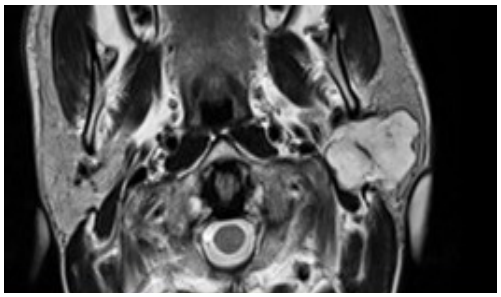
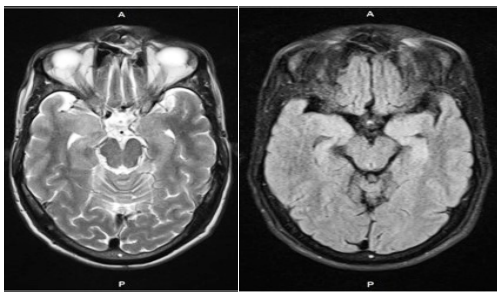
Issue 14 Oct 2022

IRIA Telangana e-Newsletter





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Indian Radiological & Imaging Association

Telangana State Chapter 2022

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From the President's Desk

Respected Senior Radiologists and Radiology Colleagues.

Hope you all had a pleasant Dussera festival and advance wishes for a happy and safe Deepawali.

Happy to inform you all that the next issue of IRIA Newsletter of Telangana State Chapter is ready and prepared with sincere and innovative ideas by Dr. Jagan Mohan Reddy sir and his team.

I am very happy to inform you that Dr. Jagan Mohan Reddy will be confered Life time achievement award in 8th state annual conference of Telangana State chapter of IRIA in Karimnagar on 15th and 16th of October 2022, which is being conducted in association with Karimnagar IRIA Sub Chapter and hosted by Pratima Institute of Medical Sciences.

I request all the Radiologists to encourage membership drive of State chapter and also request all who are not registered to get registered for E voting for elections scheduled in 1st week of November.

Warm Regards
Dr. Venkata Nageshwar Goud

From the General Secretary Desk

Dear esteemed members,

Happy festive season and warm wishes

I feel happy that after a long gap of three years we are all participating in our own Telangana state IRIA conference conducted physically.

I specially thank Prathima Institute of Medical Sciences for hosting the State Conference and given all the necessary support. I thank Dr. Prabhakar Reddy for his valuable support and guidance throughout the execution of this wonderful conference.

For the benefit of Radiology residents paper and poster presentations, film reading sessions and Quiz programs are organized.

Monthly meetings of the IRIA are a huge success with good participation from students and consultants. Quiz competition is being held in all the monthly meetings, well-coordinated by our Joint Secretary Dr. Sudheer.

Newsletter is being unveiled every 3 months at regular intervals with special interest and hard work by chief editor Dr. Jagan Mohan Reddy.

I request all the Radiology consultants to attend the academic programs of State IRIA and make it grand success.

Long live IRIA

Dr. Krishna Mohan Pottala
General Secretary

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RADIATION EXPOSURE TO PATIENT: DO WE NEED TO BE MORE CAREFUL?

In the past few decades, advances in medical imaging have considerably impacted the way medicine is being practiced. Imaging has become integral & inescapable tool in the practice of medicine of present era. As a result, overall number of the imaging procedures are on the rise. Significant proportion of medical imaging procedures like X-rays, Fluoroscopy, Mammography, Bone densitometry, CT scan, Nuclear medicine and PET-CT scans utilize ionizing radiation to produce clinically useful image. With advanced imaging equipment, radiation exposure to patient form these imaging modalities for a single procedure ranges from < 1 mSv to 25 mSv. However, it is not uncommon for a patient to undergo multiple medical imaging procedures which increases their cumulative exposure.

Biological effects from radiation exposure can be divided into deterministic or stochastic effect. Deterministic effects occur when a specific exposure threshold (usually above 100 mSv) has been exceeded and unlikely to happen with kind of exposure one get from these imaging procedures. Stochastic effect represents an outcome that occurs with a certain probability but without a defined threshold and probability increase with the level of exposure. Stochastic effects are discovered many years after radiation exposure and include the development of cancer.

Observational studies has shown an increased risk of cancer among long-term survivors of the Hiroshima and Nagasaki atomic bombs who received exposures of 10–100 mSv. A single procedure of imaging modalities like CT scan, Fluoroscopy, Nuclear medicine and PET-CT scans are capable of delivering an equivalent radiation exposure. Based on certain observational epidemiologic studies involving workers of nuclear industry and atomic bomb survivors, few articles have been published which calculated life time attributable risk of cancer due to radiation exposure from imaging modalities. Berrington de González et al. estimate that approximately 29,000 future cancers could be related to 72 million CT scans performed in the U.S in 2007. Smith-Bindman et al. estimate that 1 in 270 women and 1 in 600 men who undergo CT coronary angiography at age 40 will develop cancer from that CT scan; the risks for 20-year-olds are estimated to be roughly twice as large.

Currently, the way ionizing imaging modalities are being used in clinical practice, cumulative radiation exposure to many patients would easily be > 100 mSv, which if not a significant individual risk, is definitely an alarming population risk. Reason for this kind of radiation exposure is multifactorial. Firstly, referring physicians as well as radiologists generally underestimate the magnitude of radiation doses and their associated effects. This could be due to general lack of epidemiologic data specific to ionizing radiations of medical imaging and long lag period between the exposure and its stochastic effect which makes it difficult to attribute a cancer to specific exposure. On the other hand, clinical benefit or impact on management from these imaging modalities are clearly evident. Secondly, it is not uncommon for a patient to be referred for a variety of medical imaging tests and procedures by different physicians which increases their cumulative exposure and makes it difficult to track also.

Although, there is uniform agreement that care should be taken to weigh the medical necessity of a radiation exposure against the risks and if possible advise an alternate imaging which does not cause radiation exposure or decreases it substantially, but in reality how seriously and effectively is it been followed? Moreover, there are no strategies or national program in place to track the radiation dose of a patient such as TLD badges used for radiation professionals. The implementation of such a strategy to track and document radiation doses may help referring physicians, radiologists, and patients to stay aware of their cumulative exposure and would ensure more justified use of imaging modalities.

ACHIEVEMENTS

Congratulations

Dr. P. Krishna Mohan

Promoted as

Director of Radiology of Entire

Vijaya Diagnostic Group

Congratulations

Dr. Sikandar Shaik

DMRD, DNB, MNAMS, FICR

Consultant PET-CT & Radiology

Yashoda Hospitals, Hyderabad

BIR
The British Institute of Radiology
1897 125 Years 2022

Dr Sikandar Shaikh
Email: idrsikandar@gmail.com

24 August 2022

Dear Dr Shaikh

BIR Nuclear Medicine & Molecular Imaging SIG

I am writing to you on behalf of the Chair of the Nuclear Medicine & Molecular Imaging Special Interest Group to formally invite you to join the Management Group of the SIG. I am aware that you and the SIG's Chair, Dr Daniel McGowan, have already spoken and agreed this informally.

The normal term as a member of the Management Group is 3 years, with an option to renew for a second term. The Group meets three times a year to discuss the planned events and courses, education, publications and other agenda items. Two of the three meetings are designed to take place by videoconference, with one taking place in person each year. Obviously, in your particular circumstances, video attendance to all three meetings would be perfectly acceptable. The next two meetings are planned to take place:

- Friday 18 November 2022 at 2pm (videoconference)
- Friday 3 March 2023 12:00 to 14:30pm (f2f with lunch)

Please do not hesitate to contact either Dr McGowan or myself if you have any questions.

Yours sincerely

Lucy Stewart

Lucy Stewart
BIR Committee Secretary

cc: Dr Daniel McGowan

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ARTICLES

Dr. Sudheer
Consultant Radiologist
Continental Hospital Gachibowli

IMAGING APPROACH TO NECK SPACES

BOUNDARIES OF NECK

The neck extends from the head above to the shoulders and thorax below. Its superior boundary is along the inferior margins of the mandible and bone features on the posterior aspect of the skull. The posterior neck is higher than the anterior neck to connect cervical viscera with the posterior openings of the nasal and oral cavities.

