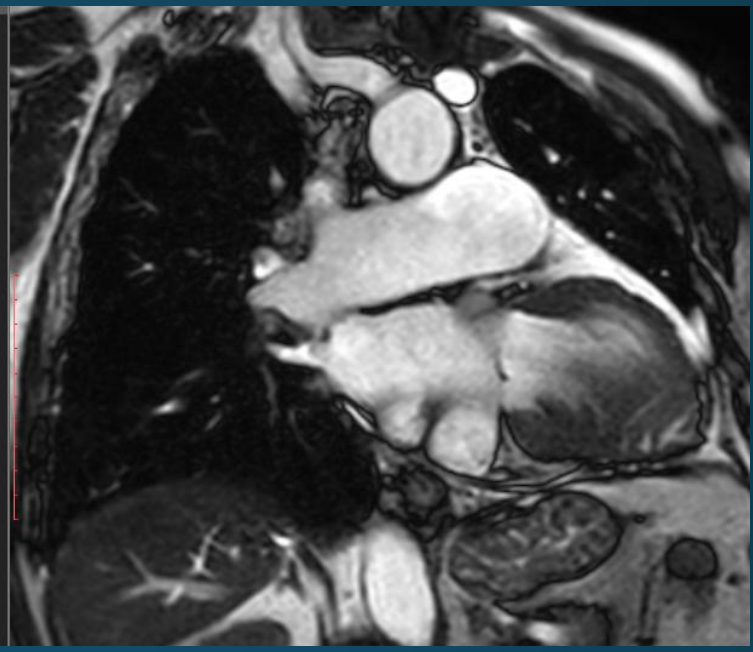


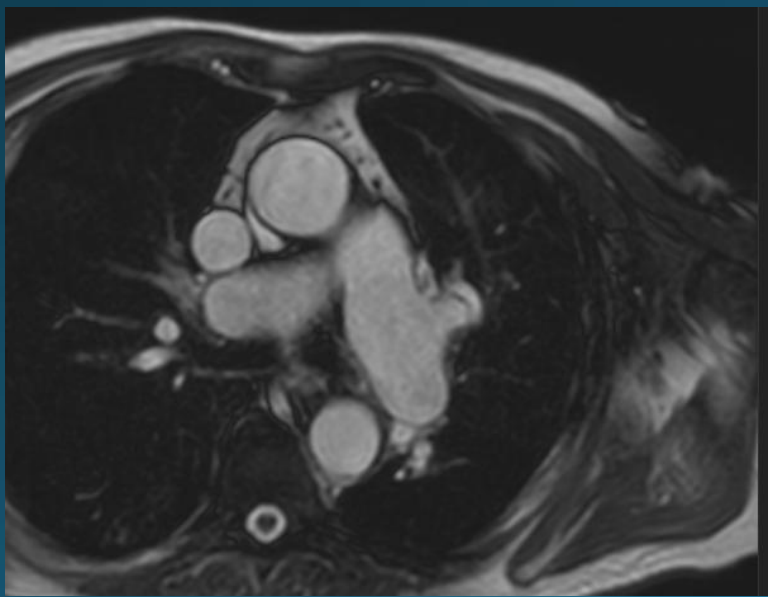
MRI 2019

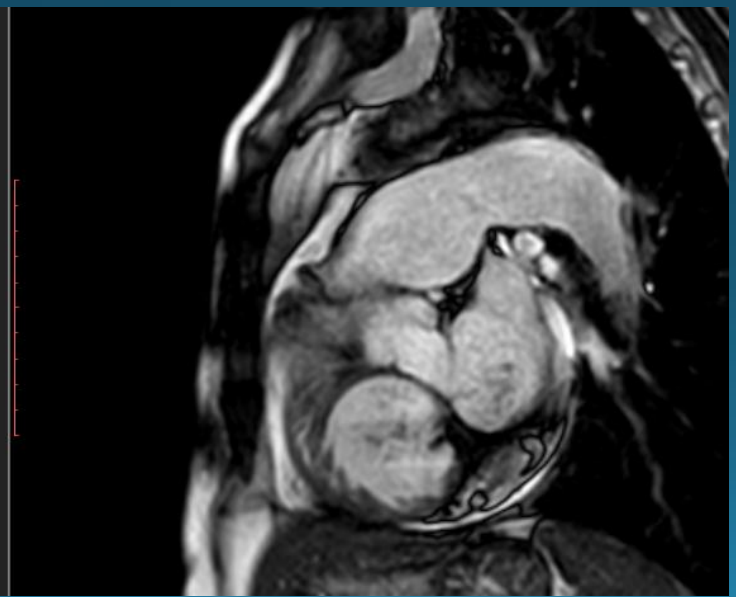


MRI 2019

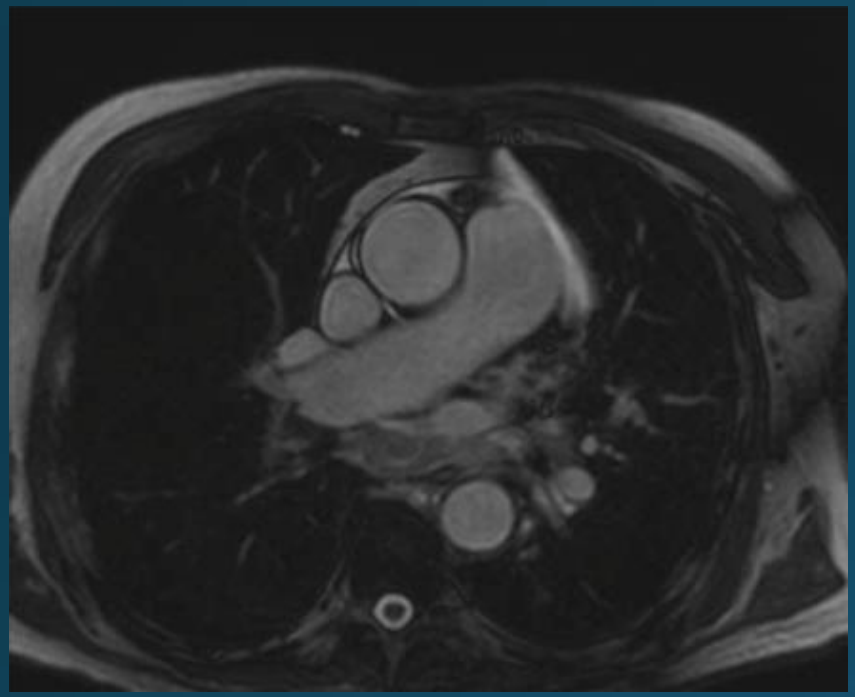


