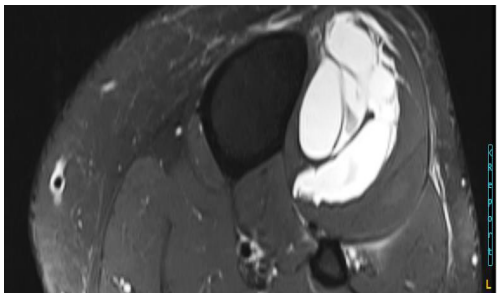
Issue 17 Jul 2023

IRIA Telangana e-Newsletter



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Indian Radiological & Imaging Association

Telangana State Chapter 2023

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From the President's Desk

Dear friends, senior and junior colleague members of TS IRIA chapter,

I am happy to share this edition of IRIA newsletter.

The news letter provides information regarding the Academic activities of the Radiology Association at various levels and highlights the Academic and personal achievements of the members.

It provides the details of the monthly meetings, special programmes of the IRIA TS chapter in a colorful presentation.

I request the members to contribute interesting cases and provide personal achievements to the editorial team.

I thank and congratulate Dr. Jagan Mohan Reddy and all other members of the editorial board for their hard work and coordination to bring the news letters.

Wishing you all the best.

Dr. Randhi Venkata Ramana
President TS Chapter IRIA

From the General Secretary Desk

Dear Esteemed Members of IRIA Telangana State Chapter,

Warm greetings to all of you!

As we gear up for another eventful year, I am delighted to announce the upcoming Annual State Conference of IRIA Telangana, which is set to take place in Hyderabad from 13th to 15th October 2023. This conference promises to be an exceptional learning and networking opportunity for all radiology professionals in our region.

We have curated a diverse and enriching program for this conference, and I am thrilled to inform you that we have confirmed the participation of five distinguished international speakers. Their expertise and insights will undoubtedly add immense value to our event, fostering an atmosphere of learning and collaboration.

To make this conference a resounding success, I earnestly request the participation of all our esteemed consultants, faculty members of medical colleges, and postgraduate students of radiology. Your presence and engagement are crucial to creating an environment of knowledge-sharing and growth in the field of radiology.

I extend a special appeal to all our consultants and medical college faculty to not only register for the conference yourselves but also to encourage your postgraduate students to actively participate. Their involvement will not only enrich their academic journey but also contribute to the overall success of the event.

I take this opportunity to congratulate all of you for the successful musculoskeletal ultrasound and interventions Cadaveric Hands-on Workshop conducted recently. The overwhelming response and positive feedback received were heartening. We are committed to organizing more such workshops in the future, covering various subjects, to further enhance the skillset and expertise of our members.

I am confident that with your enthusiastic participation and support, the Annual State Conference in Hyderabad will be a grand success, leaving an indelible mark on the radiology community in our state.

Let us come together as a strong, united community and make this conference a memorable and transformative experience for everyone involved. Your active involvement and dedication to the field of radiology are what make IRIA Telangana truly exceptional.

Looking forward to meeting each one of you at the conference!

Warm regards,

Dr. Krishna Mohan Pottala
General Secretary

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Author's: Dr. Vijaya Bhaskar Nori, Dr. Chesnal Dey Arepalli,
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ADVANCED CARDIAC MRI - BEYOND CONTRACTIONS AND SCARS

Cardiac MRI(CMR) has become an indispensable imaging modality in the evaluation of heart diseases. It is routinely used for functional analysis and to broadly identify whether the heart disease is ischemic or non-ischemic by the focal myocardial fibrosis patterns of late gadolinium enhancement (LGE). However, LGE has low sensitivity in detecting diffuse myocardial fibrosis where there is decreased contrast difference between the presumed normal/ unaffected and affected myocardium.

Routine CMR protocols have several sequences resulting in a long duration study and involve patient discomfort due to breath-holding in an already diseased patient.In addition, of late there is an increased interest in identification of sub-clinical diseasefor better patient care disease outcomes by using non-invasive cardiac imaging techniques with and without gadolinium administration. In this editorial, we describe several recent advancements in CMRwhich overcome the current limitations/ barriers and assessmentswhich are beyond contractions and scarsof a routine study.

1.QUANTITATIVE MYOCARDIAL PARAMETRIC MAPPING

Myocardial fibrosis refers to an increase in the extracellular interstitium of the myocardium; evaluation of it has prognostic implications as it affects diastolic and/or systolic functions. Increased extracellular matrix could be reactive due to edema or infiltrative due to fat, iron or abnormal protein deposition (for e.g. as in amyloid) or replacement type due to fibrosis/scar as seen in myocardial infarction. Thus, alteration in myocardial tissue composition due to various heart diseases modifies the inherent myocardial properties of T1, T2 or T2* which are now better assessed through corresponding myocardial/ parametric mapping techniques.

Mapping techniques refer to pixel-by-pixel quantification of relaxation times of longitudinal (T1 map), transverse (T2 or T2* maps) magnetization (depending upon the acquisition method) and are displayed through image-based signal intensities. Detailed description of different mapping techniques is beyond the scope of this editorial (1-4). Areas of fibrosis and increased ECV are associated with increased water content and thus prolong T1 relaxation times, an example of it is presented (Figure1).

2. CMR FINGERPRINTING (cMRF)

cMRF is an exciting multiparametric imaging technique that enables simultaneous myocardial T1 and T2 mapping in a single breath hold. In this novel technique, pulse sequence parameters are deliberately varied to produce a unique signal that depends exclusively on myocardial tissue properties of T1 and T2 (5).

3. STRAIN IMAGING

Left ventricular ejection fraction (LVEF) is used as a key quantitative parameter of left ventricular systolic function. However, LVEF is not a sensitive metric as EF may be normal in regional pathologies like ischemic cardiomyopathy, myocarditis or in conditions of heart failure with preserved ejection fraction (HFpEF). Myocardial impairment/ deformation occurs much earlier than the objective change in EF values.Myocardial deformation (strain) is altered in ischemic and non-ischemic cardiomyopathies. Several strain imaging methods

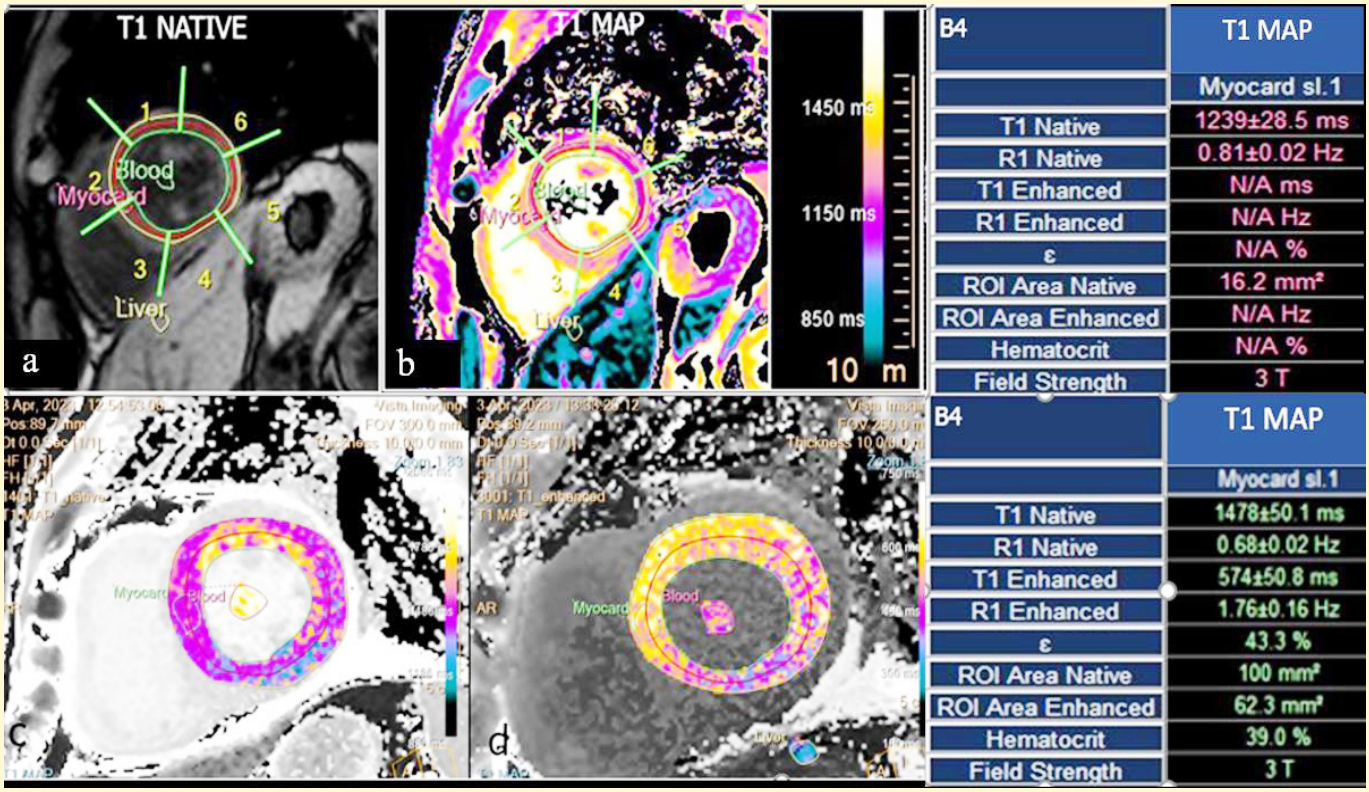

Figure 1. Normal patient (a) T1 native, (b) T1 map in short axis. Note the color map scale (b) and the myocardial values in a table (top panel B4). Amyloidosis patient (c) Short axis T1 native and (d) T1 enhanced with increased T1 native and T1 enhanced values besides the increased E: 43.3%) in a table (below panel B4)suggestive of increased extracelluar matrix. E – Extracellular Volume