KEY CONCEPTS:-

- To understand how the layers of the deep cervical fascia, along with muscles and bones, help to define compartments or "spaces" in the neck.
- To understand the normal anatomy and contents of each of these spaces in the suprahyoid and infrahyoid neck.
- To accurately localize neck pathology into a specific space in order to generate the best differential diagnosis.
- The superficial layer, below the platysma, enveloping the most superficial neck structures such as the parotid glands, sternocleidomastoid, and trapezius muscles.
- The middle layer, surrounding the larynx, pharynx, trachea, proximal esophagus and thyroid.
- The deep or prevertebral layer, enfolding the prevertebral and paraspinal muscles.

Some neck spaces are contiguous with one another-

- The prestyloid parapharyngeal, submandibular

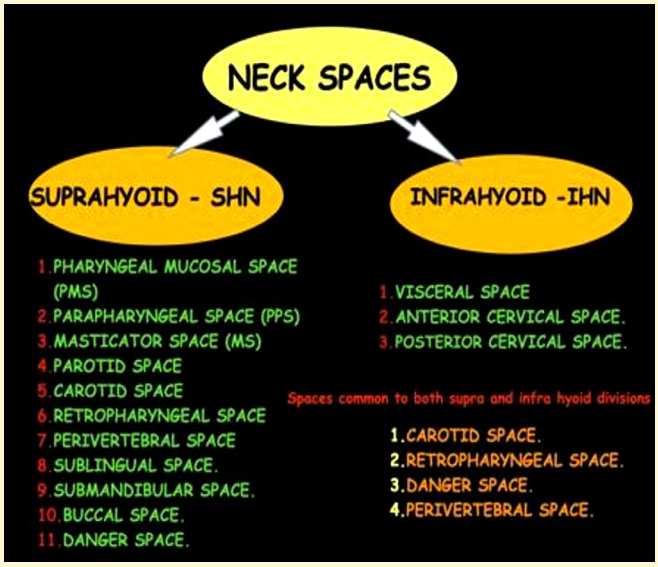


Fig 1:- Neck spaces based on fascia.

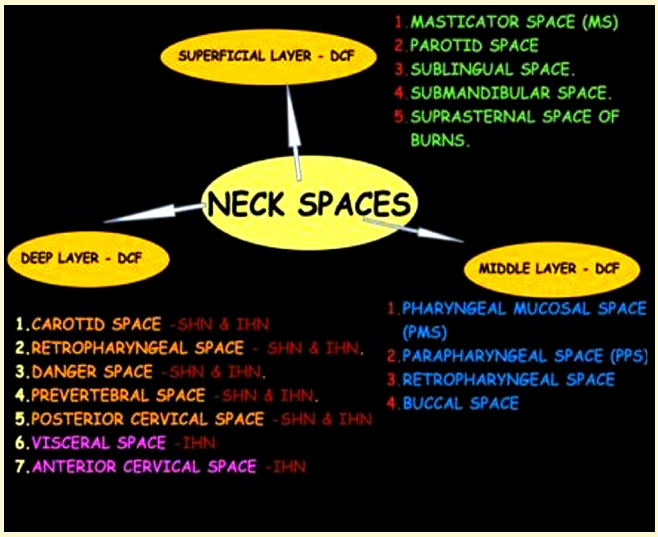


Fig 2:- Classification of neck spaces.

space, and sublingual space are interconnected because there is no fascia between the pre-styloid parapharyngeal space and the submandibular space, and because the sublingual space is not fascia lined.

- The prestyloid parapharyngeal and parotid space are considered continuous because the fascia in between is very thin and incomplete, and does not function as a barrier for lesion expansion.
- The masticator space and buccal space are connected through a fascia-free corridor medial to the tendon of the superficial part of the masseter muscle.
- The pharyngeal mucosal space and visceral space communicate freely. The level of the hyoid bone is used as a practical landmark to separate the two spaces, but there is no fascia between them.
- The carotid space and retropharyngeal space are variably continuous as the carotid sheath can be defective, especially in the suprahyoid neck.
- The perivertebral space is in continuity with the epidural space at the level of the neural foramina.

Several neck spaces abut the skull base, and in four of these spaces a foramen facilitates the intracranial extension of neck pathology or vice versa:

- Masticator space: foramen ovale, spinous foramen
- Carotid space: foramen jugulare, carotid canal, hypoglossal canal
- Parotid space: stylomastoid foramen
- Pharyngeal mucosal space: foramen lacerum.

H&N SPACE CONTENTS

Space	*Boundaries*	*Major contents*	*Common pathology*
Pharyngeal mucosal (PMS)	Mucosa to pharyngeal constrictors of nasopharynx and oropharynx (some include hypopharynx and oral cavity)	Mucosa, Waldeyer's ring, minor salivary glands, constrictor and levator palatine muscles, cartilaginous Eustachian tube	Malignant tumors (mucosal = SCC or NPC and submucosal = minor salivary tumors or lymphoma) Pharyngitis, tonsillitis
Parapharyngeal (PPS)	Skull base to mandibular angle, borders PPS, CS, PS, MS	Fat, minor salivary glands	Benign salivary tumors, rare branchial cleft cyst or vascular malformation
Masticator (MS)	Skull base to mandibular angle, lateral to PPS	Muscles of mastication, posterior body and ramus of mandible, CNV3	Odontogenic infections, venous malformations, sarcomas
Parotid (PS)	Enclosed within the parotid fascia, lateral to PPS, MS	Parotid gland, CN7, lymph nodes, retromandibular vein, external carotid artery branches	Pleomorphic adenoma Low and high grade malignant salivary neoplasms Acute or chronic parotitis
Visceral (VS)	Hyoid to mediastinum, anterior to PVS, medial to CS	Thyroid and parathyroid glands, larynx, hypopharynx, trachea, esophagus	Thyroid nodules Thyroid cancer Parathyroid adenoma Mucosal SCC Chondrosarcoma Diverticula
Carotid (CS)	Carotid sheath, enclosed by all 3 layers of DCF, skull base to aortic arch (SHN + IFN)	SHN: ICA, IJV, CN 9–12 IFN: CCA, IJV, CN 10 (vagus)	Vascular pathology related to carotid (aneurysm, dissection, arteritis) or jugular vein (thrombosis or thrombophlebitis) Nerve sheath tumors Paragangliomas
Retropharyngeal (RPS)	Skull base to T3, between visceral and alar fascia	Fat and lymph nodes	Metastatic lymph nodes (NPC, thyroid, hypopharynx, lymphoma) Suppurative lymph nodes (children) Effusion
Perivertebral (PVS)	Skull base to T4, posterior to RPS	Prevertebral muscles, vertebral bodies, scalene muscles, brachial plexus roots, phrenic nerve, vertebral artery and vein	Discitis/osteomyelitis Longus colli tendinitis Primary bone tumors (chordoma, ABC, osteoma, giant cell) Vertebral metastases Sarcomas Nerve sheath tumors

- SCC squamous cell carcinoma, NPC nasopharyngeal carcinoma, ABC aneurysmal bone cyst, SHN suprahyoid neck, IFH infrahyoid neck, ICA internal carotid artery, CCA common carotid artery, IJV internal jugular vein

BASIC RULES OF DISPLACEMENT:-

Parapharyngeal space lesions - Push the submandibular gland inferiorly/ Push the masticator space laterally/ Push the pharyngeal mucosal space medially

Masticator space lesions - Push the prestyloid PPS posteromedially

Pharyngeal mucosal lesions - Push the prestyloid PPS laterally

Parotid space lesions - Push the prestyloid PPS anteromedially/ Widen the SMT

Carotid space lesions - Push the prestyloid parapharyngeal space anteriorly/ Narrow the SMT/ Push the submandibular gland anteriorly

Retropharyngeal space lesions - Push the prevertebral muscles posteriorly

Perivertebral space lesions - Make the prevertebral muscles bulge anteriorly/ Extend in the spinal canal through the neural foramina

KEY POINTS FOR IMPORTANT SPACES:-

PHARYNGEAL MUCOSAL SPACE:- While squamous cell carcinoma is the most common tumor of the PMS, the presence of Waldeyer's ring lymphoid tissue and submucosal minor salivary glands may result in lymphoproliferative and salivary neoplasms, respectively.

Parotid Space (PS):-The parotid gland is the only salivary gland to contain lymph nodes; the differential diagnosis for lymphadenopathy must be considered for any PS mass. Pleomorphic adenoma shows characteristic T2 hyperintense signals (fig :-3) and delayed enhancement. They are benign, represent most common parotid masses.

