CHAPTER 1

Approach to Clinical Radiology Board Exams

INTRODUCTION

'Rome was not built in a day'

We always look for shortcuts for success, but real success can be achieved only by hard work and true dedication to our work. At the same time, we should study smart and invest the available time wisely such that we could cover the vast syllabus. Understanding concept is worth the time instead of just cramming facts. Revision plays a key role so that all our valuable efforts may not be wasted. Presentation plays an important role as preparation so that we could effectively communicate our knowledge to the examiner.

1. Study Smart and Hard

A "strategy" for taking the exam was to simply revise everything before the big day. We spent hours upon hours highlighting everything in exam prep book. In retrospect, we could have saved tons of time by simply identifying the key subjects we did not really understand. The best advice usually comes from your seniors (every one of them—don't be happy with just asking one person) who recently took the exam.

Preparation involves

- Knowledge of the subject.
- Technique or approach.
- Knowledge of the exam itself.

Knowledge of the subject: This comes from your hard work of reading books, seeing more cases, attending various courses. Knowledge to pass is one aspect and knowledge to go out and practice is another aspect—both need to be acquired.

Technique or approach: This consists of various skills you need to tackle each component of the exams.

a. **Spotters:** The success in this component is entirely based on your knowledge, seeing more and more 'Aunt Minnie' type of cases.
b. **Cases writing:** Follow the standard case presentation proforma. Time is very important. Do not get bogged down by a difficult case, resulting in below par performance in an easy case due to lack of time. Unknown diagnoses can be handled by giving appropriate differential diagnosis.
c. **Viva:** FRCR Viva is a case presentation, using standard case presentation proforma. For MD/DNB exam viva, we have equipment/instruments.
d. **OSCE:** Some universities are introducing objective structured clinical examination (OSCE) components or in the process—don't be alarmed and in fact, these are easier components and more objective assessment is done and the examiners are silent observers.

Knowledge of the exam itself: Every university/national board exams are slightly different and you need to understand that; again the exams can vary slightly depending on the examiners as well. It is important that you need to look for information about the exams and be updated with the latest information from the university.

Studying for the exam is undoubtedly a daunting task. Not only can the sheer amount of material that one needs to learn seem overwhelming, but also the vast amount of resources available can be more of a burden than an asset. Residents often scramble to make time to go over every single review book out there, in an effort to have all of their bases covered. This strategy is not only nearly impossible but is likely to be counterproductive. Rather, one should focus on one comprehensive textbook while supplementing with question banks.

It seems daunting with all the resources out there. Don't be afraid to use many, but use them wisely. Ideally you should have read the standard textbooks once or twice during the first two and half years itself. Below is a rough plan:

4 months before the exam (first revision of the following books you have read before)

- Review standard textbooks of radiology
- Go through question banks.

- Make notes on what to write for each question, for a quick last minute revision
- Make small study groups among yourself and start doing case discussion/case presentation.

2 months before the Exam

- Continue above
- Review standard textbooks of radiology physics
- Case atlas for spotters
- Exercise spotter bank—you can practise spotter sets in exam pattern on daily basis/2–3 spotter sets per day.
- Case presentation practice in any radiology museum—you may present each case for 15 minutes without any break, others will help to improve the presentation, likewise practise 10 cases per day.
- Stanley Radiology Museum—permits all eager radiology postgraduates to utilise the radiology museum throughout the year for learning purpose.

< 1 month before the exam

- Final revision of standard textbooks
- Review facts/notes.
- Review questions.
- Practise drawing simple self-explanatory diagrams.

2. Stop Freaking Out!

Seriously, even though this seems like the biggest single moment of your life, it really isn't. This is just a test. Stress decreases your memory. To reduce stress, we recommended that students should just put things into perspective. Like I mentioned earlier, this is just a test! Even if you do not pass the first round, you still have family who loves you, friends that support you, and hopefully a roof over your head. Since passing a test will not affect any of these things, you need not make it gigantic impending thing. Take a few deep breaths in and out and begin to look at the exam as simply the culmination of your radiology education. Look, you have made it far so obviously you are capable of passing a test.

3. You Are What You Eat

Avoid foods with high amounts of sugar (including sugar soft drinks), fried foods, sugar substitutes and heavily processed foods (like white bread, pasta, and pizza). The effect these foods have on your brain is they can cause a condition commonly known as brain fog. Brain fog is best described as those times where everything in your head just seems muddy like you are trying to remember a word and no matter how hard you try you just cannot think of it. Avoid excess caffeine intake. Take fresh fruits and vegetables. For best results, don't just clean up your diet the night before the big test but instead try a month or at least the week before.

4. Sleep

This is a huge one as well. What you might not realize is that if you are getting less than 7–8 hours of sleep a night, then your brain is probably not functioning optimally. We recommend trying to stick to a consistent bedtime schedule. Good sleep will improve you memory and concentrating capability. On the day before the exam, you must sleep for 7–8 hours, only then you can concentrate, recollect and perform well in exam. After a night of good sleep, go to the examination hall with a clear and fresh mind without any anxiety or over expectation.