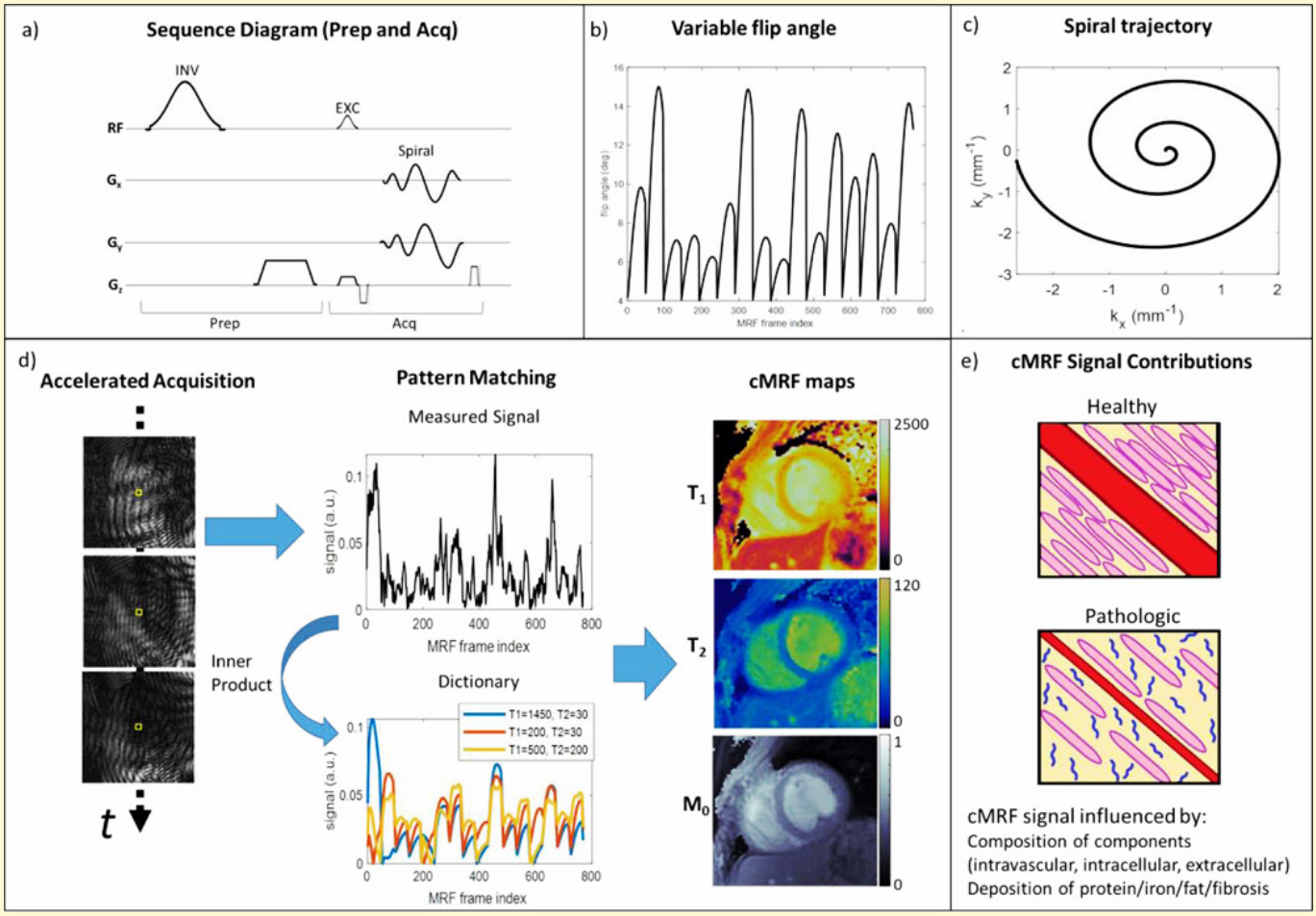

Figure 2:CMR Fingerprinting - Image courtesy from article by Brendan Eck et al (5) - ProgNuclMagnResonSpectrosc. 2021 February ; 122: 11–22. doi:10.1016/j.pnmrs.2020.10.001

allow for very sensitive assessment of myocardial deformation which has both diagnostic and prognostic implications. Some of the methods include feature tracking (FT), fast-strain encoded imaging (fSENC), and left-ventricular long axis strain (LVLAS) (6-8).

A. FEATURE TRACKING CMR

Feature tracking (FT) CMR is an emerging technique that assesses myocardial fiber deformation (strain in longitudinal, radial and circumferential) and motion at the global and segmental levels which would facilitate early detection or progression or severity of the disease.

The unique feature of FT CMR is that the strain patterns and values in these fibers could be acquired from routine (that are acquired for LV function) cine images of b-SSFB (balanced steady-state free precession) although dedicated software is required for delineating epicardial and endocardial contours and analysis.

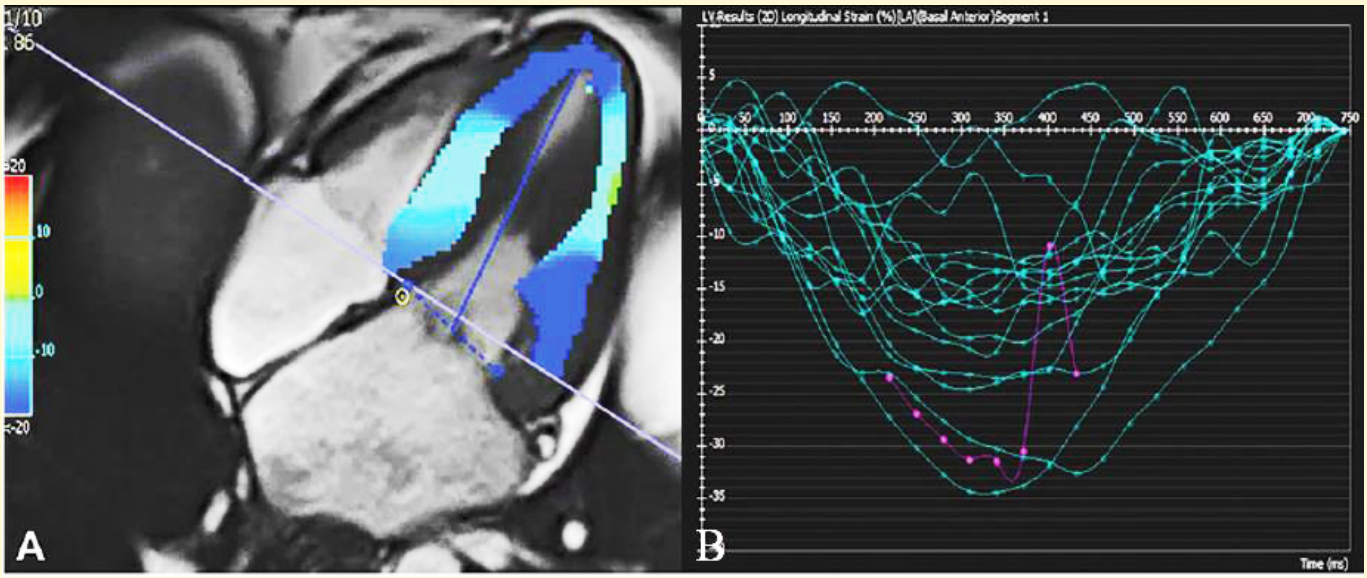

Figure 3: FT strain measurements. A. Color overlaid longitudinal strain data on four chamber cine SSFP image. Color scale ranges from -20% to + 20%. B. Graph shows the strain rate (%) of all 17 American Heart Association segments. Vertical axis shows longitudinal strain and horizontal axis shows time in milli seconds.