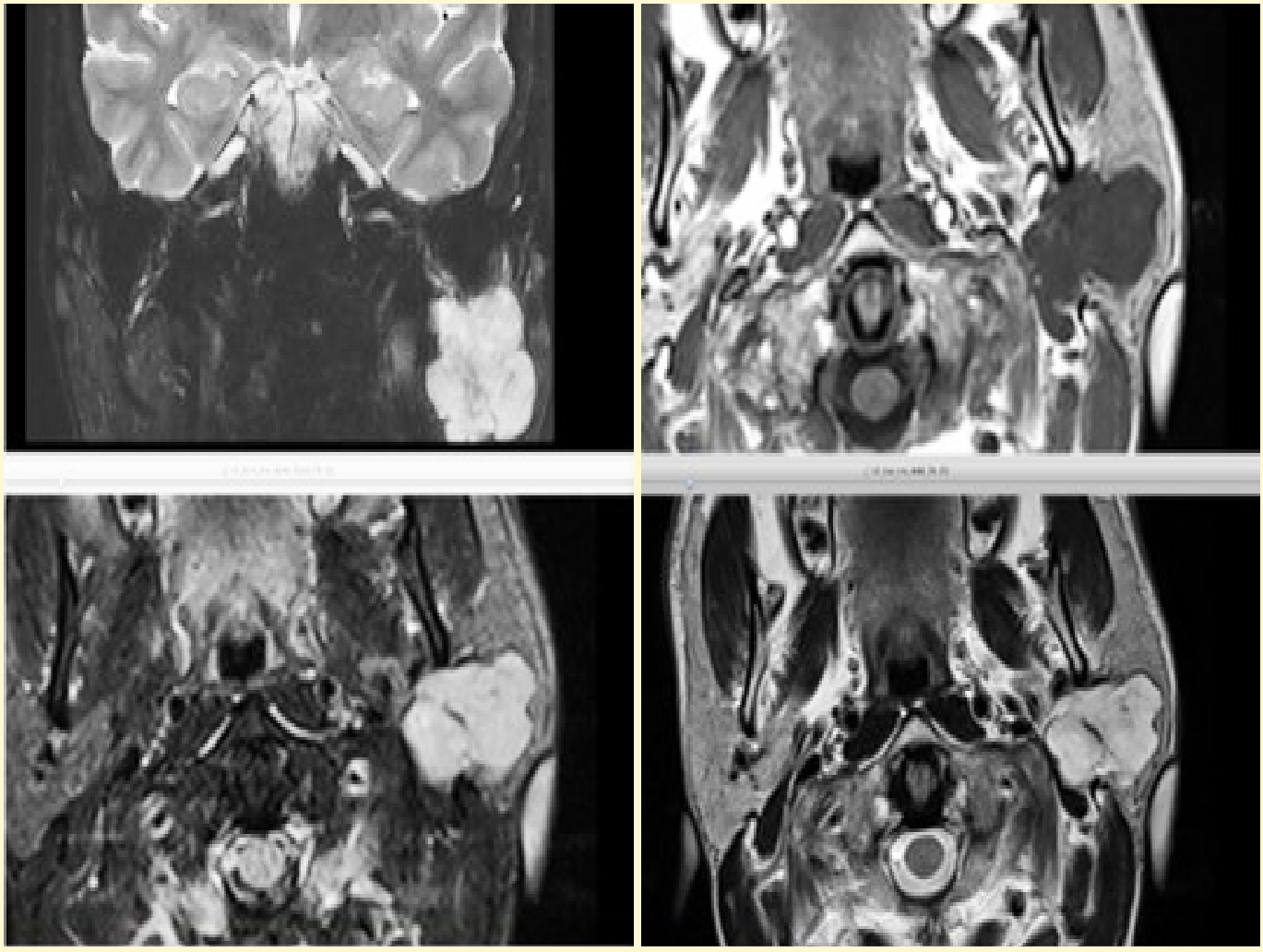


Fig 3:- Pleomorphic adenoma.

Infrahyoid Neck:

Visceral Space (VS):-US is the primary imaging modality for both inflammatory disease and work-up of thyroid nodules, with cross-sectional imaging used when there is concern for extrathyroidal extension of tumor, or to evaluate potential airway compression or substernal extension.

Carotid Space:- The CS spans the suprahyoid and infrahyoid neck, with all three layers of the DCF contributing to the carotid sheath.

Retropharyngeal, Prevertebral and Danger Spaces (RPS):- Retropharyngeal collections that have entered the danger or prevertebral spaces can descend to the mediastinum and as far inferiorly as the coccyx.

Differentiation between a carotid body tumor and vagal paraganglioma based on the location of the tumor center

- Carotid body paragangliomas are seen at the carotid bifurcation, while vagal paragangliomas are located at the level of the nodose ganglion, 2 cm below the skull base.
- Differentiation between a carotid body tumor and vagal paraganglioma based on the direction of large vessel displacement
- Paragangliomas can only grow substantially in a cranial or caudal direction as the carotid sheath inhibits growth in the axial plane. A vagal paraganglioma can therefore also reach the level of the carotid bifurcation when large.

Large lesions at the carotid bifurcation can be differentiated based on the displacement of the vessels:

- Vagal paragangliomas push the internal carotid artery anteromedially and the external carotid artery laterally, separating internal carotid artery and internal jugular vein.
- Carotid body paragangliomas (fig:-4) push the external carotid artery anteriorly and the internal carotid artery posteriorly, keeping internal carotid artery and internal jugular vein together.

Fig :4-Carotid body tumor – paraganglioma.

Dr. K. SUDHEER, MD (MGIMS)
Fellow Diagnostic Neuroradiology (KIMS)
European Diploma in Radiology (EdiR) Diplomate of ICRI
Ex. Associate Professor (PIMS). Ex. Consultant Radiologist – VDC.
Consultant Radiologist – Continental Hospitals

Dr. Tharani Putta,
Professor and Senior Consultant Radiologist, Asian Institute of Gastroenterology Hospitals

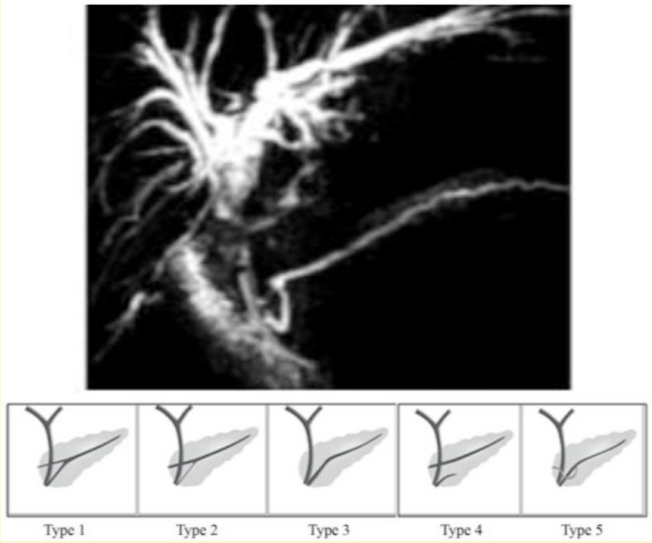
"PANCREAS-RADIOLOGICAL ANATOMY AND PATHOLOGIES"

On June 24, IRIA monthly meet was held. Dr. Tharani Putta, Professor and Senior Consultant Radiologist, Asian Institute of Gastroenterology Hospitals had delivered a guest lecture on "Pancreas – Radiological Anatomy and Pathologies".

During this talk a brief overview was given on Radiological anatomy, variant anatomy and developmental and vascular abnormalities of the pancreas and the ductal system.

Pancreatic ductal anatomy & variants

- ***Main pancreatic duct*** - portion of the dorsal duct proximal to the dorsal-ventral fusion point.
- ***Pancreatic duct of Wirsung*** - segment of the ventral duct between the dorsal-ventral fusion point and the major papilla.
- ***Accessory pancreatic duct (of Santorini)*** - portion of the dorsal duct distral to the dorsal-ventral fusion point.



Importance of using the correct nomenclature for Acute pancreatitis based on Revised Atlanta Classification was highlighted during the talk. The terminology used in structured reporting were explained in detail using illustrative cases.