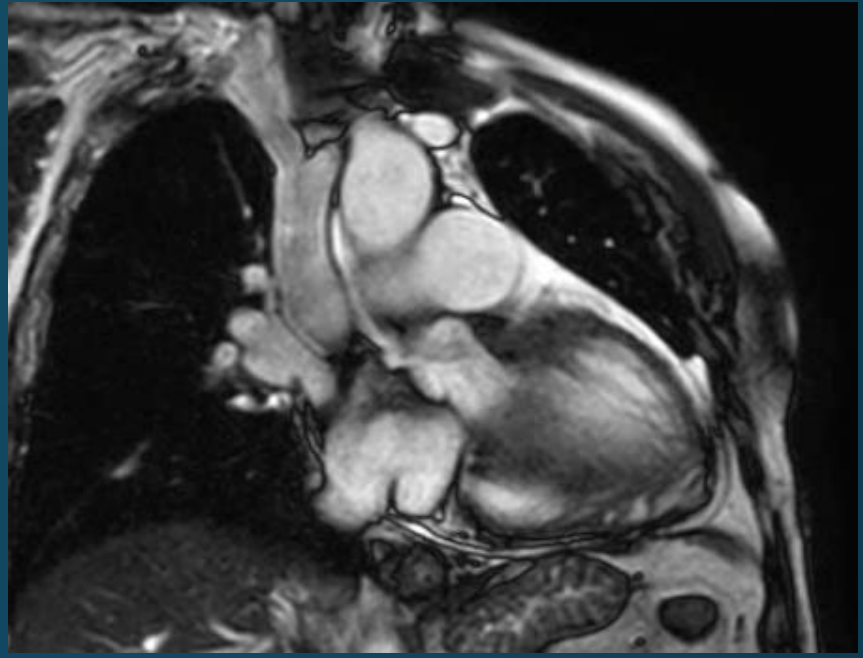


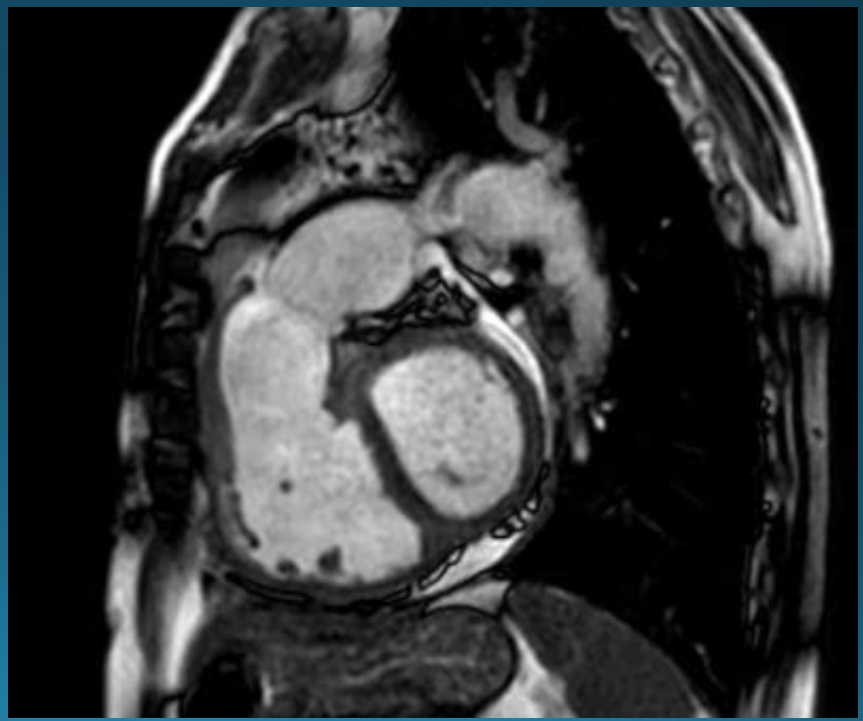




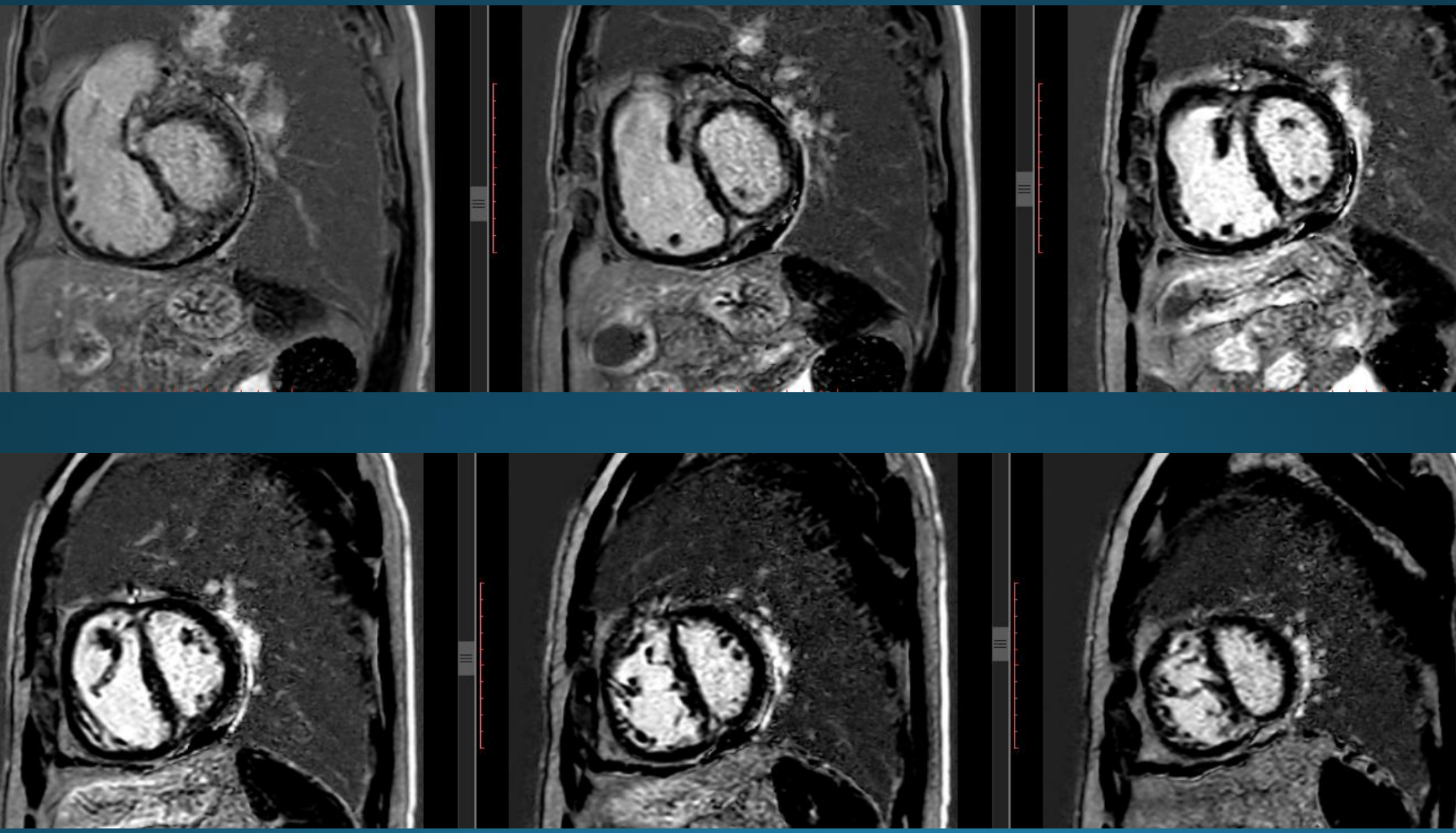
MRI 2019



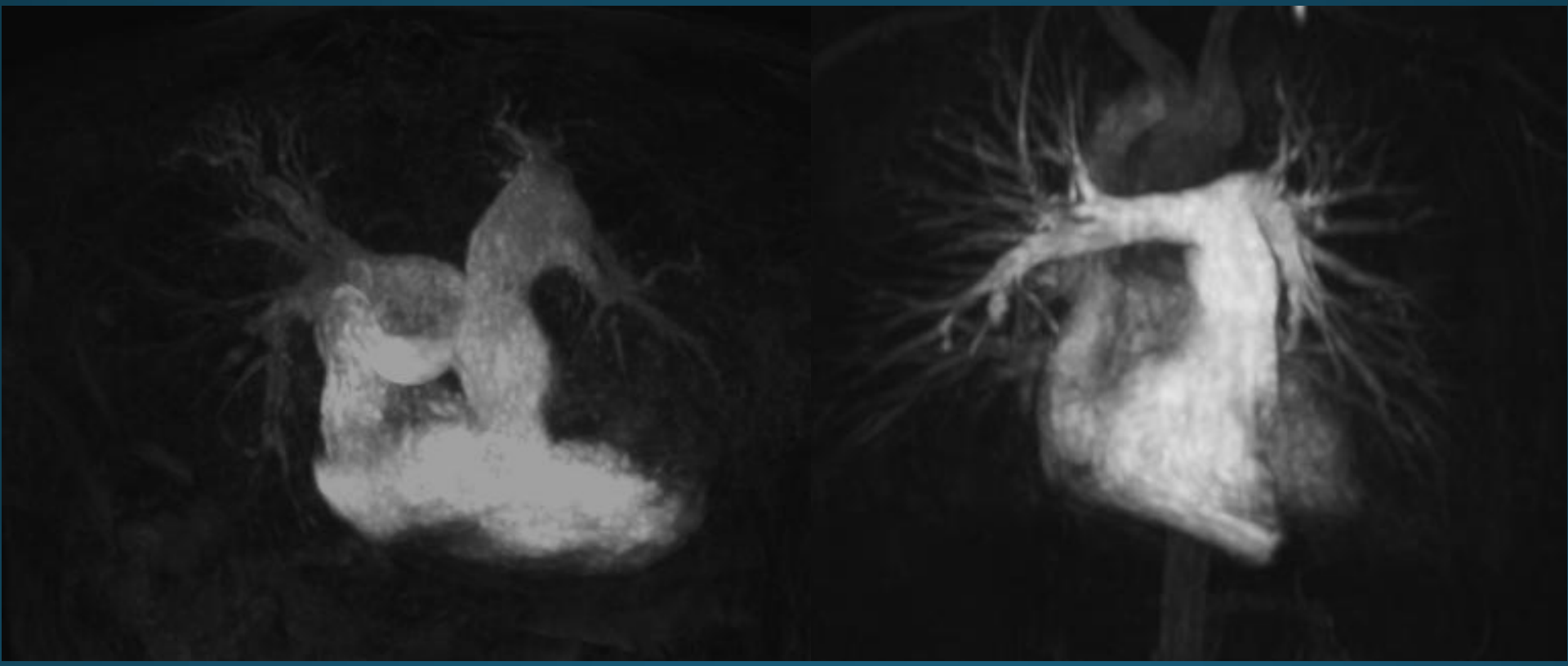