B. FAST-SENC

Fast strain-encoded (SENC) CMR imaging (fast-SENC) is a novel technique that enables assessment of cardiac contractility in a single heartbeat. fSENC uses tag lines that are oriented parallel to the imaging plane and thus measures longitudinal strain.

4. SINGLE BREATHHOLD – ULTRAFAST CMR

CMR is time-consuming as it involves the acquisition of a stack of slices in multiple planes. Isotropic 3D cine (Enhanced sensitivity encoding [SENSE] by Static Outer volume Subtraction [ESSOS]) and isotropic 3D LGE sequences arehighly accelerated cardiac cine acquisition methods which acquire the data in a single breath holdof ~15-24s(9).

ESSOS reconstruction is based on the acquisition of 2 interleaved datasets, one static and the other dynamic. Although ESSOS works without contrast administration, however, due to technical limitations related to saturation of blood at 3T, contrast administration is necessary to delineate cardiac function. This proposed protocol does not involve any specific hardware upgrades and has demonstrated its utility in standard 16-channel phased array coil. However, the protocol has been tested at 3.0-T field strength, and further validation will be required for more established 1.5-T systems with lower signal to noise.

5. FREE-BREATHING CMR

Compressed sensing (CS) techniques with sparse sampling and iterative reconstructions reduce drastically the acquisition time of CMR, however, the image quality is poor. A new technique – Real time (RT) CMR promises to overcome the limitations with superior image quality. RT CMR images are binned based on respiration and ECG-derived RR and thus allow free breathing during cine acquisition (10). Although, the scan time is slightly increased, it allows for patient comfort and compliance especially in children and patients with congestive heart failure.

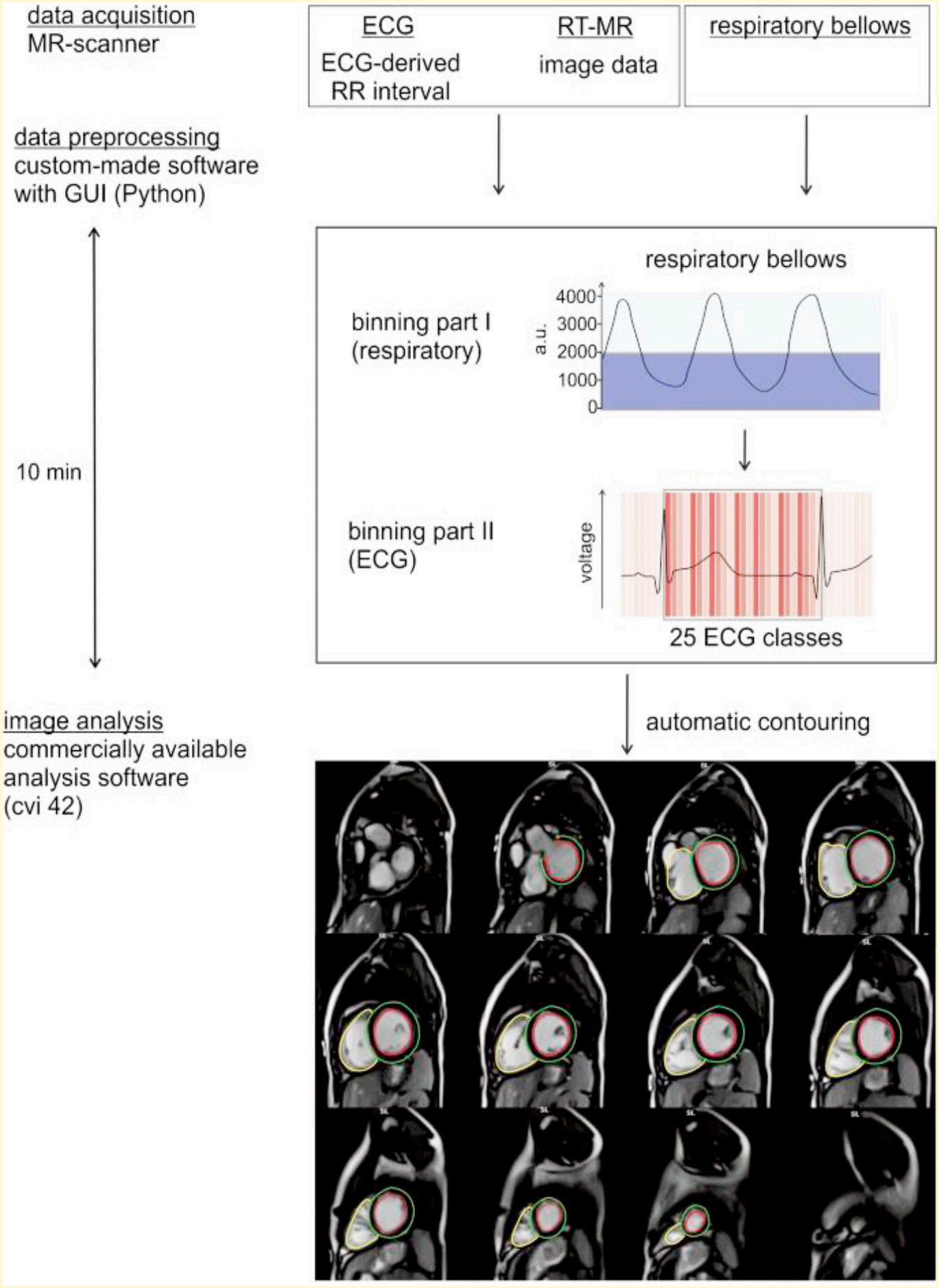

Figure 4: RT-MRI processing protocol. RT – real time. Image courtesy - Lena Maria Röwer et al. Pediatric Radiology volume 52, pages1462–1475 (2022)

6. 4D Flow:

In many practices, 2D phase-contrast (2D-PC) MRI has been used for blood flow quantification. Four-dimensional flow MRI is a time-resolved volumetric acquisition that captures the vector field of blood flow along with anatomic images. It also provides a simpler acquisition compared with 2D-PC and facilitates a more accurate and comprehensive hemodynamic assessment (11- 12).4D flow MRI will likely become an essential component of cardiac imaging in practices involved in the management of congenital and acquired structural heart disease.

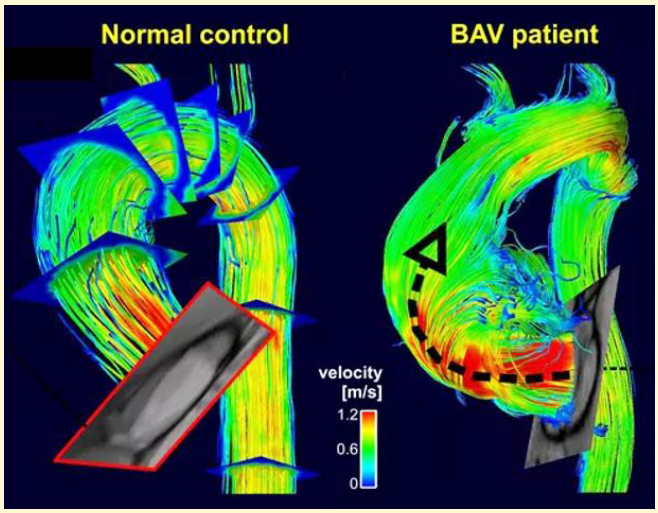

Figure 5: 4D flow in a normal and bicuspid aortic valve (BAV) patient. Segmentation shows location of analysis planes. Note increased velocities in proximal ascending aorta as seen in color map scale in BAV patient.

Conclusions:

Novel CMR techniques have wide clinical applications. CMR mapping and strain imaging techniques facilitate identification, quantification of inherent myocardial tissue properties which help in early detection, identification of progression or severity of heart disease. Advancements in accelerated imaging have significantly shortened scanning times to a single breath hold or to real time free breathing data acquisition which adds to patients comfort and compliance. Some of the new advanced techniques do not require gadolinium to be administered and are particularly useful in chronic renal failure patients.

