IRIA Telangana **CONTROLATION CONTROLATION CONTROLATION**



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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 Members of IRIA Telangana,

I am delighted to extend a warm invitation to our Annual Conference, a momentous event for IRIA Telangana, scheduled from 13th to 15th October in Hyderabad. This year's theme, "Recent Advances in Onco Imaging, Indo-US Imaging Update," promises insightful discussions, enriched by the presence of four distinguished speakers from Moffitt Cancer Center, Florida, USA.

I am thrilled to announce that this conference marks our reunion in Hyderabad after a hiatus of 4 years, and the response has been overwhelming. With more than 300 registrations, we anticipate engaging sessions and around 200 papers and posters presented from post graduate students.

In addition to the stimulating conference sessions, we have organized two workshops that will provide learning experiences for our attendees.

I am delighted to share that we are honored to have the esteemed presence of Dr. (Smt.) Tamilisai Soundararajan

Hon'ble Governor of Telangana and Hon'ble Lt. Governor of Puducherry as the Chief Guest for the inaugural program. Her esteemed presence will not only lend prestige to our event but also motivate us to strive for excellence in the field of Radiology.

I express my heartfelt gratitude to Dr. Prabhakar Reddy for his invaluable contributions and guidance that have made this event possible.

I also extend my thanks to Dr. Rajesh and the entire AIG Hospital Radiology team for their generous support throughout the conference.

Furthermore, I am excited to invite all residents to participate in our upcoming Conventional Radiology One-Day Conference on 26th November at Kamineni Institute of Medical Sciences, Narketpally.

Let's make this conference a resounding success, showcasing the strength of our community and the brilliance of our members. Together, let's uphold the spirit of IRIA.

Long live IRIA, and Jai Hind!

Warm regards,

Dr. P Krishna Mohan General Secretary, IRIA Telangana

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BREAST CANCER SCREENING - THE INDIAN SCENARIO

Breast cancer is the leading cause of cancer in Indian women. As per the Globocan2020 data, in India, breast cancer accounted for 13.5% of all cancer cases and 10.6% of all deaths with a cumulative risk of 2.81.According to the National Cancer Registry, the number of Indian women diagnosed with breast cancer in 2020 was estimated at 2 lakhs per year and the number is likely to reach 2.3 lakh by 2025. Current trends point out that a higher proportion of the disease is occurring at a younger age in Indian women, as compared to the West.

Breast cancer screening- why is it important

Screening for breast cancer has been proven to be very effective in that it decreases cancer related mortality by 30%. The essence of breast cancer screening is simple yet profound – the earlier the detection, the better the prognosis. A robust screening program can translate into early intervention, less invasive treatments, and improved patient outcomes, ultimately enhancing the quality of life.

Unlike the Western countries, there is no established comprehensive screening program in India resulting in 60% of breast cancer cases being diagnosed at stage III or IV of the disease

Mammography, clinical breast examination, and breast self-examination together constitute the primary arsenal of screening tools. Resource constraints, size of the population and lack of awareness make comprehensive screening a challenging process in India. Nevertheless, opportunistic screening of interested women can be performed at many centres / Institutes, as a step towards achieving the same goal.

Screening Protocols in India – Guidelines

The American College of Radiology has categorised women into average (< 15% risk), intermediate risk (15-20%) and high risk (>20%) groups based on their lifetime risk of developing breast cancer and has set screening protocols.Taking into purview, the cancer burden in our society, increasing numbers of younger women being afflicted and our sociocultural challenges, the Breast Imaging Society of India adapted these guidelines for breast cancer screening and diagnosis in India.

Mammography (with digital breast tomosynthesis-DBT, wherever possible) is the first line of imaging for screening. DBT provides 3D views of the breast resulting in improved sensitivity owing to reduced tissue overlap. This also results in decreased recall rates and enhanced cancer detection.

Annual screening for women with average risk of cancer should begin at the age of 40years and continue till 70 years. Beyond 70 years, screening would depend on overall life expectancy based on co morbidities and the patient's choice.In high risk women, such as carriers of genetic mutations, first degree relatives of breast cancer patients, history of radiation during childhood, etc.; the screening should begin at the age of 30 years with an addition of MRI every 6 months.Intermediate risk group constituted by women with personal history of cancer, lobular neoplasia and high risk breast lesions,annual surveillance is recommended even when age is<40years.

Ultrasound is widely used for evaluating breast symptoms in India owing to multiple reasons- easy

availability of ultrasound imaging in the rural as well as urban areas, less expensive and does not need specific training or special equipment. However, it must be stressed that it is not a screening tool for breast cancer. It can be used as a first line of imaging in younger women presenting with breast lump/pain/nipple discharge and also in lactating and pregnant women. It can be used as an adjunct to mammography in dense breasts where the sensitivity of mammography decreases significantly. Diagnostic mammography should be performed even in younger women with a suspicious lump on ultrasound.

MRI of breast , owing to its high sensitivity and negative predictive value is indicated for selected reasons such as screening in highrisk population, assessing the extent of tumour for planning of breast conservation surgeries, response to neoadjuvant chemotherapy, ominous nipple discharge with negative mammography/USG, metastatic axillary lymphadenopathy with unknown primary and as a problem solving tool in ambiguous mammography findings.MRI should not be used as an imaging modality to decide whether a BIRADS 4-5 lesion requires biopsy.