Acute pancreatitis: Revised Atlanta Classification

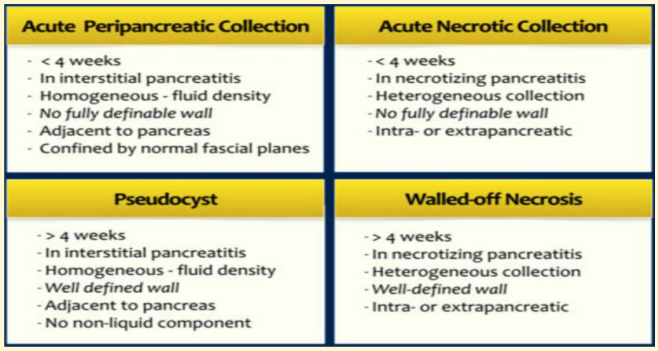
- Early Vs late
- Acvle Ireetsmol edematous pancreatins Vs Acute Necrobung patateautts
- Seventy:

Mild acute pancreatitis: no organ failure; no local or systemic complications; mostly resolve in a week.

Moderately severe acute pancreatitis: transient organ failure, local complications or exacerbation of co-morbid illness.

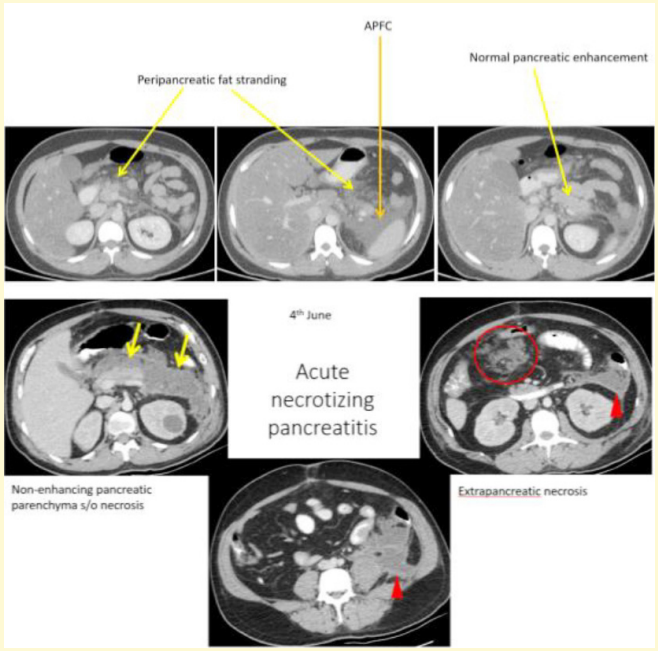
Severe acute pancreatitis: persistent organ failure > 48 hrs.

- Local complications: peripancreatic fluid collections, pancreatic and peripancreatic necrosis, pseudocyst, walled -off necrosis, GOO venous thrombosis, colonic necrosis.

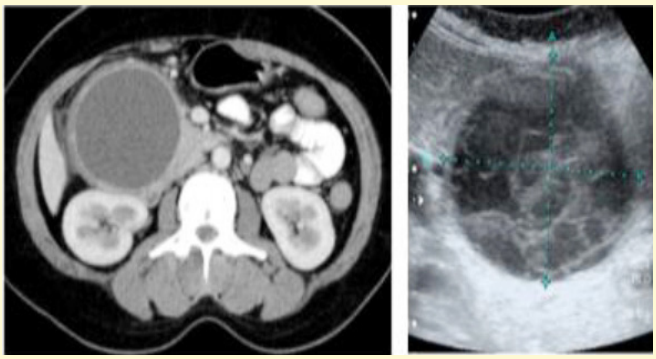


Various other forms of pancreatitis namely chronic pancreatitis, its complications, groove pancreatitis, autoimmune pancreatitis and their diagnostic criteria were also emphasized up on.

Acute interstitial edematous pancreatitis

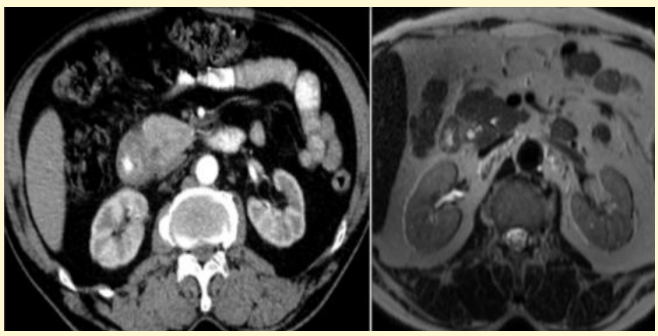


Walled off necrosis



h/o severe acute abdominal pain requiring hospital admission

Walled off necrosis



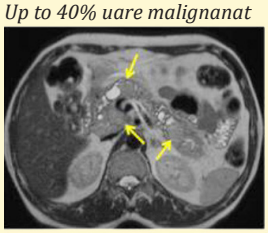
Finally, a brief overview was given on pancreatic neoplasms. Solid and cystic pancreatic neoplasms were discussed as case based approach for each neoplasm.

Pancreatic neoplasms

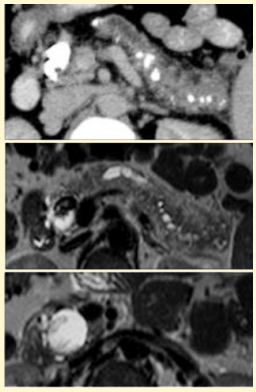
• Cystic pancreatic lesions	• Solid lesions
- IPMN	- Adenocarcinoma
- Serous cystadenoma	- Neuroendocrine tumours
- Mucinous cystic neoplasm	- SPEN
- SPEN	- Lymphoma
- Cystic neuroendocrine tumours	- Metastasis
- True cyst	- Pseudotumours
- Pseudocyst	

IPMN Main duct type

Do not present as cystic lesions of pancreas
Cause diffuse PD enlargement
Up to 40% uare malignanat

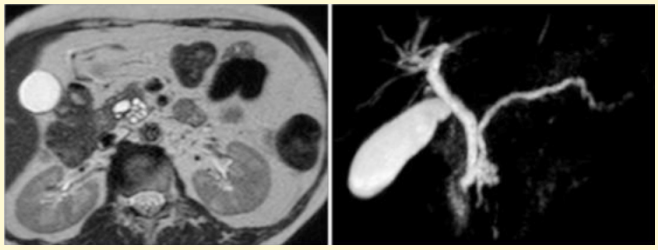


Multifocal intraductal polypoidal lesions



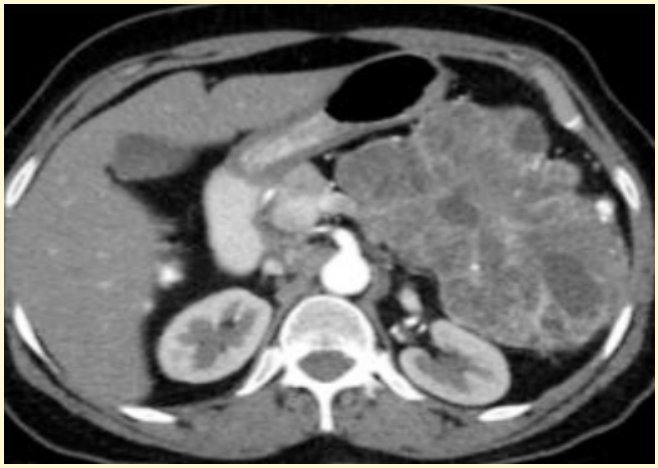
Imaging features s/o malignancy:
- Enhancing mural nodules
- Main duct dilatation > 1cm

IPMN - side branch type



"Cluster of grapes appearance" Communication with the pancreatic duct

Serous cystadenoma



- *No malignant potential, slow growing*
- *Predisposition to older women & pancreatic head*
- *Multiple small cysts (>6 in number; 1mm to 2cm)*
- *Central scar, sometimes calcified*
- *Lobulated outer border*
- *Thin enhancing septa, honeycomb appearance*
- *Pseudo-solid appearance*

Atypical findings:

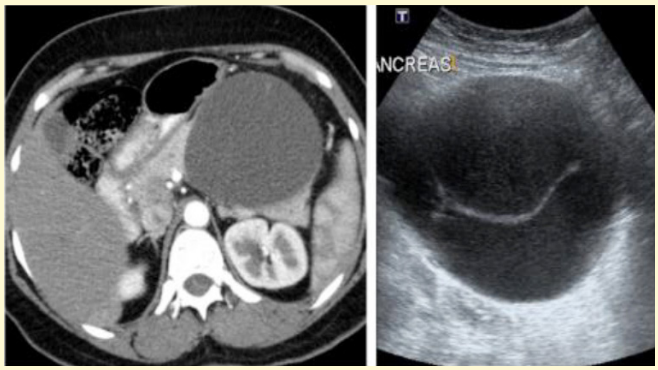
- ***Oligocystic / macrocystic variant***
- ***Lack of central scar***

EUS-aspiration:

- No mucin

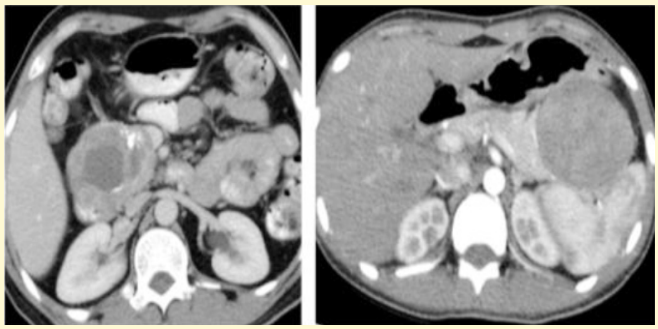
- Glycogen-rich epithelial cells

Mucinous cystic neoplasm Vs Pseudocyst



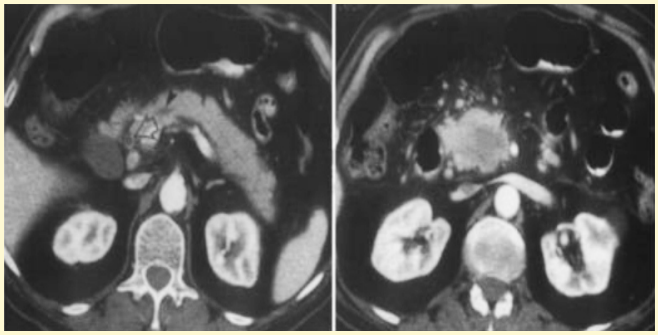
HPE: Benign mucinous cystic neoplasm

SPEN - Solid Papillary Epithelial Neoplasm

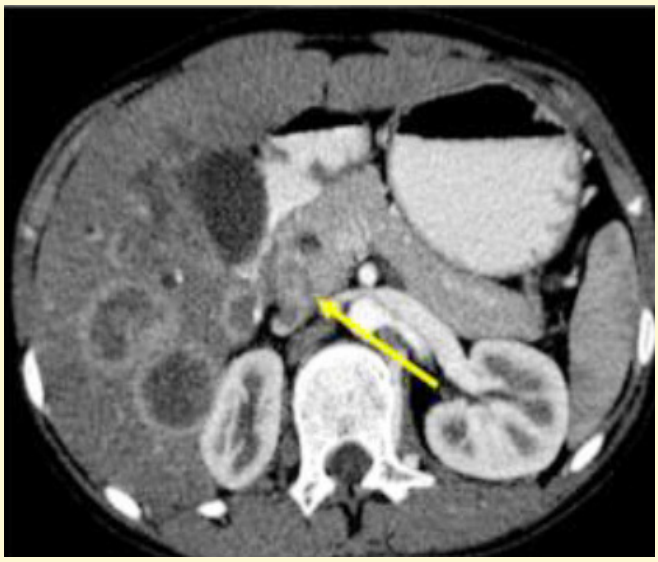


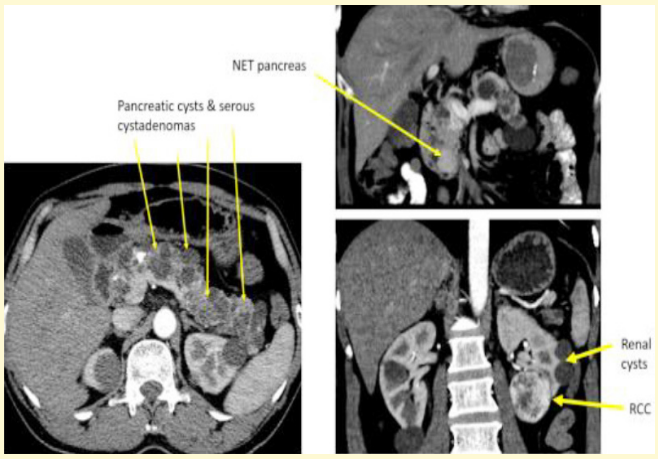
Case 1 - 29 year old *Case 2 - 18 year old*

Adenocarcinoma pancreas



NET pancreas with hepatic metastases



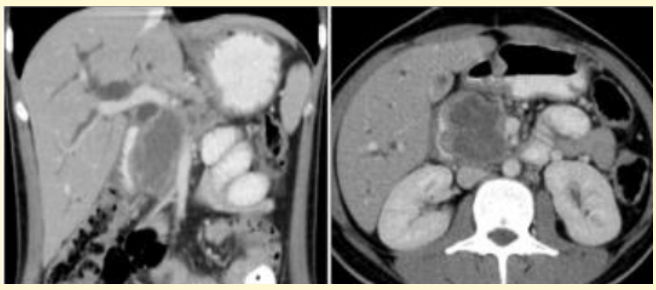


24 year old PLHIV with obstructive jaundice

CA 19-9 = 703

CD4 count = 2

- *FNA - necrotic tissue with occasional ill-defined histiocytic aggregate-to exclude infectious etiology*
- *No other systemic evidence of TB*
- *Also has bowel CMV infection*



Dr. Y Sai Rahul
1st year Radiology Resident
SVS medical College

CASE REPORT:

A CASE REPORT OF PARANEOPLASTIC LIMBIC ENCEPHALITIS AS A MANIFESTATION OF SMALL CELL LUNG CANCER

Dr. Yakkanti Sai Rahul[1], Dr. Sandeep Madineni[2], Dr.Subhash Reddy[3]
Dr. G. Ramakrishna Reddy[4], Dr. K. Venkat Ram Reddy[5].
[1]Resident, [2]Assistant professor, [3]Professor, [4]Professor and HOD of Dept of Radiodiagnosis,
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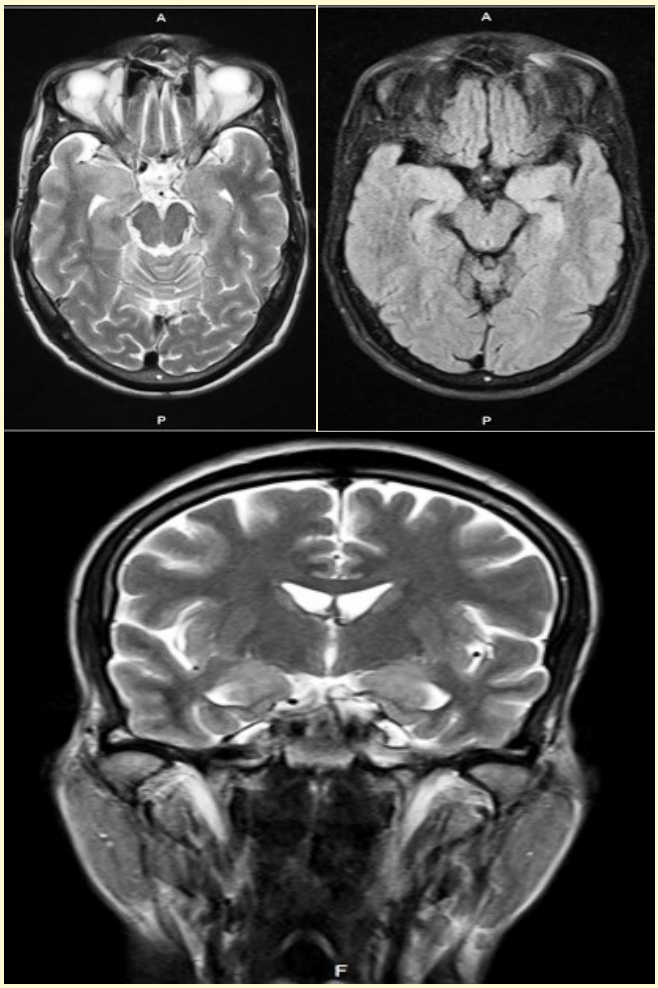
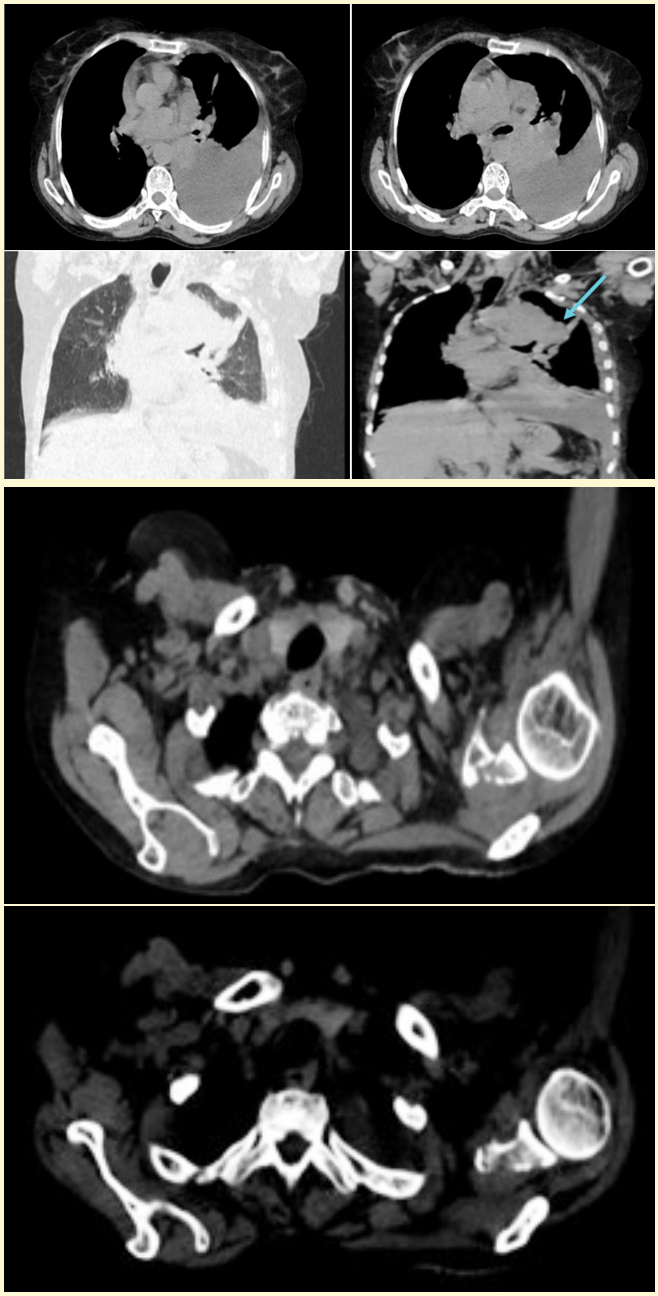
A 50 year old female came with complaints of disturbed sleep, shortness of breath since 10 days, altered behaviour since 2 weeks and two episodes of seizures in 3 days .

On MRI:



Bilateral mesial temporal lobes are thickened and shows T2/T2 FLAIR mild hyperintense signal intensity.

ON NCCT:



Ill defined Soft tissue density mass noted in left supra hilar region. Mild shift of trachea & mediastinum towards right side with moderate left pleural effusion.

Permeative destruction of left scapula noted at the base of glenoid with surrounding soft tissue thickening.

All above imaging F/s/o carcinoma left lung with metastasis in left scapula.

HPE suggestive of Small cell carcinoma of lung.

DISCUSSION:

The Imaging features are suggestive of Paraneoplastic limbic encephalitis with small cell lung cancer .

Paraneoplastic limbic encephalitis (PLE) is a rare neurological syndrome associated with cancer, and selectively affects limbic system structures, including the hippocampus, hypothalamus, and amygdala. Patients often present with cognitive impairment, personality change, short-term memory loss, and seizures.

The presence of neuronal antibodies in patients with limbic encephalitis supports an autoimmune pathogenesis and can guide the search for an underlying tumor. Since the initial description of antibodies that recognized intracellular antigens present in tumor cells and neurons (onconeural antibodies), there has been an ever-growing number of circulating neuronal antibodies associated with paraneoplastic and nonparaneoplastic limbic encephalitis, and other autoimmune encephalitis that targets neuronal surface antigens. Unlike onconeural antibodies, antibodies against surface antigens do not always indicate that the limbic encephalitis is paraneoplastic.

Antineuronal antibodies, when present in the serum and CSF, facilitate the diagnosis of PLE and often allow the early detection of the associated tumour . However, the frequency of antineuronal antibodies in patients with PLE is largely unknown.

PLE is most frequently associated with lung cancer (approximately 50% of PLE cases), and 80% of these cases are small cell lung cancer (SCLC).

IMAGING FEATURES:

Most common location of involvement is the mesial temporal lobes and limbic systems, typically manifested by cortical thickening and increased T2/FLAIR signal intensity of these regions. Bilateral involvement is most common (60%), although often asymmetric . The lateral temporal lobe and insula are less commonly involved, whereas the basal ganglia, in contrast, are frequently involved, helpful in distinguishing it from HSV encephalitis which characteristically spares the basal ganglia. Although far less common, essentially any part of the central nervous system can be involved .

DIFFERENTIAL DIAGNOSIS:

Several noninfectious diseases may involve the temporal lobes and be mistaken for ALE. Gliomas may cause diffuse temporal lobe changes on MRI, while imaging features of high-grade neoplasm such as necrosis, irregular enhancement and vasogenic edema are absent early on. Although classically thought to present unilaterally, in one retrospective series, bilateral medial temporal lobe involvement was seen in 54% cases of patients with suspected ALE who later developed glioblastoma.

Seizures can also cause temporal lobe imaging abnormalities, but early control of seizures with anti-epileptic drugs alone, lack of prodromal neuropsychiatric symptoms and the resolution of temporal lobe changes after cessation of seizure activity are all supportive of seizure-related MRI changes rather than ALE. Ischemic stroke involving the medial temporal lobe usually presents acutely but sometimes causes only mild neurocognitive deficits; patients may delay seeking medical attention. Careful history-taking is needed to differentiate a subacute progression of symptoms over days from a static neurologic insult that occurred days earlier. On MRI, signal abnormality restricted to a vascular territory helps distinguish ischemic stroke from ALE.

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3. Gozzard, P. et al. Paraneoplastic neurologic disorders in small cell lung carcinoma: A prospective study. Neurology 85, 235–239, (2015).
4. Saiz A, Dalmau J, Butler MH, Chen Q, Delattre JY, De Camilli P, et al. Anti-amphiphysin I antibodies in patients with paraneoplastic neurological disorders associated with small cell lung carcinoma. J Neurol Neurosurg Psychiatry. 1999 Feb;66(2):214–7.

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CASE REPORT:

A CASE REPORT OF VON HIPPLE LINDAU SYNDROME :

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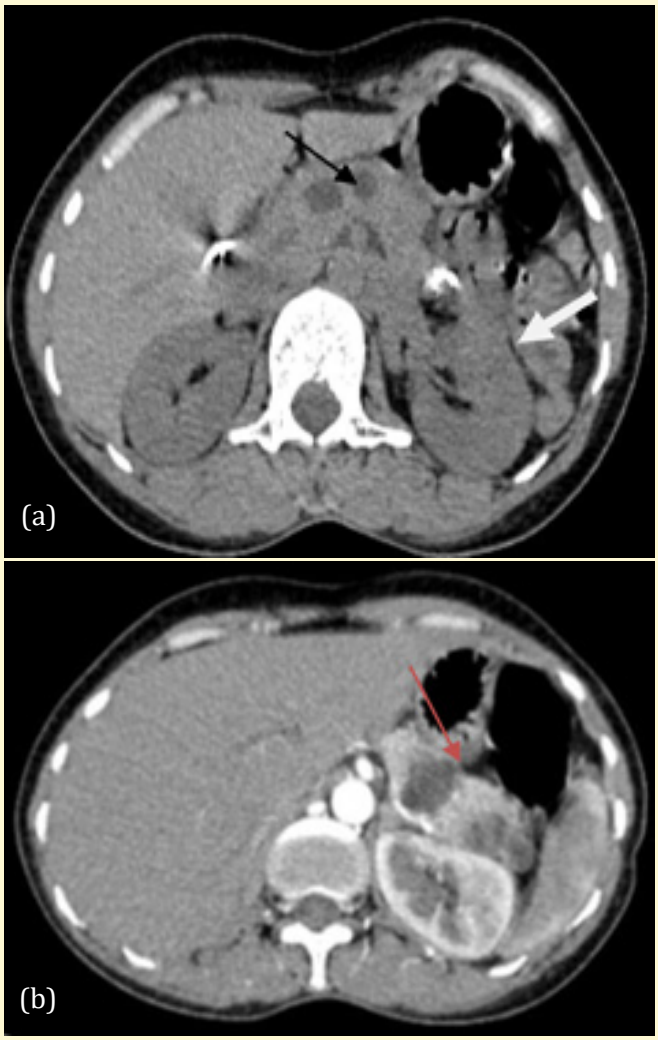
A 32 year old female came for regular health checkup and was incidentally diagnosed with multiple pancreatic cysts and renal cysts on ultrasound.