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ACHIEVEMENTS

Dr. KPR Gold Medal Winner in 7th KARE-2023

Dr. Dhanalakshmi V
Government Stanley Medical College and Hospital

2nd Prize Winner

Dr. Mohammed Faizan Ul Haq
Shadan Institute of Medical Sciences

3rd Prize Winners

Dr. Sharath Chandra Goud
NIMS

Dr. Bhargavi P
NIMS

Congratulations Dr Krishna Mohan for receiving Vaidya Siromani award on the occasion of Doctors day

ARTICLES

Dr. Uma Karri
Senior Consultant Radiologist
Gleneangles Global Hospital

MDCT EVALUATION OF ABDOMINAL HERNIAS: PICTORIAL ESSAY

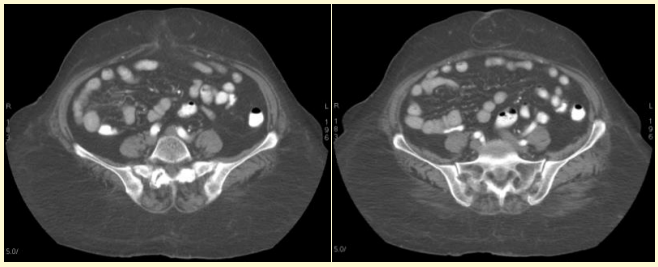
Abdominal wall hernias are the protrusion of abdominal contents through an area of abdominal weakness.

External abdominal wall hernias are classified as:

- **Abdominal wall hernias** (Epigastric hernia, Ventral hernia, Spigelian hernia, Lumbar hernia, Hypogastric hernia ,Interparietal, Parastomal) and Incisional hernias.
- **Pelvic wall and groin hernias** (Inguinal, Femoral and obturator hernias)

Multi detector CT helps in diagnosis, identification of contents and its complications. Differentiating between obstructed, strangulated and incarcerated hernia.

UMBILICAL HERNIA:



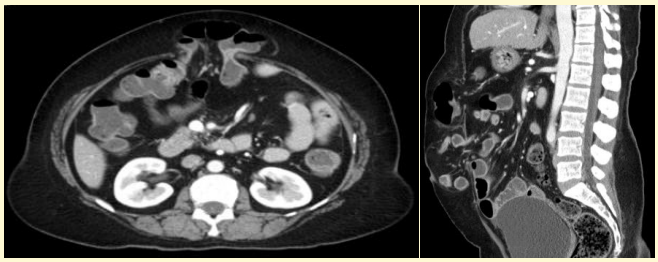
72/F presented with swelling and pain the umbilical region

EPIGASTRIC HERNIA:

- The defect is seen in mid-line above the umbilicus in the linea alba. Occurs in 3-5% of the population. Most commonly in men aged 20-30 years. In 20% cases there are multiple hernias.

52/F presented with vague swelling in the epigastrium

INCISIONAL HERNIA: Incisional hernias are delayed complications of abdominal surgery. More commonly encountered in association with vertical over transverse.



53/F presented with swelling in the supra and infra umbilical region with intermittent pain, showing multiple incisional hernias, containing omentum and bowel loops(colon and ileum) .

LUMBAR HERNIA:

Lumbar hernias may occur at weak points in the

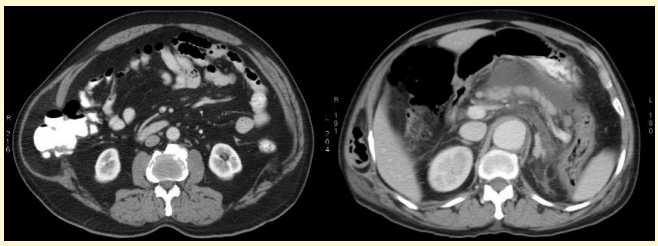
posterolateralabdominal wall through superior (triangle of grynfelt) or inferior (Petit triangle) lumbar triangles. Cause may be Spontaneous /post traumatic.

65/F presented with swelling in left lumbar region

Large left lumbar hernia noted containing both small and large bowel loops.

INTERSTITIAL/ PARIETALHERNIA:

Inter-parietal (interstitial) hernia refers to a hernia sac located in the fascial planes between the abdominal wall muscles that does not exit into the subcutaneous tissue. The hernial sac is characteristically confined between muscular layers.



57/M presented with small swelling in right lumbar region

SPIGELIAN HERNIA:

Anterolateral aspect of lower abdomen along **semilunar line** i.e. between fibrous union of rectus sheath with Transversus abdominis aponeurosis.

Cause may be congenital weakness. Contents prolapse through the lateral abdominal wall muscles like interstitial/interparietal plain

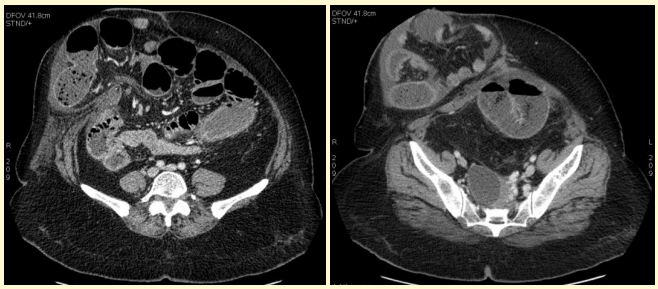
Small bowel loop trapped in the interstitial plain of abdominal wall along semilunar line

RICHTER HERNIA:

Richter hernia is a rare hernia where herniation of the antimesenteric wall of bowel noted, not the entire wall/ circumference herniate.

Herniation of antimesenteric border of bowel loop seen herniating into umbilicus

PARA-STOMAL HERNIA: This hernias occur along the ileostomy or colostomy stomas.

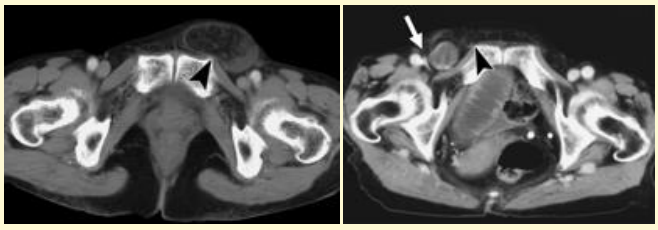


45/F presented with swelling around the colostomy with pain abdomen and vomiting.

CECT abdomen showed obstructed parastomal hernia in right iliac fossa with no strangulation.

PELVIC HERNIAS:

- Femoral vs Inguinal Hernia
- Relationship between the hernia sac and pubic tubercle.
- Relationship between hernia sac and inguinal ligament.
- Compression of the femoral veins



Inguinal hernia medial to tubercle

Femoral hernia Lateral to tubercle

Dr. Vikas Chennamaneni
Professor & HOD
Prathima Institute of Medical Sciences

ACUTE PANCREATITIS

Acute pancreatitis is a common condition but correct terminology is required to describe acute pancreatitis with complications on imaging. According to revised Atlanta Classification , acute pancreatitis is classified as early phase (<1wk) and late phase (>1wk) depending on duration. Based on severity, it is classified as mild, moderate and severe pancreatitis depending on presence and duration of organ failure and morphologically classified as interstitial edematous pancreatitis and necrotizing pancreatitis.

- Acute edematous pancreatitis is usually self limiting with mild changes like pancreatic enlargement and peripancreatic fat stranding. There may be pleural effusion and ascites. Peripancreatic collections are described as acute peripancreatic collections. These collections may organise over a time into pseudocysts.
- Acute necrotizing pancreatitis is classified based on extent of necrosis as pancreatic, peripancreatic or both. Collections noted adjacent to pancreas are termed as necrotic collections which may organise over time into walled off necrosis.