Image guided Biopsy or FNAC – The big dilemma

In most parts of India, owing to the lack of trained radiologists, lack of awareness and ease of procedure, FNAC of breast lumps in a common practice. It has been established and advised to perform image guide biopsy using a wide bore (14G) gun for breast abnormalities as the chances of false negatives is less and results in increase in confidence of pathology reports. Also, with cancer treatment becoming more and more dependent on immunohistochemistry and molecular markers, biopsy is the standard of care. FNAC can be performed for metastatic nodes and synchronous cancer in multifocal malignancies.

It is also very important that a multidisciplinary team is involved in breast cancer diagnosis and treatment so that personalised and optimal treatment protocols can be followed for enhanced and empathetic patient care.

The Radiologist's Odyssey: Challenges Galore

Indian radiologists are the linchpin of breast cancer screening, yet they grapple with formidable challenges. A severe shortage of radiologists, especially in rural areas, results in limited access to screening services. The demand for specialized training and state-of-the-art equipment compounds these hurdles. Moreover, the accurate interpretation of mammograms is a demanding task that necessitates ongoing skill refinement and stringent quality assurance.

Pioneering a Holistic Solution

To overcome these challenges, a multi-faceted strategy is imperative. Government bodies and healthcare organizations must invest heavily in infrastructure development and radiologist training throughout the nation. This includes expanding the accessibility of mammography machines and instituting stringent quality control measures. Public awareness campaigns must be intensified to destigmatize breast cancer and stimulate demand for screening.

Embracing emerging technologies such as artificial intelligence (AI) could significantly bolster the capabilities of radiologists. AI-driven algorithms have demonstrated great promise in augmenting early breast cancer detection, and their integration into screening protocols warrants thorough exploration.

In conclusion, breast cancer screening in India carries profound significance in mitigating the disease's impact. While challenges are manifest, collaborative efforts by healthcare stakeholders, fortified by technological advancements, hold the key to bridging the gap between Indian and international screening standards. It is incumbent upon us to work collectively to ensure that every woman in India gains access to timely and effective breast cancer screening.

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ACHIEVEMENTS

Delighted to share Dr Sikander Shaik appointed as member of Society of Pediatric Radiology International.

1. SPR EDUCATION and CURRICULUM COMMITTEE 2. SPR MR COMMITTEE

Both committees for 2023-2024. Dr Sikander is the only member outside USA and CANADA Hearty congratulations Dr Sikander for this wonderful academic achievement



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Hearty congratulations Dr Sikandar for being elected as President of ASMRM

Quiz Winners in Monthly Meeting on 25th August, 2023



1st Winner Dr. G Bhavya NIMS



2nd Winner Dr. Ramya Osmania Medical College



3rd Winner Dr. Syed Maqsood NIMS

21st ANNUAL SCIENTIFIC MEETING OF ASIAN AND OCEANIC SOCIETY FOR PAEDIATRIC RADIOLOGY





Congratulations to all the participants in the 21st Annual Scientific Meeting of Asian and Oceanic Society for Paediatric Radiology, from Apollo Medical College Happy to share that the much awaited 8th edition of David Sutton's Textbook of Radiology and Imaging is finally ready. Super proud to be associated with this project as an Associate Editor and contributor for 2 chapters.



Dr. Varsha Joshi Senior Member of IRIA President of ISHNR Senior Consultant Radiologist Vijaya Diagnostic Center



Dr. Srinivasarao Gummadidala,

Associate Professor & Vascular Interventional Radiologist (Endovascular Surgeon), Shruthi Super Specialities, Vijayanagar Colony, Hyderabad-500057, & Dr. VRK Womens Medical College and Research Centre, Hyderabad, Congratulations to Dr. Srinivasarao Gummadidala for the Award in connection with-Top 10 Vascular and Endovascular Surgeons in the state of Telangana for the year 2023 conducted by HMTV



ARTICLES



Dr Sarika Bolenwar Consultant Radiologist, Vijaya Diagnostic Center, Hyderabad

MR IMAGING IN EVALUATION OF SHOULDER INSTABILITY

Shoulder is a structurally simple, but biomechanically a complex, multiaxial, spheroidal type of joint formed by the articulation of glenoid fossa of the scapula with the head of the humerus. It has got a widest range of mobility than any other joint in the body but at the cost of its vulnerability to injury and development of instability.

Shoulder instability is defined clinically as symptomatic humeral head displacement from bony glenoid and its abnormal sequelae. Patients with shoulder instability present to a clinician either with typical history of prior dislocation or sometimes may present with chronic pain or disability.Pain produced by unstable shoulder has wide range of clinical differentials pathologies like shoulder impingement with or without rotator cuff tears, AC joint pathologies, cervical disc disease.

MRI is a noninvasive modality for evaluating these group patients to assess various pathologies resulting from recurrent dislocations or subluxations.

Anteroinferior instability is most common pattern of instability, accountsfor in 95% cases, whereasPosterior,inferior, superior and multidirectional accounts for remaining 5% .Depending on cause of instability it is divided into two main types, traumatic and atraumatic. In traumatic etiology, most common pattern is fall on an outstretched hand (FOOSH) or can be seen in acute injury in athletes colliding with other players as in football or rugby players, repeated high- load activities or resulting from chronic repetitive microtrauma. Atraumatic etiology could be congenital e.g., dysplastic glenoid, capsularligamentous laxity or acquired due to overstretching activities like in swimming and gymnastics.