5. Believe in Yourself

In this final step, we simply want you to stop for a second and take a moment to do a heart check. As I mentioned, you have made it this far. You put up with all the mean, unhelpful scenario of a medical curriculum, you passed all the required tests at your school, and you most of all put your life on hold for your radiology education. You can do this! Ok, so maybe you are not the best test taker in the world, or maybe you have zero confidence. We simply want you to take a few moments to reflect on all that you have achieved so far and begin to believe that you can do it!

1.1 APPROACH TO PRACTICAL EXAM WITHOUT TEARS

The practical examination system is different for MD, DNB, FRCR, EDiR and MICR candidates who often have to attend it in a different institution. Staying confident and not getting stressed is important for success. Feel confident about the portion of the syllabus you have covered instead of worrying about the things you didn't cover.

Before the Examination

Prepare answers to potential viva voce questions in advance and practice interactive sessions with seniors who have previously succeeded in the examination. It is imperative to study the viva voce books and actively participate in online radiology forums offering an extensive repository of radiology cases. The residents may participate in small study groups. It is advisable to present a case to senior faculty members, especially those who have been previous examiners; this will improve your presentation skills and confidence. It will

reduce your exam phobia. Utilise the local/neighbouring institutions, and their radiology museums.

In the Examination Hall

The candidate is expected to be well groomed and formally dressed to create a good first impression on the examiner and the candidate should reach the examination hall one hour before the exam which will calm the unnecessary nervousness. Avoid nervousness while presenting cases, to convey the content effectively to the examiner.

If a film is given for discussion, begin by describing the film without delay. Start with the modality, anatomical region, radiographic view, etc., and avoid staring at the film in silence. While describing a case, the candidate should, preferably, face the film instead of looking at the examiner. Only after completion of the case description and concluding the case, candidate should turn to face the examiners for further remarks and questions.

Always maintain eye to eye contact with examiner while answering questions. First start describing baseline modality, then proceed to higher modalities, so don't jump into higher diagnostic modalities, without describing the findings in the baseline modality. Choose the modality based on relevant clinical history provided.

During the discussion, listen keenly and understand the examiner's line of questioning. During the course of the examination, the examiner may drop hints if the candidate seems to be going down a wrong path. It is vital to catch such 'lifelines' and change one's line of thought, and answer accordingly.

Do not criticize the quality of film given. Avoid touching the film or using the tip of a pen as a pointer while describing findings of a case. When necessary, use a pointer (like a plastic ruler) rather than the finger. Never contradict/argue with the examiner.

Use correct terminology and definitions during case presentation and while answering specific questions. If the examiner says "plain radiograph", the candidate may use the same terminology. Use of the phrase "I don't remember" is preferable to "I do not know" when the candidate does not know the answer to a certain question. If the question was not clearly heard, politely request the examiner to repeat the question. Listen carefully; answer appropriately to the questions asked especially when the examiners are giving clues.

When the diagnosis is not known/when stuck in a case:

In situations when one fails to arrive at a diagnosis it may be useful to start thinking aloud, saying that one is looking at certain parts, e.g. I am now looking at the lung, I am now looking at the cardiac shadow, etc. Try to pick up the findings while talking, instead of waiting to get the diagnosis. Even if you cannot come up with an aetiological diagnosis, it is possible that, while discussing with the examiner and listening intently to the guiding question, one will strike upon the diagnosis.

Checklist before attending practical exams:

- White over coat.
- Radiology exam kit.
- Hall ticket.
- Number badge and TLD badge.
- Dissertation/Logbook

Come relax and attend the exam.

1.2 TIPS FOR WRITING THEORY EXAM

Theory exams form an important part of the assessment of a candidate's knowledge (as well as imagination!). Expect the best, plan for the worst and prepare to be surprised… and to be prepared is half the victory. The other half depends on your presentation style and the evaluator.

Time Management

Totally we have 180 minutes, the first 5 minutes to be allotted to writing the registration number, page number and drawing margins in the answer sheet, then use the next five minutes for reading the questions and make sure to keep the last 15 minutes for verification of the answer sheet.

In MD exam pattern, there will be 25 min for each essay and 10 min for each short note, whereas for DNB (Diplomate in National Board) examinations there will be 15 mins for each short structured question. For each mark spend 1½ minutes writing, plan accordingly.

Question Type

Varies on a course and university basis. In most of the university examinations (MD) they have 2 long questions and 10 short notes, while in DNB examinations they have 10 short structured questions, each carrying 10 marks (Table 1.1). It is vital not to spend more time on the initial questions; otherwise you will run short of time for attempting rest of the questions.

Different universities have different marking schemes. Some universities allot 20 marks for each long note and 6 marks for each short note so that you have to spend 35 minutes for each long note and 8 minutes for each short note.