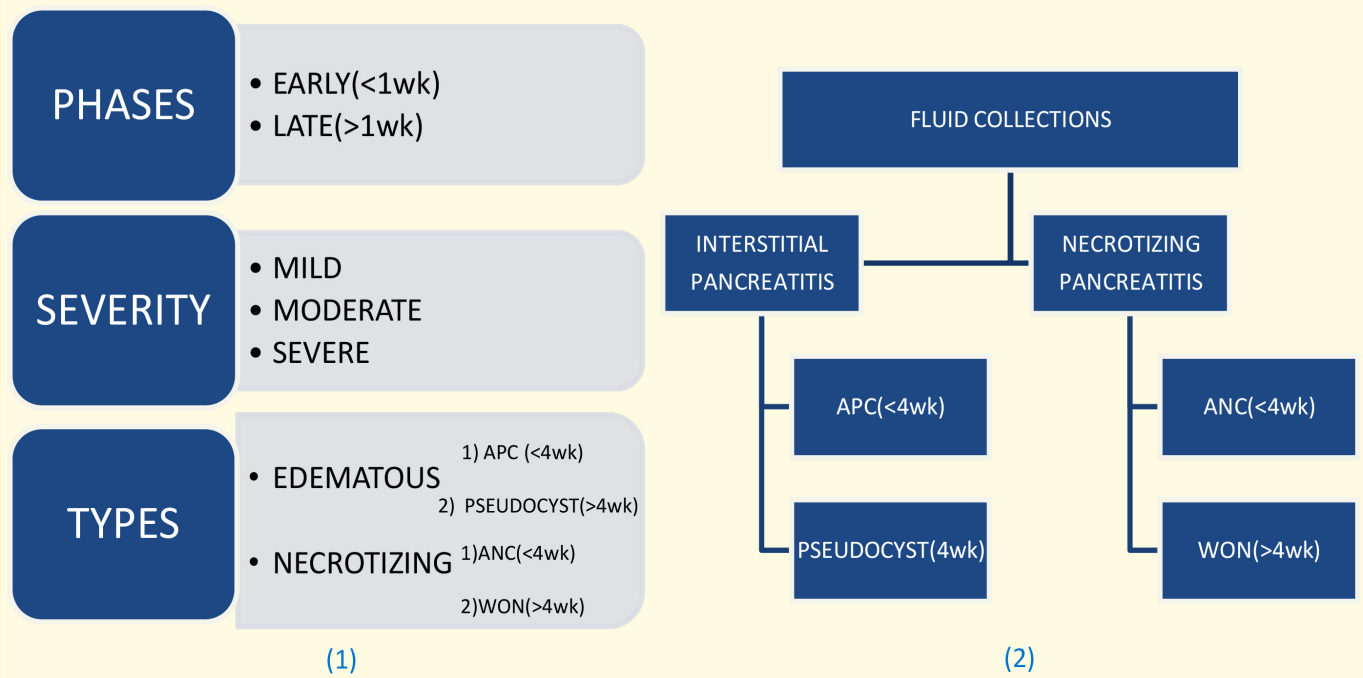
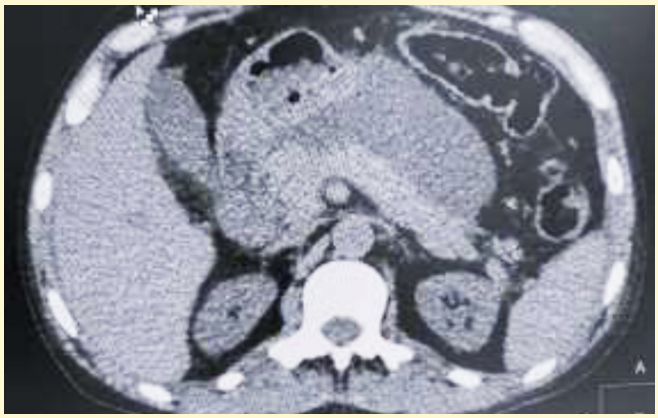


Fig. 1, 2 : APC Acute peripancreatic collection, ANC Acute necrotic collection, WON Walled off necrosis

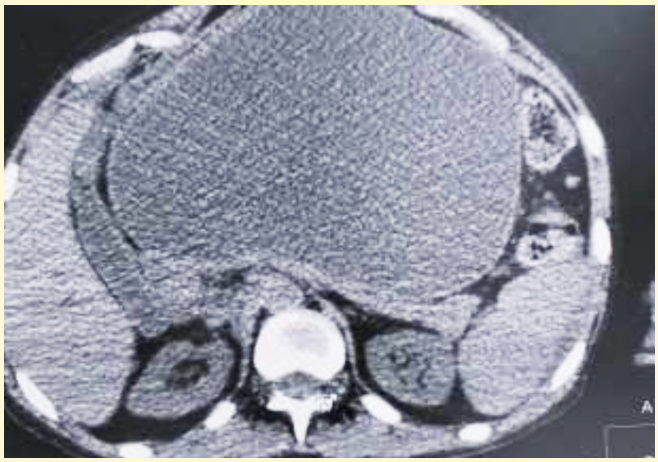
Complications like bleeding from pseudoaneurysm can be catastrophic. Diagnostic studies like CECT to be performed at appropriate time. Imaging is best performed 72hrs or late from the onset pancreatitis.

Medical and conservative management is the norm in treatment of acute pancreatitis and its complications. Aggressive medical management is required in the initial stages. Interventions are delayed as much as possible, as they introduce infection and are aimed at unresolving and symptomatic complications.

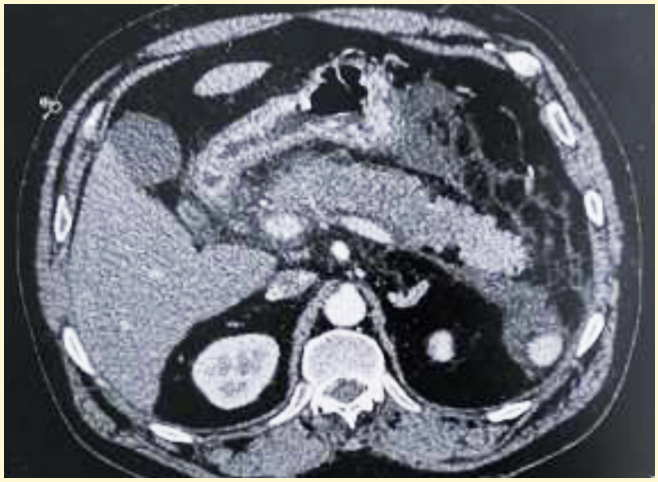
Cystogastrostomy is one of the common treatment strategy for large unresolving pseudopancreatic cysts. Open surgical debridement is reserved for infected pancreatic necrosis.



1: Acute peripancreatic collection



2: Pseudocyst of pancreas



3: Acute necrotizing pancreatitis (post contrast)

4 : Walled off necrosis

Dr. Sneha Sirigireddy
Consultant Radiologist, Yashoda hospitals

CT IN AORTIC DISSECTION AND OTHER ACUTE AORTIC SYNDROMES

INTRODUCTION:

Acute aortic syndromes encompass a spectrum of interrelated life-threatening conditions involving disruption of the intima-medial layer, including 1. Acute aortic dissection 2. Intramural hematoma 3. Penetrating atherosclerotic ulcer.

IMAGING:

Imaging is crucial in rapidly diagnosing, distinguishing between types of AAS, and evaluating additional complications for prompt and effective management. Non-contrast CT from the base of the neck to below the lesser trochanters to evaluate for intramural hematoma. This is followed by post-contrast imaging in the arterial phase with the same anatomic coverage, and ECG-gated imaging of the thoracic aorta to minimize cardiac motion artifacts for comprehensive evaluation.

AORTIC DISSECTION:

1. Classification: Stanford and DeBakey classification. Stanford type A dissection involves the ascending thoracic aorta regardless of the site of the entry tear and extension pattern. They require urgent surgical intervention. Stanford type B dissection involves the descending thoracic aorta distal to the left subclavian artery, which is managed medically unless there are complications necessitating surgical intervention.

2. Entry and exit tears: Large entry tears > 10mm indicate poor prognosis. Entry tear along lesser aortic curvature has a higher risk of retrograde extension.

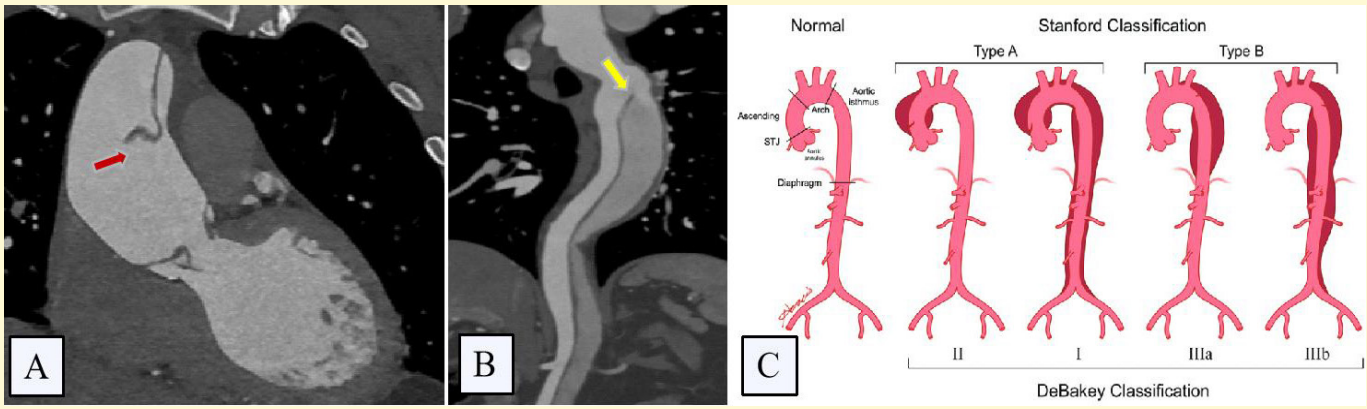


FIGURE 1. (A) Stanford type A Aortic Dissection involving ascending aorta with a large entry tear (red arrow). (B) Stanford type B Aortic Dissection arising distal to the origin of left subclavian artery involving descending aorta with a small entry tear (yellow arrow). (C) Graphical representation of Stanford and DeBakey classification of Aortic Dissection (1).