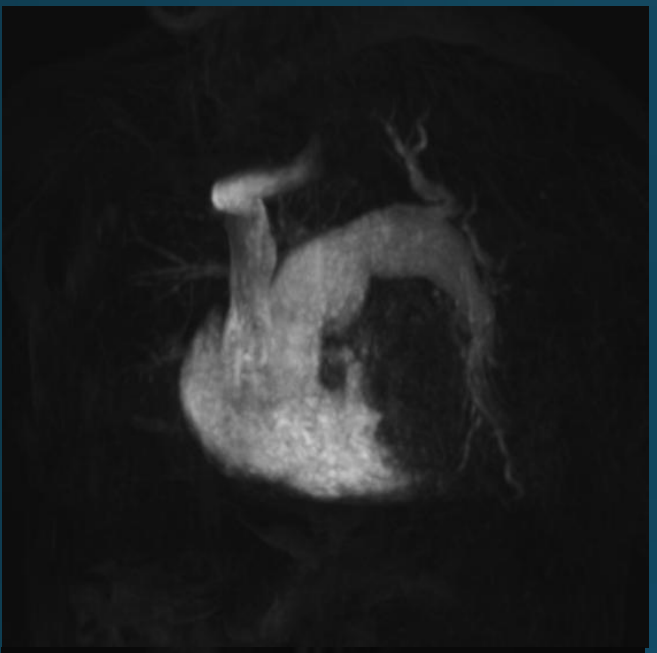
MRI 2019

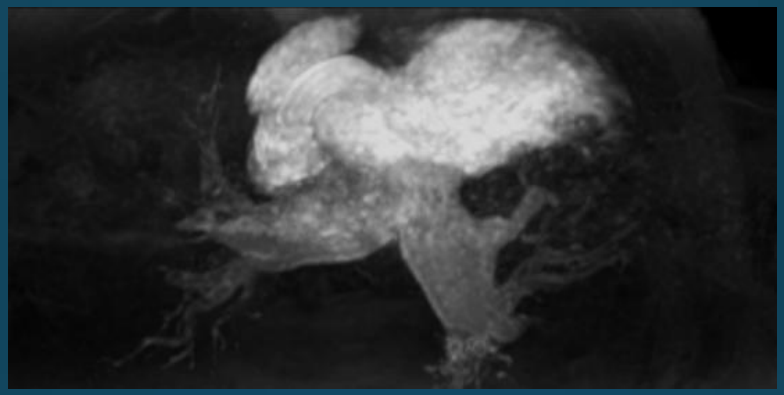


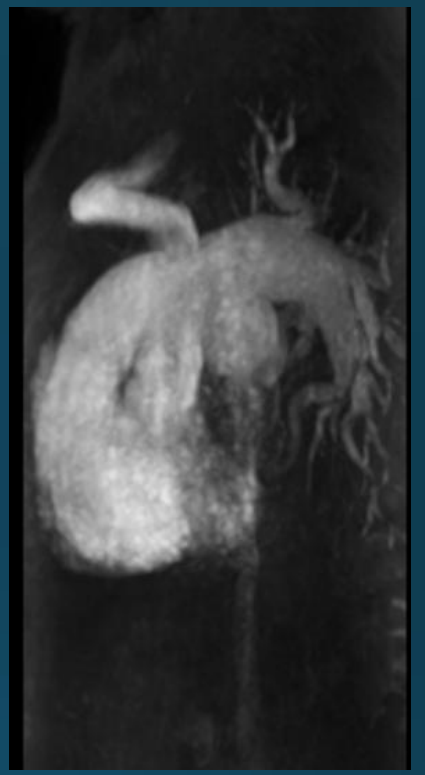
Ce-MRA

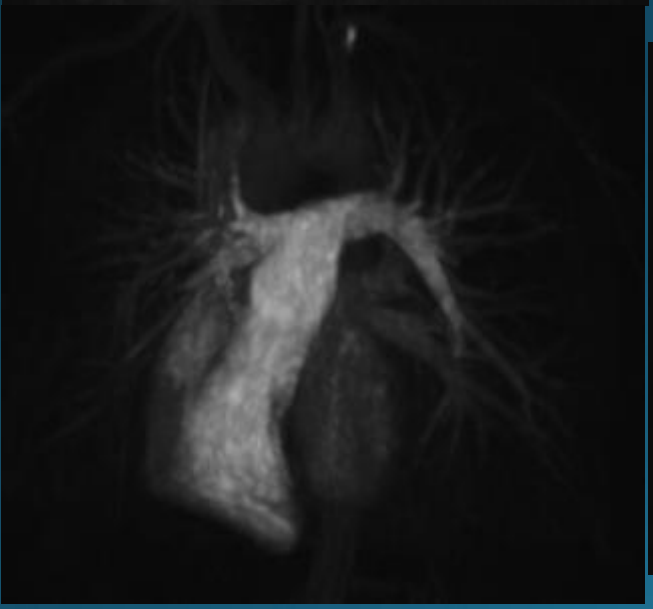