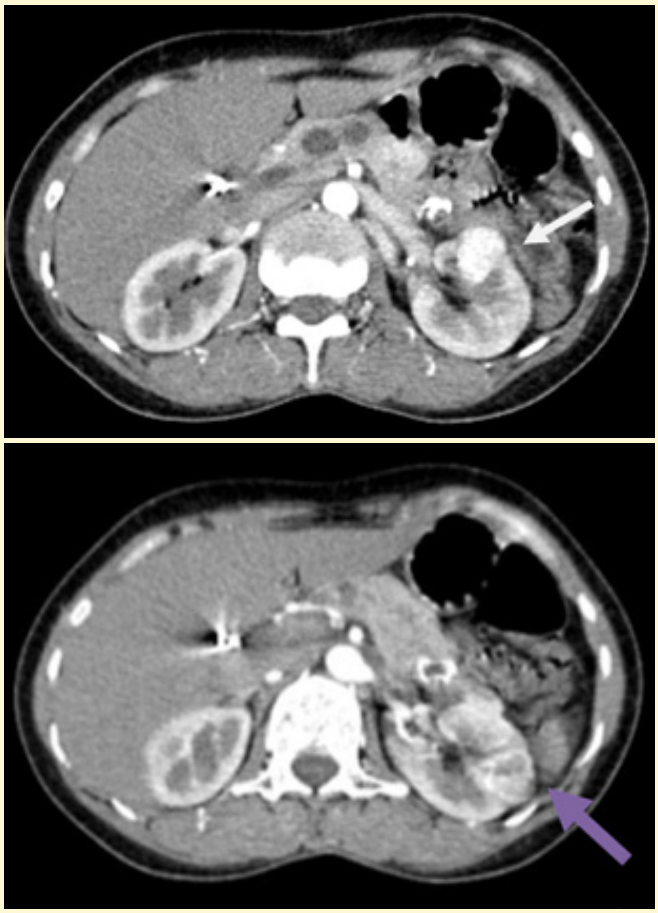


A. B

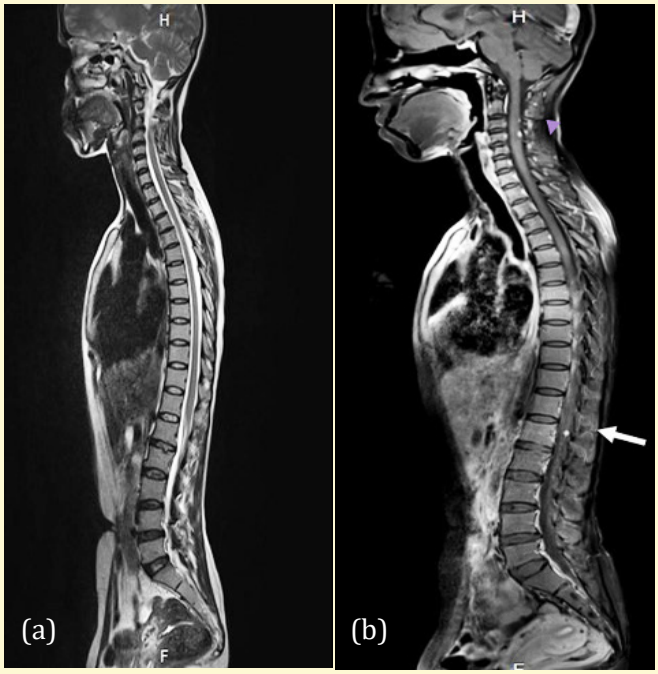
Image A: NCCT shows hypodense cystic (black arrows) lesions in the body of pancreas. Further on close examination , ill defined soft tissue mass noted in the anterior cortex of interpolar region of left kidney (white arrow), causing smooth bulge of the capsule.

Image B: CECT shows non enhancing(red arrow) pancreatic cysts in the tail of pancreas .

ON CECT:



Two solid avidly enhancing lesions noted within the cortex of inter polar region of left kidney with mild capsular bulge.



A.ON PLAIN MRI : The spine appears apparently normal

B. ON CE-MRI: There are three tiny intense nodular enhancements noted within the intramedullary portion of spinal cord posteriorly at C2, C3 & L1 vertebral body levels
– S/o Hemangioblastomas.

HPE of Left Kidney lesions: Clear cell type of Renal Cell Carcinoma.

DISCUSSION:

The Imaging features are suggestive of Von Hippel Lindau Syndrome.

VHL is a familial cancer syndrome caused by a mutation in the VHL tumor suppressor gene, mapped on human chromosome 3p25 with multiple benign and malignant tumors involving various organ systems.

ETIOPATHOGENESIS:

Mutations in the VHL tumor suppressor gene located on chromosome 3 cause VHL. These mutations prevent the production or cause abnormal production of the VHL protein (pVHL). pVHL is primarily responsible for the degradation of hypoxia-inducible factor (HIF), a protein responsible for oxygen regulation in the cells. Abnormal or absent pVHL results in the uninhibited upregulation of HIF and multiple downstream growth factors leading to the formation of cysts and hypervascular tumors characteristic of VHL.VHL is an autosomal dominant disorder, with a prevalence of around one in 36 000 and one in 50 000 live births . Around 80% of patients with VHL inherit the disorder from an affected parent, while it may arise denovo in 20% . The mean age of initial tumor diagnosis in VHL is 26 years (range, 1–70 years). VHL is a highly penetrant disease, with more than 90% of patients developing symptoms by 65 years of age .

IMAGING FEATURES:

VHL Manifestations include: Retina: Retinal Hemangioblastomas(HBs); CNS - Cerebellar and spinal HBs ; Head and neck - Endolymphatic sac tumors ; Pancreas - Pancreatic cysts, Serous cystadenomas, Pancreatic Neuroendocrine tumours ; Kidney - Renal cysts, Clear cell RCCs ; Adrenal gland – Pheochromocytoma ; Reproductive organs - Epididymal cysts , Papillary cystadenoma of epididymis, Broad ligament cystadenoma.

DIFFERENTIAL DIGNOSIS:

The differential diagnosis of VHL contains the various VHL-associated tumors seen in isolation. The disease is associated with numerous tumors, so a differential diagnosis would include isolated retinal hemangioblastoma, renal cell carcinoma, or CNS hemangioblastoma. Patients presenting with pheochromocytoma and no history of VHL may be presenting with either multiple endocrine neoplasia type 2, hereditary paraganglioma-pheochromocytoma syndrome, or neurofibromatosis 1. These diseases are caused by a mutation in a gene separate from VHL. Menière disease is another disease that presents similarly to endolymphatic sac tumors. Renal cell carcinoma may be caused by hereditary leiomyomatosis and renal cell cancer.