3. True vs False lumen:Differentiation of the true lumen from the false lumen is important for planning endovascular interventional procedures. True lumen is usually smaller with higher contrast attenuation than false lumen. False lumen demonstrates cobweb (medial fragments) and beak sign (acute angle). True lumen is seen continuous with the undissected aorta.

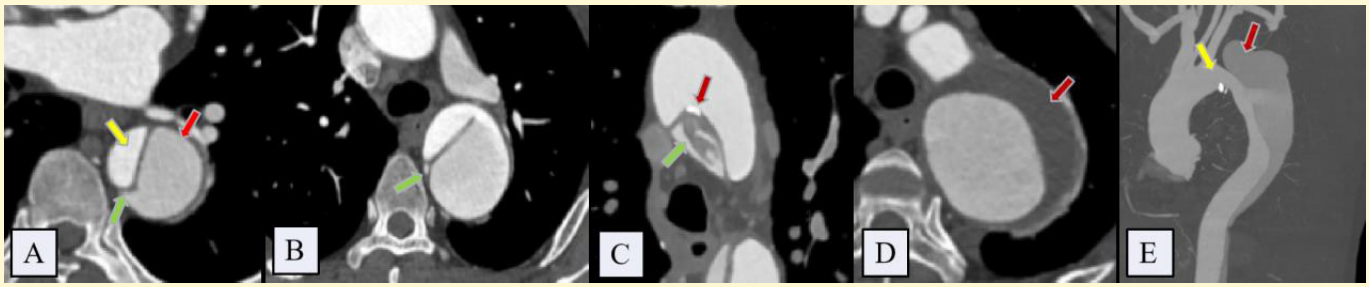


FIGURE 2. (A) demonstrates a true lumen (yellow arrow) smaller in size with higher contrast attenuation and a false lumen (red arrow) larger in size with lesser contrast attenuation. Green arrow indicates beak sign. (B) Demonstrating the cobweb sign. (C) Demonstrates cobweb sign (green arrow) and internally displaced intimal calcifications (red arrow) (D) Demonstrating hypodense thrombusin the false lumen. (D) True lumen (yellow arrow) continues with the undissected aorta and false lumen (red arrow) shows no communication with the undissected aorta.

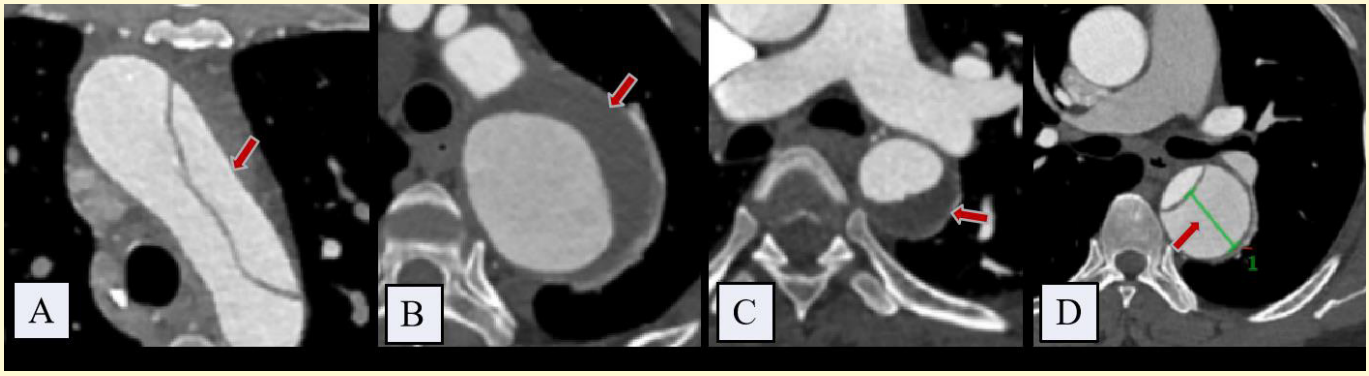


FIGURE 3. (A) demonstrates a false lumen along greater curvature with no evidence of thrombosis. (B) Demonstrates partial thrombosis in the false lumen. (C) Demonstrates complete thrombosis of false lumen (D) Demonstrates false lumen measuring 23mm. (False lumen >22mm indicates poor prognosis).

4. Branch vessel perfusion and visceral organ hypoperfusion - Static vs Dynamic. In static obstruction, the intimal flap enters the branch-vessel origin without a reentry point. In dynamic obstruction, the intimal flap spares the branch vessel but prolapses and covers the branch-vessel origin like a curtain during various phases of the cardiac cycle.

5. Complications: Hemopericardium, hemothorax, renal hypoperfusion and intimo-intimal intussusception.

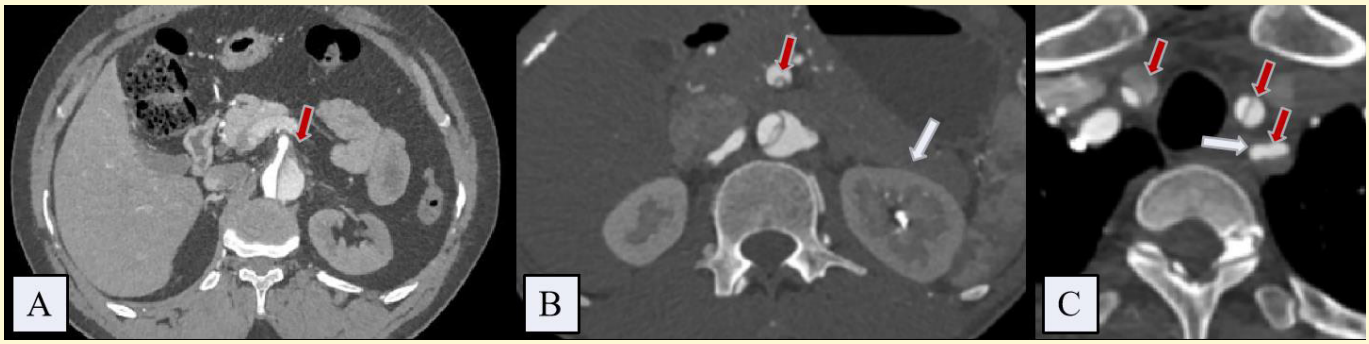


FIGURE 4. (A) Intimal flap seen extending into the superior mesenteric artery with partial thrombosis of false lumen (B) Left main renal artery is seen arising from false lumen with associated hypoperfusion of left kidney (shown as reduced contrast uptake), also demonstrates intimal flap within the superior mesenteric artery (C) Intimal flap seen extending into the brachiocephalic, left common carotid and left subclavian arteries with thrombosis of the false lumen of the left subclavian artery. The left vertebral artery is seen arising from the true lumen (white arrow).

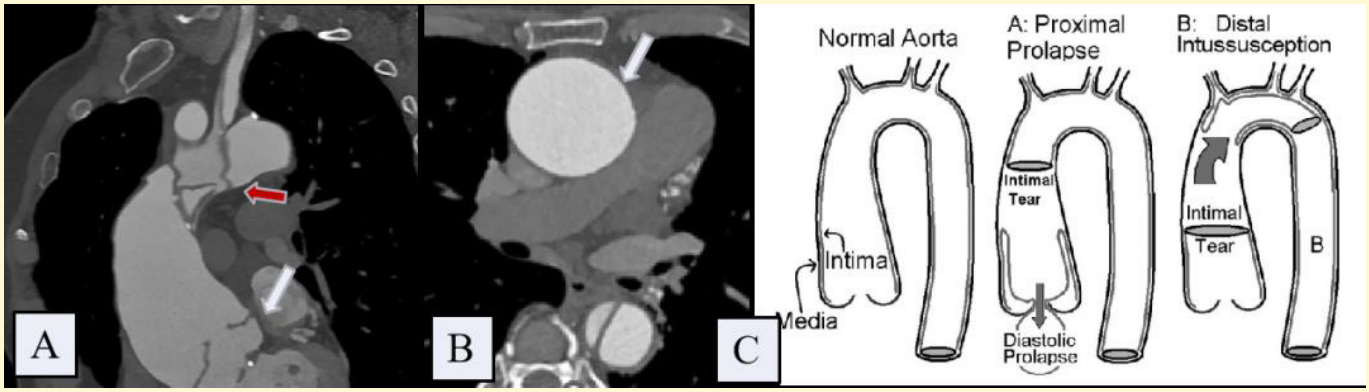


FIGURE 5. Intimo-intimal intussusception (A) Dissection flap seen proximally at the aortic root (white arrow) and distally at the proximal arch (red arrow) with no dissection flap seen in ascending aorta. (B) Dilated ascending aorta with no dissection flap visible. (C) Graphical image showing circumferential intimal tear with proximal/ distal intussusception of the flap (2).