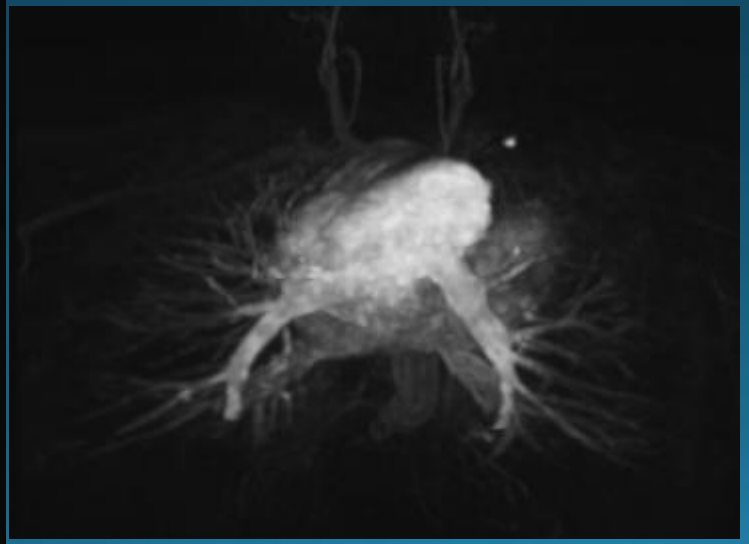
Ce-MRA

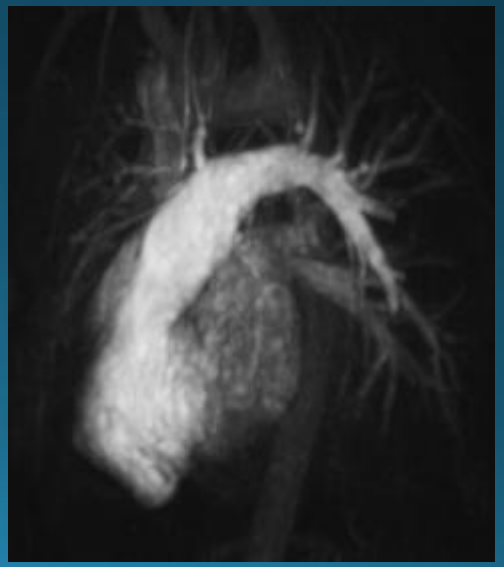












Φαρμακευτική αγωγή

- Προσθήκη Riociguat 1.5x3

Original Article

Noninvasive Assessment of Pulmonary Hemodynamics in Patients With Chronic Thromboembolic Pulmonary Hypertension by High Temporal Resolution Phase-Contrast MRI

Correlation With Simultaneous Invasive Pressure Recordings*

Karl-Friedrich Kreitner, MD, PhD; Gesine Maria Wirth, MD; Frank Krummenauer, PhD; Stefan Weber, PhD; Michael Bernhard Pitton, MD, PhD; Jens Schneider, MD; Eckhard Mayer, MD, PhD; Christoph Dueber, MD, PhD