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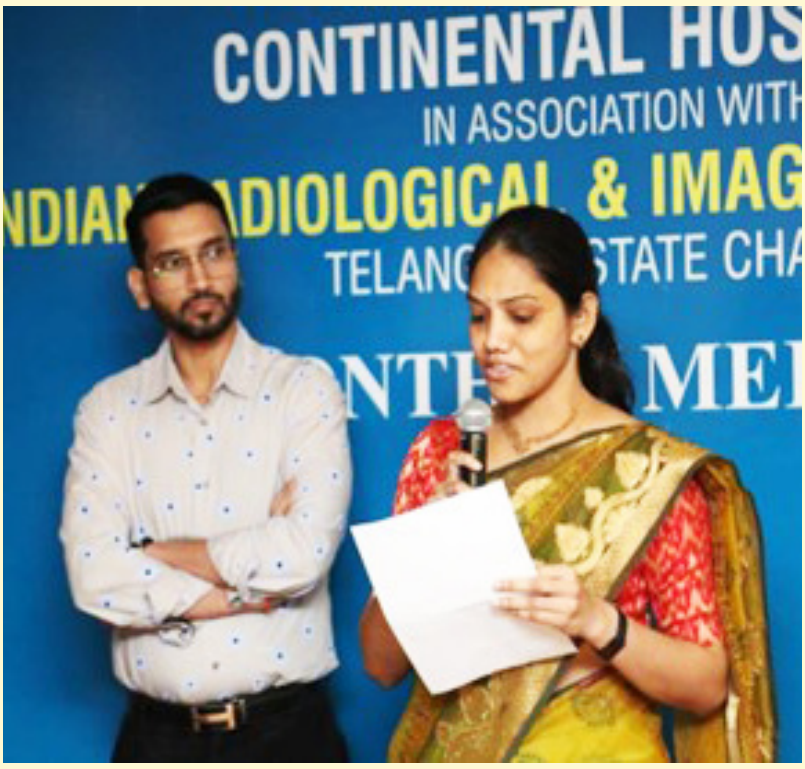
Van Leeuwaarde RS, Ahmad S, Links TP, et al. Von Hippel-Lindau Syndrome. 2000 May 17 [Updated 2018 Sep 6]. In: Adam MP, Everman DB, Mirzaa GM, et al., editors. GeneReviews® . Seattle (WA): University of Washington, Seattle; 1993-2022.

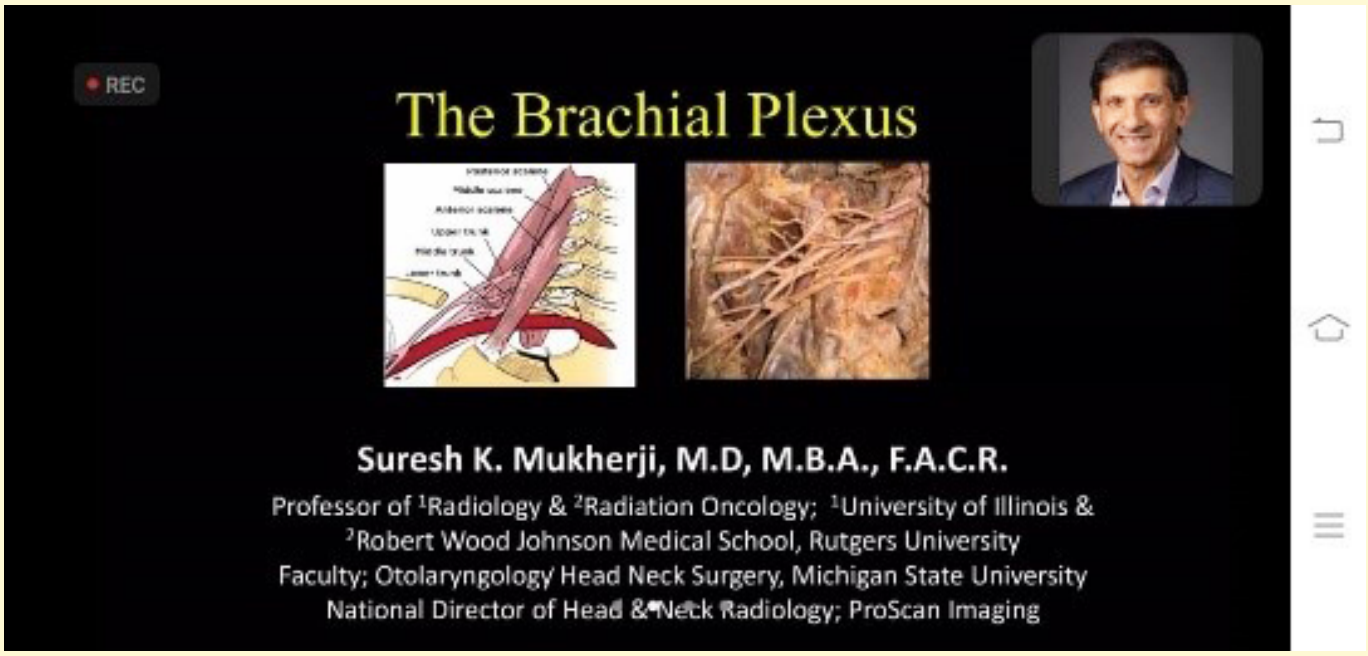
July Monthly Meeting

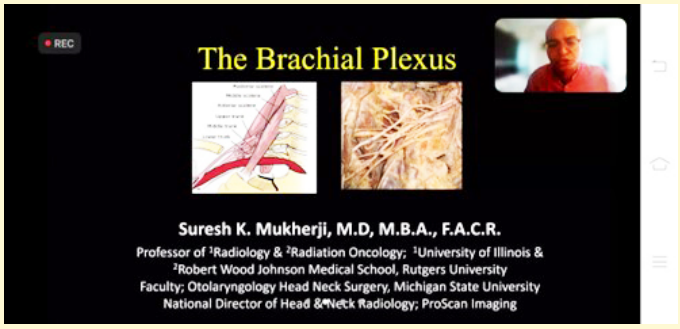


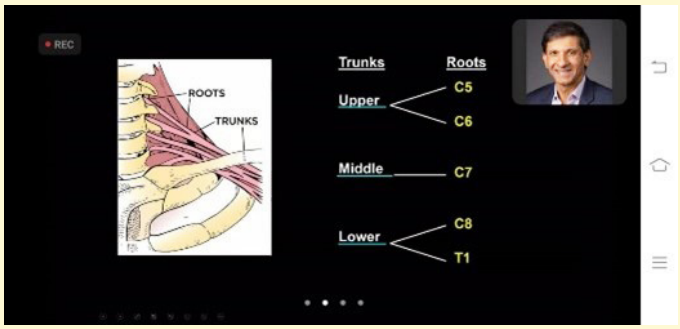
August Monthly Meeting

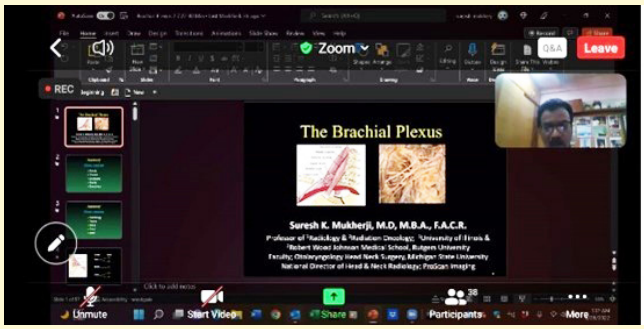
September Monthly Meeting

Webinar on “Head and Neck Imaging”

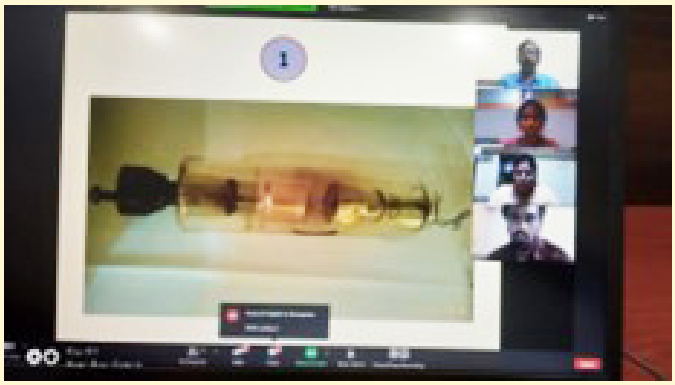








HARP in August



Karimnagar Sub Chapter Of TS IRIA - Programme of Har Ghar Tiranga

UPCOMING CMES

1. ***Monthly Meeting***
   Second Friday of every month

2. ***State Annual Conference***
   October- 2022

3. ***RAC***
   December - 2022

4. ***Webinar – Emergency Radiology***
   December- 2022

5. ***Medical Camp***
   January- 2023

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ICRI (Indian College of Radiology and Imaging):
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AOSR (Asian Oceanian Society of Radiology):
https://theaosr.org

AMS (Asian Musculoskeletal Society):
www.asianmsk.org