Thomas and Matson's classification of instability iswidely used by clinicians.This classification isbased on directionality of instability, mechanism of injury and preferred method of treatment. Two main categories in this classification includeTraumatic unidirectional instability treated with Bankart's repair (TUBS) and other is Atraumatic multidirectional instability treated with rehabilitation (AMBRI).

MR imaging provides valuable information to show osseous and soft-tissue abnormalities that can guide surgical planning and the choice of stabilization procedure.



Figure 1: Axial PD FAT SAT sequence (a) depicting normal uniformly low signal intensity anterior (black arrow) and posteriorlabrum(white arrow) Coronal PD FAT SAT (b) depicting normal uniformly low signal intensity superior (black arrow) and inferior labrum(white arrow).

There are three different clinical scenarios in which patients withshoulder instability present for MR imaging. First group is Acute first-time shoulder dislocation, second is Chronic instability with repeated dislocation and third is Chronic instability without repeated dislocation. (Multidirectional instability, overuse injuries in athletes would comprise these group)

Various osseous and labral lesions associated with Acute First-Time Shoulder dislocation.

- Bone lesions
- Hill Sachs lesion. Impaction fracture of posterosuperior aspect of humeral head.
- Avulsion fracture of bony glenoid.
- Fracture of coracoid process
- Fracture of greater tuberosity- can be associated with cuff strain
- Fracture of lesser tuberosity uncommon. If associated with significant tear of subscapularis specially in elderly may preclude surgery.
- Labral lesions
- Soft tissue Bankart Cartilaginous / fibrous
- HAGL- Humeral avulsion of inferior glenohumeral ligament.
- Perthes lesion
- ALPSA lesion- Anterior labral periosteal sleeve avulsion.
- The most common labral injury following firsttime traumatic dislocation is a Bankart lesion.
- It is the classic pathoanatomic hallmark of anterior instability that Bankart described as the "essential" lesion in recurrent shoulder dislocation.



Fig 2: Axial PD FAT SAT (a) depicting Hill-Sachs lesion with associated subjacent marrow oedema in humeral head (white arrow) associated with anterior labral tear (black arrow), Sagittal PD FAT SAT(b) depicting Hill-Sachs lesion (white arrow)

Perthes lesion: It is also known as nondisplaced Bankart lesion. Less common lesion, reported 8% incidence in first-time dislocation. The labrum is detached from the glenoid surface but stays well anchored to the periosteum, which remains continuous and is only minimally medially stripped. Perthes lesion may be subtle on axial images, it may become more apparent on ABER images owing to the traction provided by the inferior glenohumeral ligament on the labrum.Perthes lesions can heal spontaneously through the synovial membrane, granulation tissue and periosteal fibers, so there may be no clinical findings despite symptoms of shoulder instability.The lesion may also be difficult to diagnose during arthroscopy. Imaging play's critical role in diagnosis of this lesion.



Fig 3 : axial PD FS (a)depicting Osseous bankart lesion (black arrow), sagittal T2(b)(black thick arrow)



Fig.4: Perthes lesiona(white arrow)



Fig 5: Perthes lesion seen as a subtle finding Axial PDFS (a), on ABER manoeuvre (b) Perthes lesion is clearly depicted as fluid signal intensity tear in anteroinferior labrum(white arrow)

GLAD lesion: Glenoid labral articular disruption is anondisplaced anteroinferior labral tear with an associated anteroinferior glenoid articular cartilage injury.It results from humeral head impaction against glenoid rather than from dislocation and is usually a stable lesion.Classic description of the GLAD lesion includes patients experiencing pain rather than clinical instability. It's a rare injury, occurs primarily in athletes.Intra-articular loose bodies are commonly identified.Treatment includes Arthroscopic debridement of labral and chondral defects.



Fig6: GLAD lesion a(white arrow)

HAGL lesion: Humeral avulsion of anterior band of inferior glenohumeral ligamentcan result in glenohumeralinstability.In the acute setting, IGL injury is associated with periarticular edema and hemorrhage that localize to the axillary pouch, quadrilateral space and proximal humerus. The "J" sign (in a right shoulder; reversed "J" in a left shoulder) is fairly specific for IGL rupture when edema and hemorrhage outline the torn, retracted stump of inferior glenohumeral ligament.At arthroscopy or open surgery, the HAGL lesion can remain undiagnosed.Treatment consists of repair of torn ligaments.



Fig 7: Coronal PDFS (a) depicting HAGL lesion

ALPSA lesion: Anterior labral periosteal sleeve avulsion(ALPSA) lesion is often the result of chronic injury.However, it has been reported that the incidence of ALPSA lesions in first-time dislocators is similar to the incidence of Bankart lesions.ALPSA lesion can heal in this displaced position, creating a small cleft between the labrum and the glenoid. In a chronic ALPSA lesion, there are proliferation and deposition of fibrous tissue on the displaced labroligamentous complex, resulting in scarring along the articular surface.ALPSA lesions are more complex and have a worse prognosis than Bankart lesions, their chronic nature, in combination with a twofold higher bone loss duringsurgery.Treatment involves arthroscopic conversion to true Bankart lesion, followed by repair.



Fig 8: Axial PDFS(a) depicting ALPSA lesion(White arrow)

Posterior instability: Accounts for 2-4% cases of shoulder instability. Can be seen in violent muscle contractions as in seizures or electric shock.The lesions associated in this pattern are Reverse Bankart lesion that is tear of posterior inferior labrum and Reverse Hill sach's lesion which is an anterosuperior medial humeral head fracture. Excessively retroverted or hypoplastic glenoid and capsular laxity have been reported in recurrent posterior dislocations.

