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Clinical History :

Previous TEVAR procedure. For HTN protocol please. Afternoon appointment if possible.

MRI Cardiac Congenital Morphology Study :

MRI Cardiac ventriculogram :

MRI Cardiac Myocardial Viability :

MRA Pulmonary Arteries :

MRA Aorta Thoracic :

MRI Head :

MRA Head :

MRI Neck :

Clinical History:

Previous TEVAR procedure. For HTN protocol please.

Height 160 cm Weight 54.7 kg Body Surface Area 1.56 m2

Body Mass Index 21.4

â€¢ Normal weight = 18.5â€“24.99

BP on scanning = 205/101

Grade 3: Systolic 180 mm Hg or greater and/or diastolic 110 mm Hg or greater.

Brain

Incomplete circle of Willis.

Vertebral arteries: Normal

Posterior communicating arteries: Normal right and absent left.

Cross section mid neck pre-common carotid bifurctation.

Right common carotid artery

Size(mm) 6.7

Max velocity(cm/sec) 38

Antegrade flow(ml) 3.96

Effective flow (l/min) 0.3

Left common carotid artery

Size(mm) 5.8

Max velocity(cm/sec) 51

Antegrade flow(ml) 3.91

Effective flow (l/min) 0.3

Right vertebral artery

Size(mm) 3.4

Max velocity(cm/sec) 36

Antegrade flow(ml) 1.74

Effective flow (l/min) 0.13

Left vertebral artery

Size(mm) 3.3

Max velocity(cm/sec) 33

Antegrade flow(ml) 2.03

Effective flow (l/min) 0.15

Coronal FLAIR â€“ moderate diffuse, bilateral patchy periventricular high signal in

keeping with evidence of small vessel ischaemic change.

Sagittal T1 MPRAGE; for cerebral volumetrics â€“ normal intracerebral appearances (formal

cerebral mass evaluation not performed)

Thorax

The lungs are clear with no evidence of any extrathoracic abnormality seen on the axial

stack. Gross cardiac connections are normal.

Small volume of probably physiological pericardial fluid.

Atria

Mild LA dilatation. The vena cavae drain appropriately.

In ventricular systole:

Left Atrial Area (4ch) = 26cm2 = 17cm2/m2

Left Atrial Area (2ch) = 24cm2 = 15cm2/m2

LA length (4ch) = 5.1cm

LA length (2ch) = 5.7cm

LA volume = (0.85) x A1 x A2/L (shortest L (cm)) = 104ml = 67ml/m2

LA enlargement is defined as â‰¥55ml/m2 (>2 standard deviations above the mean published

references value for normal, healthy volunteers).

Right Atrial Area = 21cm2 = 13cm2/m2

Atrioventricular valves

Both AV valves are thin mobile and open well with no evidence of significant valvular

regurgitation.

Ventricles

Subjectively, the right ventricle is of normal size, morphology and appears to function

well with no evidence of wall motion abnormality.

Subjectively, the left ventricle is of normal size and functions well. Normal morphology.

Reduction in long axis function.

LV hypertrophy/LV phenotype:

Concentric remodelling â€“ Increased M/V, normal LVMi

Basal anterior interventricular septum = 8mm

Basal inferolateral LV wall = 8mm

LV end-diastolic diameter = 34mm

LV end-systolic diameter = 19mm

MAPSE = 8mm

TAPSE = 24mm

Global longitudinal strain (4ch) = -15.6 %

Global longitudinal strain (2ch) = -14.6 %

Reversed E/A ratio

Imparied diastolic LV filling curve.

Ventricular functional analysis is as follows (indexed values in brackets per m2); with

Normal ranges (Male â‰¥35 years) - J Cardiovascular Magnetic Resonance. 2005;7(5):775-82.

LV shax cvi42

Ejection Fraction (%) 63;59-83

End Diastolic Volume ml(ml/m2) 103(66;53-97)

End Systolic Volume ml(ml/m2) 38(24;10-34)

Stroke Volume (ml) 65

Cardiac Output (l/min) 4.6

Cardiac Index (l/min/m2) 2.9

Mass g(g/m2) 119(76;42-78)

Gadolinium Delayed Enhancement

No evidence of gadolinium delayed enhancement to suggest intramyocardial fibrosis/scar

formation, infiltration or inflammation.

Pulmonary Valve and Pulmonary Arteries

The pulmonary valve has not been formally assessed but where seen appears normal. The

pulmonary arteries are of normal size and appearance.

Pulmonary Veins

The pulmonary venous drainage is normal

Aortic valve and aorta

The aortic valve is trileaflet, thin, mobile and opens well.

The thoracic aorta is left-sided with a normal branching pattern. TEVAR in descending

aorta; the L SCA is not covered.

No significant ascending aortic dilation. No coarctation.

Mid Ascending

Peak (cm/s) 69.7

Antegrade (ml) 54.6

Retrograde (ml) 0

Net Flow (l/min) 4.03

Time to Peak flow

A (ms) 91

Area in systole (mm2) 640

Area in diastole (mm2) 615

S/D area ratio 1.04

Mid Descending

Peak (cm/s) 144

Antegrade (ml) 35.7

Retrograde (ml) 0

Net Flow (l/min) 2.65

Time to Peak Flow D (ms) 101

Area in systole (mm2) 693

Area in diastole (mm2) 649

S/D area ratio 1.07

Distance from asc to desc arch (mm) 152

D â€“ A (ms) 10

Pulse Wave Velocity (m/sec) 15.2

PWV >5-6m/sec is elevated and may reflect increased aortic stiffness

Aortic annulus 21mm

Sinus of Valsalva 29mm

Sinotubular Junction 23mm

Mid Ascending 27mm

Proximal Arch 26mm

Mid Arch 21mm

Distal Arch 19mm

Proximal Descending 27mm; graft

Mid Descending 26mm; graft

Diaphragm Hiatus 27mm; graft

Mid Abdominal 23mm; infrarenal aneurysm

13mm aneurysmal R CIA. Stenosed proximal L CIA. Disease EIAs.

Good quality angiography.

Abdomen

No significant intra-abdominal visceral abnormality is seen. Notably, the adrenals,

pancreas and left appear normal. Patchy right renal scarring and mild global parenchymal

atrophy.

30mm simple hepatic cyst in segment VII.

This does not exclude underlying metabolic disturbance. Tiny adrenal adenomas may be

missed.

Chronic dissection and abdominal aneurysm formation.

No evidence of renal artery stenosis.

Fair sized single renal arteries:

L = 5mm; diffuse disease, mild 3mm diameter proximal stenosis

R = 5mm; 1.3mm diameter proximal stenosis

SUMMARY

1. Normal weight

2. Grade 3 hypertension on scanning

3. Absent L PCOM only

4. Moderate diffuse cerebral small vessel ischaemic change

5. Mild LA dilatation

6. Mild LV long axis systolic impairment with evidence of diastolic impairment and mildly

reduced GLS

7. Pattern of concentric LV remodelling but no replacement fibrosis

8. TEVAR in situ with no L SCA coverage; increased aortic PWV

9. Bilateral renal artery stenosis; mild left and moderate to severe right â€“ CT renal

angiogram may be useful to confirm as this could be a significant hypertensive driver.

All recommendations and suggestions are subject to the clinical judgement of the attending

physicians, at whose discretion management decisions should be made based on all clinical

evidence.