Background—Right heart catheterization is the gold standard for assessment of pulmonary hemodynamics in patients with chronic thromboembolic pulmonary hypertension. To date, MRI has not been able to produce precise measurements of mean pulmonary arterial pressure (mPAP). The purpose of the study was to create a model for estimating mPAP and pulmonary vascular resistance in patients with chronic thromboembolic pulmonary hypertension by high temporal resolution phase-contrast MRI (PC-MRI) and to correlate the results with simultaneously acquired, invasive catheter-based measurements (simultaneously measured mPAP) and with right heart catheterization measurements.

Methods and Results—A total of 19 patients with chronic thromboembolic pulmonary hypertension underwent right heart catheterization and—after digital subtraction angiography of the pulmonary arteries—subsequent PC-MRI at 1.5 T with simultaneous recording of mPAP. Velocity– and flow–time curves of PC-MRI were used to calculate absolute acceleration time (Ata), maximum of mean velocities (MV), volume of acceleration (AV), and maximum flow acceleration (dQ/dt). On the basis of these parameters, multiple linear regression analysis revealed maximum achievable model fit (B=0.902) for the following linear combination equation to calculate mPAP (mPAP_cal): mPAP_cal=69.446−(0.521×Ata)−(0.570 ×MV)+(1.507×AV)+(0.002×dQ/dt). There was a statistically significant equivalence of mPAP_cal and simultaneously measured mPAP with a goodness of fit of 0.892. Pulmonary vascular resistance was overestimated by calculated pulmonary vascular resistance on the basis of PC-MRI in comparison with right heart catheterization–based measurements by a median of −112 dyn·s·cm^{-5}, the pairwise regression formula revealed a goodness of fit of 0.792.

Noninvasive Estimation of PA Pressure, Flow, and Resistance With CMR Imaging

OBJECTIVES The aim of this study was to develop a composite numerical model based on parameters from cardiac magnetic resonance (CMR) imaging for noninvasive estimation of the key hemodynamic measurements made at right heart catheterization (RHC).

BACKGROUND Diagnosis and assessment of disease severity in patients with pulmonary hypertension is reliant on hemodynamic measurements at RHC. A robust noninvasive approach that can estimate key RHC measurements is desirable.

METHODS A derivation cohort of 64 successive, unselected, treatment naive patients with suspected pulmonary hypertension from the ASPIRE (Assessing the Spectrum of Pulmonary Hypertension Identified at a Referral Centre) Registry, underwent RHC and CMR within 12 h. Predicted mean pulmonary arterial pressure (mPAP) was derived using multivariate regression analysis of CMR measurements. The model was tested in an independent prospective validation cohort of 64 patients with suspected pulmonary hypertension. Surrogate measures of pulmonary capillary wedge pressure (PCWP) and cardiac output (CO) were estimated by left atrial volumetry and pulmonary arterial phase contrast imaging, respectively. Noninvasive pulmonary vascular resistance (PVR) was calculated from the CMR-derived measurements, defined as: (CMR-predicted mPAP – CMR-predicted PCWP)/CMR phase contrast CO.

RESULTS The following composite statistical model of mPAP was derived: CMR-predicted mPAP = –4.6 + (interventricular septal angle × 0.23) + (ventricular mass index × 16.3). In the validation cohort a strong correlation between mPAP and MR estimated mPAP was demonstrated (R^2 = 0.67). For detection of the presence of pulmonary hypertension the area under the receiver-operating characteristic (ROC) curve was 0.96 (0.92 to 1.00; $p < 0.0001$). CMR-estimated PVR reliably identified invasive PVR ≥3 Wood units (WU) with a high degree of

Συμπερασματικά

- Η απεικόνιση αποτελεί σημαντικόκομμάτι τοσο για τη διάγνωση όσο και για την παρακολούθηση ασθενών με πνευμονική υπερταση
- Η MRI καρδιάς μπορεί δυνητικά να απαντήσει αξιόπιστα σε διαγνωστικά ερωτήματα ή να επιβεβαιωσει τα ευρήματα άλλων διαγνωστικών τεχνικών (είναι one stop shop απεικονιστική μέθοδος?)

Ευχαριστώ πολύ