Paralabralcysts: Seen in high association with glenoid labral tears. Most frequently occur along the posterior, anterior, and superior aspects of the glenohumeral joint in decreasing order, with inferiorly located cysts being the least common. They may cause a compression neuropathy of the suprascapular or axillary nerves.

Traumatic dislocation can lead to chronic instability and repeated dislocations with lesser degrees of force and provocation.Role of imaging shifts from lesion detection and accurate assessment for treatment planning. Depending on the presence and severity of osseous versus soft tissue lesions, the surgeon decides on open surgical versus arthroscopic visualization. The primary goal of the stabilization procedure may be the reconstruction of humeral and glenoid osseous defects or the repair of anteroinferior capsulolabral soft tissues The glenoid rim can become flattened and deficient due to fracture, bony remodeling or a combination of both.



Fig 9: Sagittal PDFS (a) depicting anteroinferior paralabral cysts (white arrows), axial PDFS (b) depicting superior paralabral cyst(white arrow)

Image interpretation should take into account the size of the glenoid lesion and the contour of the glenoid rim.The amount of glenoid bone loss guides management. 3D CT is BEST to assess for % of glenoid loss, also for depicting small osseous fragments. MRI is equally good.Bone loss/ diameter of intact glenoid x100 gives the percentage of bone loss. It is considered to be significant bone loss when it is more than 15%.Usually managed with surgical intervention.



Assessment of glenoid on track versus off track lesion is essential in planning for type of surgical versus arthroscopic management and acts as a guide for patient management. Glenoid track is a Part of glenoid in contact with humerus in abduction and external rotation. 83% of intact glenoid subtracting glenoid bone loss is the glenoid track. The lesion is said to be on track or nonengaging lesion where Hill sach's interval < glenoid track and off track lesion or engaging lesion where Hill sach's interval > glenoid track. With regard to the choice of MR imaging versus MR arthrography for evaluation of the glenoid labrum, patient presentation plays a crucial role.Patients with acute symptoms or severe pathologic lesions are more likely to have intrinsic image contrast in the form of effusion or soft-tissue changes that allow diagnosis and characterization without an invasive procedure. On the contrary, those with chronic symptoms or a pathologic abnormality that is suspected to be more subtle on the basis of clinical assessment more often require MR arthrography. To conclude Glenohumeral instability encompasses a broad spectrum of clinical complaints and presentations.Imaging findings depend on the clinical scenario.Imagingabnormalities that occur in the acutely traumatized shoulder are substantially different from those of the chronic unstable joint. Image interpretation and reporting need to emphasize the identification of lesions for diagnosis, further assessment / differentiation of lesions for treatment planning.

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BRAIN TUMOUR FUNCTIONAL IMAGING

Nuclear Medicine makes use of radioactive isotopes for diagnosis and treatment of diseases. The use of Nuclear Medicine imaging in brain tumours is an evolving field and with time new and new radioisotopes are being used. The last decade had seen tremendous growth of PET-CT imaging and multiple new radiotracers for brain tumour imaging. We can classify brain tumour imaging radiotracers as (1,2):

- Gamma camera-based radiotracers most commonly used are Technetium 99m -Glucoheptonate (Tc99m-GHA), Tc99m-Dimercaptosuccinic acid - V (Tc99m-DMSA V).
- ii. PET based radiotracers most commonly used are Fluorine -18 Fluorodeoxyglucose (F-18 FDG), F-18 Fluorodopa (F-18 FDOPA), F-18 Fluorothymidine (F-18 FLT), Galluim-68 Prostate Membrane specific agent (Ga-68 PSMA) etc.

There are multiple other, not so commonly used, radiotracers like Carbon -11 Methyl-Methionine (C-11 MET), C-11 Choline (C-11 Cho), F-18 fluoroethyl-L-tyrosine (F-18 FET), F-18 Fluoro-thymidine (F-18 FLT), F-18 fluoro-misonidazole (F-18 FMISO), Cu-64 diacetyl-di(N4-methylthiosemicarbazone) (Cu-64 – ATSM) etc (3).

The most common indications pertaining to Brain tumours presenting to Nuclear Medicine department for imaging are: i. Gliomas, ii. CNS lymphoma, iii. Metastatic lesions and iv. Post radiotherapy differentiation of radiation necrosis v/s tumour recurrence.

 GLIOMA: It is worthwhile mentioning that F-18 FDG shows increased radiotracer uptake (hypermetabolism) only in the Highgrade Glioblastoma (Grade - IV) (Image.1)

and shows no/reduced radiotracer uptake (hypometabolism) in Grade I, II or III Gliomas. The F-18 FDG is taken by actively dividing tumour cells as energy substrate. So, the FDG uptake is also helpful for prognostication of the lesion as greater the FDG uptake, poorer is the prognosis and vice versa. For imaging of Grade - I, II or III Gliomas, also known as Low Grade Gliomas (LGG), many newer radiotracers are being used, most common of these is F-18 FDOPA (4). Normally F-18 FDOPA is used for diagnosing Idiopathic Parkinson's disease due to preferential uptake in basal ganglion and very low-grade tracer uptake in rest of the brain. However, it was found to have increased FDOPA uptake in Low Grade Gliomas (Grade I-III) and now is most commonly used for Brain SOLs when there is a diagnostic dilemma between demyelination or Low-Grade Glioma. Other radiotracers like C-11 Methionine show hypermetabolism due to increased demand of amino acid by actively dividing tumour cells.