INTRAMURAL BLOOD POOL AND PENETRATING ATHEROSCLEROTIC ULCER: Classification and management are similar to Aortic Dissection. PAU >10mm in width is associated with poor prognosis.

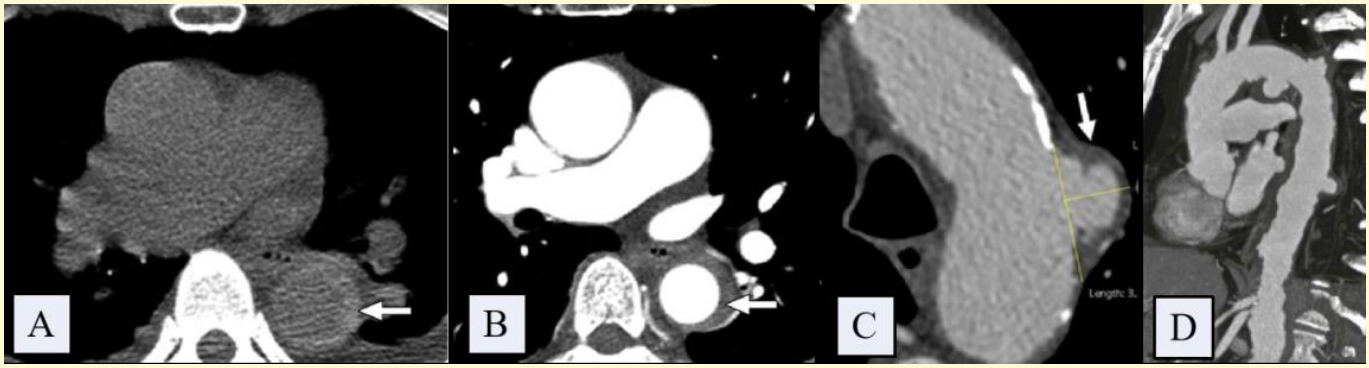


FIGURE 6. (A) Non-contrast CT. Stanford type B intramural hematoma appears as crescentic hyperdensity on NCCT and hypodense on post-contrast CTA (B). (C) Penetrating atherosclerotic ulcer along the greater curvature of arch of the aorta is seen as contrast-filled outpouching beyond the intima. (D) Oblique sagittal MPR image demonstrates multiple penetrating atherosclerotic ulcers along the arch and descending thoracic aorta.

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A CASE REPORT OF INTRAMUSCULAR MYXOMA:

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[1]Resident, [2]Assistant professor, [3]Assistant Professor, [4]Associate Professor, [5]Professor, [6]Professor & HOD, SVS Medical College

A 28 year old male patient presented with complaints of swelling in left leg. The swelling was painless and had gradually increased in size since its appearance 8 months prior to presentation to us. The swelling measured about 14 x 4.5 x 4 cm, firm in consistency and was mobile in the vertical plane.

NCCT:

An ill-defined hypodense lesion in proximal tibialis anterior muscle in left lower leg

SIGNAL INTENSITY OF LESION:

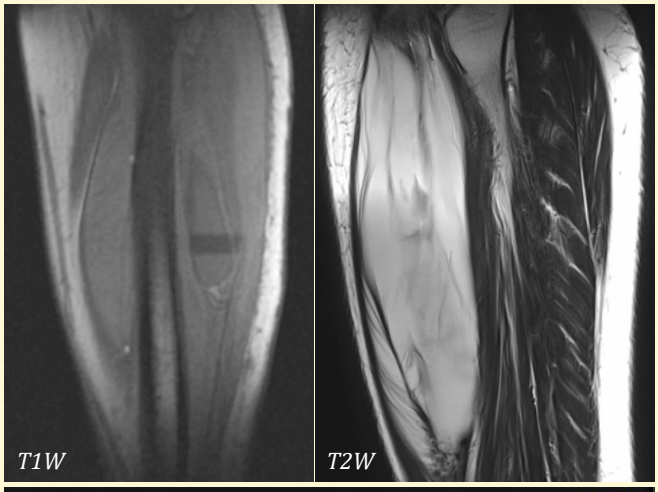
- TIW - Isointense.
- PDFS/T2W - Hyperintense signal intensity.
- Not showing restriction diffusion on DWI & reversal on ADC.

EXTENT OF THE LESION:

- Medially - The lesion is focally abutting medial cortex of tibia.
- Fascia cruris grossly appears intact.
- Minimal subcutaneous edema noted in anterior & lateral aspect of lower limb.

ON MRI:

A Large lobulated lesion with thick internal septations noted with epicentre at tibialis anterior muscle in proximal part of lower leg.



All above F/S/O - benign intramuscular lesion intramuscular myxoma.

HPE: SUGGESTIVE OF INTRAMUSCULAR MYXOMA

DISCUSSION:

Intramuscular myxoma is a rare benign tumor of the musculoskeletal system with an incidence ranging from 0.1 to 0.13 per 100,000 . Most intramuscular myxoma are solitary, painless, palpable masses that are firm in consistency and slightly movable and often fluctuant.It can occur in any location, but tends to involve the muscles of the thighs, buttocks, and shoulders. The current modes of imaging are CT and MRI. On CT, Intramuscular myxoma usually presents as a well demarcated homogeneous low density lesion within the skeletal muscle. On MRI, the lesion usually appears as low signal intensity on T 1 weighted and high signal intensity on T2 weighted images.

It is difficult to diagnose this tumor before biopsy and microscopic examination . Due to abundant myxomatous tissue and poor cellularity, it is difficult to make a diagnosis on FNAC. Macroscopically the tumor is oval or spherical in shape, it has a white or grey-white mucoid gelatinous surface. Although it appears well encapsulated, the delicate fibrous capsule is usually incomplete and most lesions show infiltration of adjacent musculature. On microscopic examination, there is abundant mucoid material and relative hypo cellularity and loose reticulin fibers. Vascular structures are sparse. The cells have a stellate shape with small hyper chromatic pyknotic nuclei and scanty cytoplasm.

IMAGING FEATURES:

On CT, Intramuscular myxomas appear as well-demarcated, homogeneous and hypodense ovoid lesions. The soft tissue mass attenuation is higher than that of water and less than that of surrounding tissue. They usually show mild diffuse enhancement or peripheral and septal enhancement seen in approximately 50% of cases.On MRI features include signal intensity that is hypointense to muscle on T1 and hyperintense on T2, intra-tumoral cysts in a small proportion of cases,pseudocapsule, perilesional/rim of fat and perilesional edema with bright cap sign.

DIFFERENTIAL DIAGNOSIS

The tumour is to be differentiated from peripheral nerve sheath tumors,myxofibrosarcoma, myxoid liposarcoma, lymphangioma, extra skeletal myxoid chondrosarcoma. Large size, high Signal on T2WI, heterogeneous enhancement, and internal fat component were more commonly observed inintramuscular myxoma, while homogenous enhancement, fat split sign, and target sign were more commonly seen in peripheral nerve sheath tumors .if the lesion is intramuscular and well defined an intramuscular myxoma is very likely. If a myxoid lesion is located in the extremities of a patient in 6th/7th decade and shows an ill-defined appearance a myxofibrosarcoma should be considered. If it is located in the distal extremities with subcutaneous involvement one should think of myxoinflammatory fibroblastic sarcoma (MIFS). The presence of fatty components should suggest a myxoid liposarcoma. Noncalcified large Lobulated tumors in the proximal lower extremities or limb-girdle regions should rise the thought of extraskeletal mvxoid chondrosarcoma. Surgical excision is curative. Recurrence is very rare. This existence of an intramuscular myxoma may be an indication for skeletal survey to detect previously silent fibrous dysplasia lesion which can be then followed up regularly.

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A CASE REPORT OF FOCAL NODULAR HYPERPLASIA

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A 20 year old female presented with complaints of right upper quadrant pain since one month. On examination a palpable mass was noted in the right upper quadrant.

NCCT:

Large ill-defined mild hypodense lesion seen involving segment V and VI of liver with superior extension into VII and VIII segments, measuring 10.5x10x7.5cm.