2. CNS Lymphoma: The most common histological variant of Primary CNS Lymphoma is B-cell Lymphoma (5). As any other B-cell Lymphoma in the body, the CNS Lymphoma shows intense hypermetabolism on imaging with F-18 FDG when compared to surrounding brain parenchyma. The FDG uptake is described as "Hypermetabolism glaring at you" (Image 2). Almost all types of CNS lymphoma show increased FDG uptake. Usually, while performing F-18 FDG imaging for Primary CNS Lymphoma, the whole-body imaging is also done to rule out any other area of involvement. Since F-18 FDG shows intense hypermetabolism, it makes it

the imaging agent of choice and not many other radiotracers have been tried in this regard.

- Metastatic disease: It is considered to be the 3. most common malignant cause of brain SOLs (6). F-18 FDG study has a distinct advantage that with dedicated brain study, a whole-body imaging is also performed which is helpful in diagnosing the primary site. The most common malignancies metastasizing to brain are lung, breast, thyroid (Image.3), melanoma etc. The F-18 FDG brain uptake in these metastatic lesions is very variable and depends on the actively dividing cells. However, as the energy substrate of brain is glucose, there is inherent FDG uptake in grey matter, making it difficult to visualize all the metastatic lesions and MRI is considered the investigation of choice in metastatic brain lesions.
- 4. Residual Disease v/s Radiation Necrosis: The treatment of brain malignant lesion includes excision which can be total excision or subtotal excision followed by radiation therapy. Post radiotherapy, it becomes challenging to differentiate between residual/recurrent disease and radiation necrosis on MRI imaging. Previously F-18 FDG was used for this purpose, but due to inherent FDG uptake in gray matter, it was difficult. Now, with use of newer radiotracers like F-18 FDOPA, which are more specific in diagnosis of residual/recurrent disease and show little uptake in radiation necrosis, the imaging shows higher sensitivity and specificity in differentiating between residual/recurrent disease and radiation necrosis (7). Moreover, FDOPA has very little physiological gray matter uptake making visualization of tracer in residual disease/recurrence easier (Image 4). Another tracer commonly used is Ga-68 PSMA which targets the neo-vascularization present around the residual/recurrent disease and absent in radiation necrosis (8). It per-se does not image the lesion directly. The Ga-68 PSMA imaging helps in accurate target delineation, hence more precise Gross tumor volume (GTVs) leading to less RT-induced side effects (8).

Due to advancement in PET imaging characteristics in terms of better image quality, better target-tonoise ratio leading to better visualization of the lesions, the Tc99m based imaging of brain tumours are more of an historical importance and are rarely used now-a-days. However, in Imaging centers equipped with only Gamma camera, it can still be used. Both Tc-99m GHA and Tc99m DMSA (V) show no physiological brain uptake and takes the advantage of tumour induced breach in blood brain barrier to reach the malignant lesion. However, the sensitivity and specificity are low when compared to PET imaging agents (9).

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Image 1: A 52 year female presented with headache, occasional vomiting and weakness of right upper limb. MRI showed suspicious brain lesion in left fronto-temporal region. PET-CT study showed solid-cystic lesion in left fronto-temporal region with FDG uptake in solid component and central necrotic component. The FDG uptake in lesion was mildly raised compared to other areas of brain parenchyma raising the possibility of Gliobastoma (Gr. IV). Patient underwent left fronto-temporal craniotomy and final histopathology was confirmed as High grade Gliobastoma.



Image 2: A 48 year male presented with headache, confusion and weakness of all limbs. MRI showed brain SOL in left capsulo-ganglionic region. PET-CT study showed mildly enhancing predominantly solid lesion in left capsule-ganglionic region with intensely increased FDG uptake which was much more when compared to other areas of brain parenchyma raising the possibility of lymphoma. The whole body PET-CT study was negative for any metabolically active disease in the body raising possibility of Primary CNS Lymphoma. The Patient underwent brain biopsy and final histopathology came as Non-Hogkins Lymphoma (DLBCL).



Image 3: A 35 year female presented with severe headache, sudden onset hemiparesis and not able to perform daily activities. MET Brain showed multiple peripherally enhancing lesions in brain -?infective/?metastatic. The patient underwent whole body PET-Ct with dedicated Brain study which showed multiple peripherally enhancing lesions in brain, some showing FDG uptake in solid component. In addition, a intensely FDG avid hypodense nodule was seen in right lobe of thyroid gland raising the possibility of thyroid malignancy with brain metastasis. Patient underwent thyroid FNAC which came out to be Papillary carcinoma of thyroid – Follicular variant.



Image 4: A 25 year male, known case of anaplastic astrocytoma, post left temporal craniotomy with gross total excision of tumor followed by radiotherapy, now presented with occasional severe headache. MRI was suggestive of radiation necrosis. Due to persistent symptoms, patient underwent F-DOPA PET-CT study which showed a small focus of FDOPA uptake near the left temporo-occipital junction suggestive of recurrence.

INTERESTING CASES



A CASE REPORT ON LEIGH SYNDROME:

Dr. Devika Badimi¹, Dr.Sandeep Madineni², Dr.Doni Subhashreddy³, Dr.Geethika Mandepudi⁴, Dr.K.VenkatramReddy⁵, Dr.G.Ramakrishna Reddy⁶ ¹Resident, ²Associate professor, ³Associate professor, ⁴Associate professor, ⁵Professor,

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Dr. Devika Badimi Resident SVS Medical College

A 4 month old female child with history of normal institutional term vaginal delivery presented with excessive irritability and hypotonia for the past 15 days. No history of fever. Arterial blood gas and cerebrospinal fluid examination showed elevated lactic acid.



Symmetrical T1W Hypointense, T2W Hyperintense signals noted in bilateral head of caudate nuclei, lentiform nuclei (globus pallidus & putamen) and bilateral cerebral peduncles and substantia nigra of midbrain.

MR Spectroscopy showed increased choline, decreased NAA and elevated Lactate.

Based on MRI findings, the clinical signs and the increased lactic acid the presence of Leigh Syndrome was the most likely diagnosis.

DISCUSSION:

Leigh syndrome (also termed subacute necrotising encephalopathy) is a progressive neurodegenerative disorder of childhood with an estimated incidence of 1:40,000 births & is the clinical result of



Restricted diffusion noted on DWI with reversal on ADC. No bloomings noted on SWI

heterogeneous biochemical abnormalities in the mitochondria. Though the exact etiology is still debatable, numerous mutations have been described in the various enzymes of the respiratory chain. The involvement of pyruvate dehydrogenase complex, cytochrome oxidase, complex V, thiamine deficiency, succinate dehydrogenase deficiency, and complex I deficiency are some of the causes of Leigh's disease.

Most of the patients present chronically with a possible acute presentation during metabolic decompensation. Affected infants and children present typically toward the end of the first year of life. Hypotonia and psychomotor deterioration is the usual presentation. Ataxia, ophthalmoplegia, ptosis, dystonia, and swallowing difficulties are the usual sequelae. Pathologic abnormalities described in Leigh's include neuronal loss, vascular proliferation, microcystic cavitation, and demyelination in the midbrain, basal ganglia, cerebellar dentate nuclei, and cerebral white matter. Some patients also present later in childhood or adulthood.

Leigh syndrome is the most common clinical phenotype of mitochondrial disorders in childhood. The diagnostic criteria are (1) progressive neurological disease with motor and intellectual developmental delay; (2) signs and symptoms of brainstem and/or basal ganglia disease; (3) raised lactate levels in blood and/or cerebrospinal fluid (CSF); and (4) characteristic symmetric necrotic lesions in the basal ganglia and/or brainstem.

IMAGING FEATURES:

On imaging, Leigh's disease has predominant gray matter involvement with associated white matter involvement. MRI is the imaging investigation of choice for all the metabolic and developmental disorders involving the brain as it can characterize the lesion with more confidence than neurosonography and CT. CT of the affected patients shows hypodense lesions in the affected regions which do not enhance on contrast. Calcification and hemorrhage are not described in Leigh's disease. The imaging findings tend to vary with the mutation that is the cause of the syndrome. Diffusion characteristics vary with the stage of the disease.

The most characteristic neuroradiological findings in Leigh syndrome are bilateral, symmetric focal hyperintensities in the basal ganglia, thalamus, substantia nigra, and brainstem nuclei at various levels on T2-weighted MRI. These high T2 signals on MRI reflect the spongiform changes and vacuolation in the affected brain structures. Often, the basal ganglia are affected before the brainstem. The upper brainstem followed by lower brainstem would be affected with the progression of the disease. Involvement of lower brainstem indicates advanced stage of the disease and the occurrence of respiratory failure and sudden death. In most patients the cerebral white matter is generally only involved in late stages of the disease. Occasionally, patients may have atypical neuroimaging features such as diffuse supratentorial leukodystrophy, unifocal or multifocal infarctions, diffuse or focal cortical atrophy, or predominant cerebellar atrophy.

Magnetic resonance spectroscopy has shown decreased N-acetyl aspartate and elevated lactate peaks. MR spectroscopy findings are more associated with more severe lesions

DIFFERENTIAL DIAGNOSIS:

- 1. Wernicke encephalopathy (WE)
- 2. Other Mitochondrial disorders
- 3. Biotin-Thiamine-responsive Basal ganglia disease
- 4. Acute necrotising encephalitis of childhood

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MRI IN HIRAYAMA DISEASE



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An 18 year old male presented in medicine department with history of weakness of left distal upper limb with thinning of left hand and forearm since 2 years.

Patient had no sensory , cranial nerve , cortical or autonomic involvement on clinical examination.

Patient had no other comorbidities.

Patient was sent for dynamic MRI cervical spine to confirm the suspicion of hirayama disease.

Plain MRI was done in neutral and flexion position followed by contrast enhanced MRI in flexion position.



Sagittal T2 weighted MRI of cervical spine in neutral position shows localized cervical cord atrophy from (C4-C6) with intramedullary T2 hyperintensity extending from C3-C6 level.



Sagittal T2 weighted imaging in flexion position shows cord flattening with anterior displacement of dorsal dura, compressing the thecal sac, with a **prominent crescent shaped hyperintense dorsal epidural compartment** as shown (curved arrow).

- Hirayama disease also known as non progressive juvenile spinal muscular atrophy is characterized by insidious onset of unilateral or asymmetric oblique amyotrophy that affects the C7, C8 and T1 myotomes.
- It occurs in **young men** and is characterised by muscle weakness and atrophy in the hand and forearm.
- Chronic microcirculatory changes in the territory of anterior spinal artery induced by repeated or sustained flexion account for the necrosis of anterior horns of lower cervical cord.
- Another theory proposed could be that increased intramedullary pressure during flexion causes loss of dorsal dural attachment from the pedicle due to immunological abnormalities of the dura or posterior ligaments.