CT TRIPLE-PHASE LIVER PROTOCOL

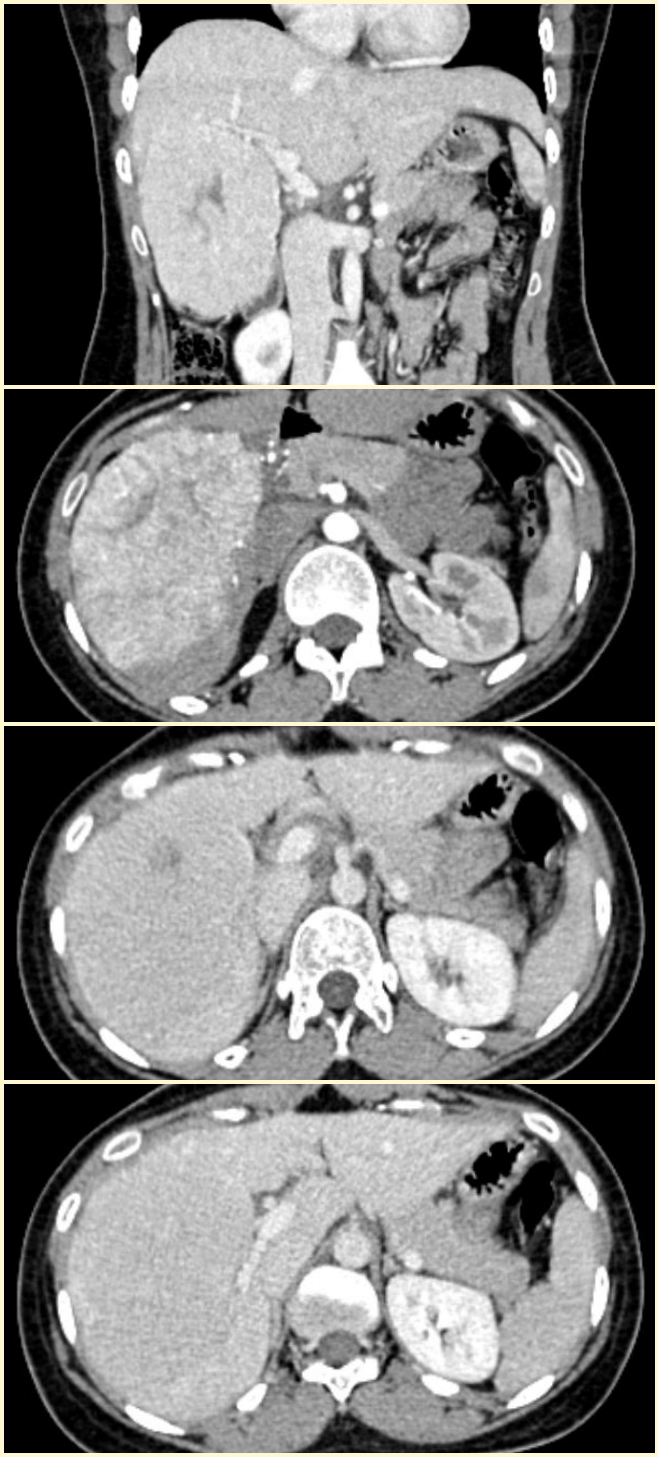
Post contrast study: The lesion shows avid enhancement on late arterial and porto-venous phases with non-enhancing central stellate hypodense area. The mass appears circumscribed with lobulated margins.

In delayed phase the central stellate area appears hyperdense.

NON-CONTRAST MRI

On T1W images the lesion is iso to hypointense with hypointense central scar. On T2W images the lesion is isointense with hyperintense central scar and not showing restriction on DWI.

HPE suggestive of Focal Nodular Hyperplasia(FNH).



Discussion

Liver is the only regenerative internal organ in the body, with an inherent risk for developing atypical masses.

Previously, FNH was referred to by a variety of synonyms including pedunculated adenoma, solitary hyperplastic nodule, focal cirrhosis and hepatic hamartoma.

The etiology of focal nodular hyperplasia has not been definitely established, however it is thought to be caused by arterial malformations within the liver. These malformations coupled with changes in perfusion, cause a regenerative, hyperplastic response of the normal hepatocyte. Interestingly, hepatocytes may respond with hyperplasia after both hypoperfusion and hyperperfusion.

Hereditary hemorrhagic telangiectasia also known as osler-weber-rendu syndrome can cause an increased incidence of FNH.

The incidence of FNH also increases in the presence of hemangiomas.

In adults the most common benign hepatic lesion is the hemangioma, however FNH is the second most common accounting for approximately 8% of all non-hemangiomatous liver lesions. FNH can present as early as childhood, however it disproportionately affects women more than men at a ratio of approximately 8:1.

The incidence of FNH is increased in females age 20-50, further suggesting that the condition may be linked to increased estrogen.

Imaging features

On CECT-

In the early arterial phase, focal nodular hyperplasia will enhance relative to background liver. Typical focal nodular hyperplasia shows early arterial centrifugal filling (from the center outwards). Prominent feeding vessel may be seen in late arterial phase (opposite to hemangioma and adenoma). Portal venous phase-There is sustained enhancement in the portal venous phase (as opposed to adenoma) with unenhanced scar may be present.

ON MRI-

T1 the lesion is iso to moderately hypointense with hypointense central scar. T2 the lesion is iso to somewhat hyperintense with hyperintense central scar.

Differential diagnosis

Hepatic adenoma: usually more heterogeneous CT portal and delayed phases contrast washout; no gadoxetate retention on delayed phase MR and associated with fat, calcification or hemorrhage.

Hepatocellular carcinoma: usually in cirrhosis; vascular invasion.

Fibrolamellar (FL) hepatocellular carcinoma -both focal nodular hyperplasia and fibrolamellar hepatocellular carcinoma commonly have a hypointense “central scar” representing fibrosis, so this feature is less useful for differentiation. Fibrolamellar hepatocellular carcinoma tends to appear more distinct from adjacent liver parenchyma on pre-contrast T1 / T2-weighted imaging. Often larger (>12 cm). Calcification (uncommon in focal nodular hyperplasia), corresponding to necrosis and foreign body type reaction histopathologically. 70% present with metastases, or evidence of biliary, vascular, and nodal invasion. Decreased activity

on Tc-99m / sulfur colloid scan. Hypervascular hepatic metastases: usually multiple; CT portal and delayed phaseshypodense (washout); older patients with known primary tumor. Hepatic hemangioma: peripheral and centripetal enhancement; blood vessels isodense; no central scar; only small ones with rapid enhancement simulate focal nodular hyperplasia. Intrahepatic cholangiocarcinoma: hypoenhancing in earlier arterial/venous phases with delayed enhancement, dominant large central scar

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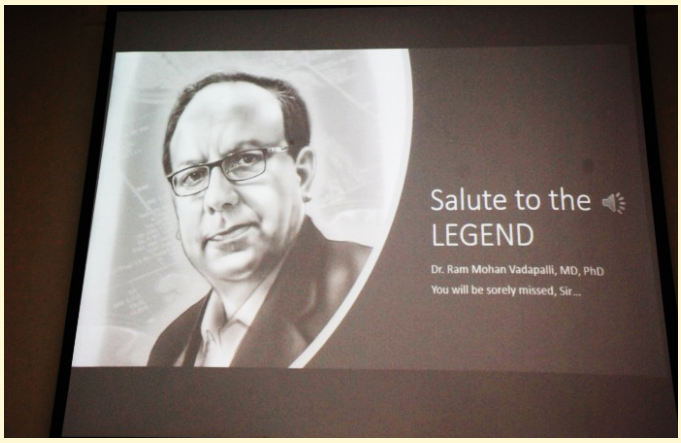
ACADEMIC ACTIVITIES OF IRIA TS CHAPTER

7th KARE on 15th & 16th April, 2023 at Basavatarakam Indo American Cancer Hospital and Research Institute



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Monthly Meeting on 20th May 2023 at Century Hospital

Advanced Joint Imaging & Interventions Hands On-Cadaver Workshop on 24th & 25th June 2023 at Mamata Academy of Medical Sciences









UPCOMING CMES

1. ***August -2023***
   Monthly Meeting
2. ***September-2023***
   Monthly Meeting
3. ***September Last Week Webinar***
4. ***13th, 14th & 15th October 2023***
   9th State Annual Conference (INDO US IMAGING UPDATE ON RECENT ADVANCES IN ONCO IMAGING)
5. ***08th November- 2023***
   IDOR Day Celebrations (Monthly Meeting)
6. ***19th November-2023***
   Outreach Program
7. ***November – 2023***
   7th Radiology Anatomy Course (RAC)

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