Sagittal and axial flexion T1 weighted contrast enhanced magnetic resonance image shows strong homogenously **enhancing epidural crescent**.

 Diagnosis of hirayama is suspected in a young male between 10-20 years of age presenting with insidious onset of distal upper limb muscle weakness and atrophy with lack of sensory and pyramidal signs.

Hallmarks of radiological diagnosis-MRI

Neutral position: abnormal T2-weighted signal of the spinal cord with reduced bulk of spinal cord at site of maximum forward shift without an obvious cause

Flexion position: the posterior dural sac crescent appears as high signal intensity on T1- and T2-weighted sequences and enhances uniformly on T1 C+ (Gd), with or without epidural flow voids

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Unilateral perisylvian syndrome - A rare presentation of malformed cortical anatomy

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Dr Sharath potla First Year Resident Mamta Medical College

CASE REPORT

A 30-year-old male patient visited the neurology Outpatient department, reporting a history of recurring generalized tonic-clonic seizures dating back to birth. Given this concerning medical history, the patient was subsequently referred to the Radiodiagnosis department for an MRI brain epilepsy protocol to conduct a comprehensive evaluation and assessment of the condition.

MRI brain revealed malformed anatomy of right perisylvian cortex. There was thickening of gray matter intermixed with thin and shallow sulci and broad gyri involving the right peri-Sylvian regionsuggesting focal areas of pachygyria and few microgyria.

DIAGNOSIS AND MANAGEMENT

The patient's condition was diagnosed as unilateral Perisylvian syndrome, which was determined based on the previously mentioned findings.

In recent years, advancements in MR neuroimaging have greatly improved our ability to establish correlations between radiological patterns and the clinical features associated with cortical malformations.

As part of the patient's treatment plan, conservative management was implemented, involving the administration of anticonvulsant medications to address the seizures associated with the condition.

The diagnosis of unilateral Perisylvian syndrome was made based on the findings presented. Recent advancements in MR neuroimaging improved the capability to establish connections between radiological patterns and the clinical characteristics linked to cortical malformations.

PERISYLVIAN SYNDROME

Shelvell reported the first documented case of a structural malformation in cortical development, characterized by a primitive Sylvian fissure and a dysplastic operculum. This opercular dysplasia is often attributed to conditions like polymicrogyria or pachygyria. Thickened cortex with signal changes in the adjacent subcortical white matter, as well as irregularities in the cortical-white matter junction. The etiology of this syndrome is primarily genetic and can manifest sporadically or within familial contexts. Perisylvian syndrome has also been linked to congenital cytomegalovirus infection or patients with Aicardi syndrome. Bilateral involvement is more prevalent than unilateral cases.

POLYMICROGYRIA and PACHYGYRIA

Polymicrogyria is characterized by an excessive number of small and prominent convolutions in the cerebral cortex, while pachygyria presents as thin and shallow sulci and broad gyri. Both conditions result from disruptions in the normal cortical development process, occurring during the late stages of neuronal migration and cortical organization. These abnormalities affect the deeper layers of the cerebral cortex, leading to the formation of multiple small gyri. Clinically, they encompass a wide spectrum of manifestations, ranging from early-onset epileptic encephalopathy to the selective impairment of cognitive functions, highlighting the complexity and variability of these developmental disorders.



T2 weighted imges show dysplastic right sylvian fissure on the coronal image and pachygyria on the axial images.



FLAIR coronal images show the dysplastic right perisylvian cortex with subcortical FLAIR hyperintensity .sagital and axial images show polymicrogyria.

CONCLUSION

The cerebral cortex is susceptible to a range of anatomical malformations that can result in conditions ranging from mild to life-threatening. Congenital perisylvian syndromes are disorders due to delayed migration and cortical organization, characterized by distinct clinical and imaging features. Bilateral abnormalities tend to be more prevalent than unilateral. A comprehensive understanding of cerebral anatomy is crucial for healthcare professionals as it not only aids in recognizing these anatomical malformations but also plays a pivotal role in guiding effective patient management strategies.

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IMAGING FINDINGS IN A TYPICAL CASE OF RACEMOSE NEUROCYSTICERCOSIS IN SYLVIAN FISSURE

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45yr old female presented with chief complaints of headache since 15 days and 5 episodes of generalised tonic clonic seizures 2 weeks back with post ictal confusion lasting for about 30mins, with past history of similar episodes of seizures(3 episodes) in the past 10 years. On examination higher mental functions, and cranial nerves were normal, with no sensory/motor deficits



Axial NCCT brain section showing well defined lobulated extra axial hypodense cystic lesion involving the left Sylvian fissure and expanding it, noted causing no significant perilesional edema in adjacent cortex



MR Axial T2/FLAIR image showing well defined lobulated hypointense cystic lesion in the left Sylvian fissure

- Neurocysticercosis is one of the most common parasitic(cestode) infection of the central nervous system and is considered the most serious form of cysticercosis
- It is caused by encysted larval stage(cysticercus) of Taenia solium/ pork tapeworm
- The disease can present in two formsparenchymal and extraparenchymal or racemose form
- Patients affected with the parasite usually present with headache, generalised./focal seizures, features of raised intracranial tension,psychiatric disturbances, focal neurological deficits and meningoencephalitis



- a. DWI shows cyst content to be freely diffusing fluid
- b. Post Gadolinium contrast axial, and coronal sections of brain showing ring enhancement pattern of the lesion
- Racemose NCC refers to cysticerci lodged either in subarachnoid space of basal cisterns, Sylvian fissure, spinal medulla or within the ventricular system and is thought to be due to aberrant cysticercus of T.Solium
- It is characterised by excessive growth of cystic membranes following degeneration of scolex, and appears like a cluster of grapes, and the name racemose cyst. The scolex would be absent on imaging
- This is a rare presentation of the disease, and often can be missed in a Non contrast Computed Tomography study, but can be picked up on MR imaging
- The parenchymal variant is characterised by well defined tiny cystic lesions with an eccentric hyperdense scolex noted within in NCCT, which is hyperintense on MR imaging, and can present in 4 different stages(vesicular,colloidal,granular and calcified nodular)

HALLMARKS OF RADIOLOGICAL DIAGNOSIS

1. Non contrast CT imaging shows well defined hypodense lobulated cystic lesion in extraparenchymal location with no detectable scolices within

- 2. MR imaging shows typical lobulated cystic appearance which is hypointense on T1 weighted images, hyperintense on T2 images showing suppression on FLAIR images, with DWI showing freely diffusing fluid content within, with absent scolices within the lesion.
 - The closest differential diagnosis is a cerebral Hydatid cyst, and this can be differentiated with MR Spectroscopy, in which Hydatid cyst shows characteristic Pyruvate peak, which is absent in Racemose variant of Neurocysticercosis. Other differentials include arachnoid cysts, porencephaly and cystic astrocytoma



A CASE REPORT OF JUVENILE NASOPHARYNGEAL ANGIOFIBROMA:

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Dr. Kothakapu Sai Spoorthi Reddy Resident SVS Medical College

A 19 year old male patient presented with left hemi facial pain and difficulty in opening mouth since 5 months



NCCT shows Ill defined soft tissue density lesion with epicenter at the body of left pterygoid bone causing lytic destruction of medial, lateral & posterior walls of left maxillary sinus, pterygoid plates and greater wing of sphenoid bone. Anteriorly the lesion extends upto the left posterior choana, anterolaterally into the left maxillary sinus and posterolaterally into the left masticator space.



The lesion appears Isointense to adjacent pterygoid muscles on T1W, heterogeneously hyperintense on T2W/FLAIR .Showing restricted diffusion on DWI & reversal on ADC.





Posteriorly the lesion extends into the Nasopharynx causing left Eustachian tube blockage and resulting in mastoiditis on the ipsilateral side. Anterolaterally extending into the left maxillary sinus.

Post contrast there is intense enhancement of the lesion.

Clinical and imaging findings, together with gender and age of the patient, were suggestive for nasopharyngeal juvenile angiofibroma.

Histopathological examination revealed a proliferative connective tissue stroma interspersed

with a thick vascular network, thereby confirming the diagnosis of Juvenile Nasopharyngeal Angiofibroma.

DISCUSSION:

Nasopharyngeal angiofibroma, also called juvenile angiofibroma (JNA), is a benign, non-encapsulated, fibrovascular tumor ,that grows very slowly. It is a common tumor of the nasopharynx, but represents only 0.05% of all head and neck neoplasms. The tumor has its onset in childhood with highest incidence in teenage males between 14 and 17 years old. Its etiology is unknown, but relation to sex hormones is strongly suggested.

From the nasopharynx, the tumor grows slowly into the pharynx, nasal fossa and nares. It may also originate in the pterygopalatine fossa. Cranially, the tumor may extend into the sphenoid sinus or even through the sinus into the sella turcica and cavernous sinus, involving the middle cranial fossa by extradural spread. Intracranial extension is less common. Finally, tumoral extension is also observed into the subtemporal space through the sphenomaxillary fissure, or through the inferior orbital fissure into the cranial floor.

IMAGING FEATURES:

JNA is demonstrable as an avidly enhancing lobulated non-encapsulated soft tissue nasopharyngeal mass on CT scan following intravenous administration of contrast medium reflecting its characteristic vascularity. Intraorbital and intracranial extension is also better demonstrable. Anterior bowing of the posterior maxillary sinus wall is better demonstrated on CT and is referred to as the Holman-Miller sign. Widening of the sphenopalatine foramen may also be observed. Bony erosion is common& extensive bony destruction may also be seen especially in advanced disease.

MRI because of its better soft issue characterization in comparison to CT is able to delineate mucosal inflammation versus sinus fluid. It is also valuable at evaluating tumour extension into the orbit and intracranial compartments and is accurate in tumour staging. JNA is of intermediate signal intensity and heterogenous signal intensity on T1 and T2 weighted spin echo sequences respectively. Multiple flow voids will also be seen within the tumour on both sequences. The presence of prominent flow-voids seen on MRI scans represents enlarged tumour vessesls giving characteristic salt and pepper appearance. MR imaging also better delineates spread in the region of the cavernous sinus and into the middle cranial fossa.

DIFFERENTIALDIGNOSIS:

Imaging differential considerations include:

- Nasopharyngeal Carcinoma
- Rhabdomyosarcoma (head and neck)
- Ewing sarcoma.

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ACADEMIC ACTIVITIES OF IRIA TS CHAPTER

Monthly Meeting on 25th August 2023 at Century Hospital





UPCOMING CMES

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



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