Table 1.1: Pattern of various examinations and sample for planning the exams

Exam type		*Pattern*	*Marks*	*No. of questions*	*Pages*	*Duration (marks × 1.6)*
DNB		Long notes	10	10	6–7	15 minutes each
MD		Long notes	15	2	10–12	25 minutes each
		Short notes	7	10	3–4	10 minutes each
FRCR 1	Anatomy module	MCQs	200	100	–	90 minutes
	Physics module	MCQs	200	40 × 5 (true or false)	–	2 hours
FRCR 2A		Single best answer questions (2 sets)	–	120	–	3 hours (for each paper)
FRCR 2B		Rapid reporting	–	30	–	35 minutes
		Long cases	–	6 cases	–	60 minutes
		Viva (2 sessions)	16 marks for each session	–	–	30 minutes (each session)

Answer Contents

For long answers, it is necessary to write 10 to 12 pages each with **diagrams/tables/boxes/classification** minimum of 2 to 3 per essay. For short notes—3 to 4 pages each, diagrams/tables minimum 1 per short note. Pay attention to writing the page number in the top and write 15–18 lines per page. Use spacing between words and underline important key points/sentences. Using **bullets, flow diagrams, diagrams, boxes/squares for highlighting the key points** is more vital if handwriting is not good (or) if you run out of time.

Use colour pens and sketches for diagrams, headings and **underlining important key points** and subtitles. **Answer all the questions and in the order** given in question paper. Write in medium size letters. There must be logical organization of the contents or the key points with one or two points in one paragraph. Writing more will not fetch more marks. The examiner looks for key points. If they are covered, you will get marks irrespective of the number of pages written. Writing controversial points is better avoided.

Good presentation in the examination is a skill and this differentiates a topper from a mediocre student (Table 1.2).

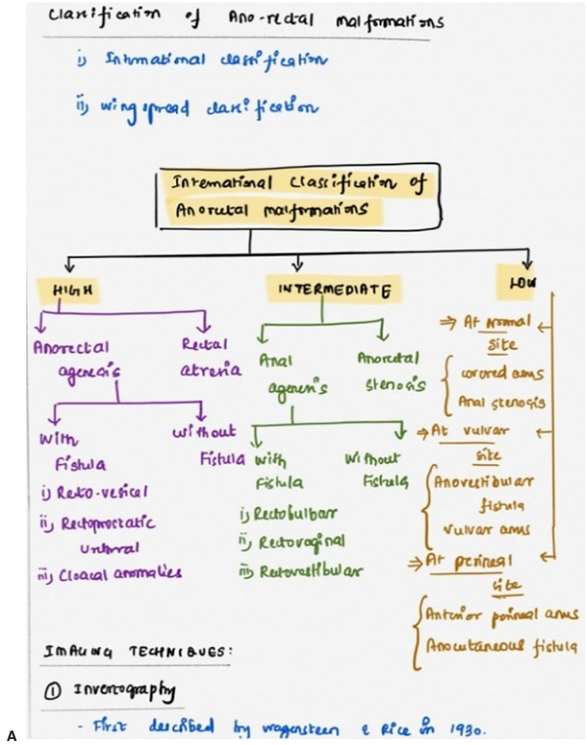
Some model theory exam answer papers given as follows for use of colour pencils/charts/diagrams (Fig. 1.1A to D).

Table 1.2: Sample answer outline for theory questions

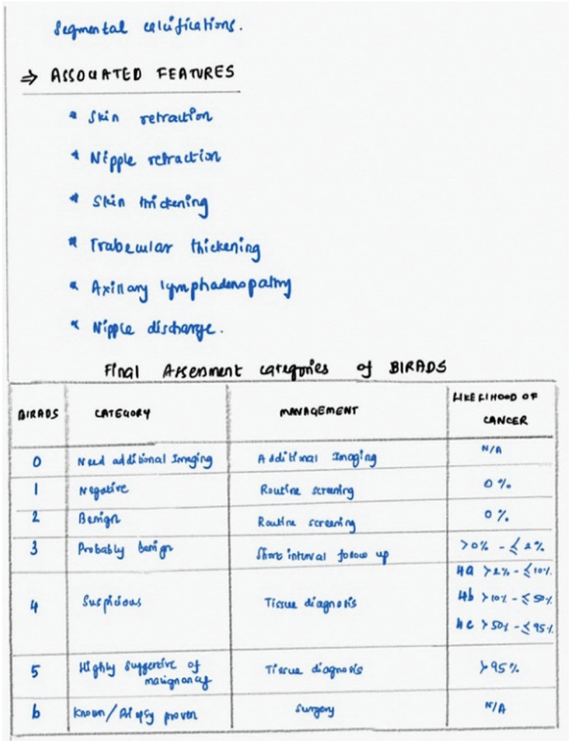
1. Definition
2. Demography
3. Incidence
4. Causes/anatomy/types /embryology
5. Clinical features
6. Location
7. Pathophysiology
8. Stages/manifestations
9. Radiology: Plain X-ray, barium, USG, CT, MRI, DSA, intervention, PET/SPECT
10. Diagnostic test
11. Associated conditions/manifestations in other systems
12. Differential diagnosis
13. Complications
14. Prognosis
15. Management—further investigations and treatment.

Scurvy Radiological Findings

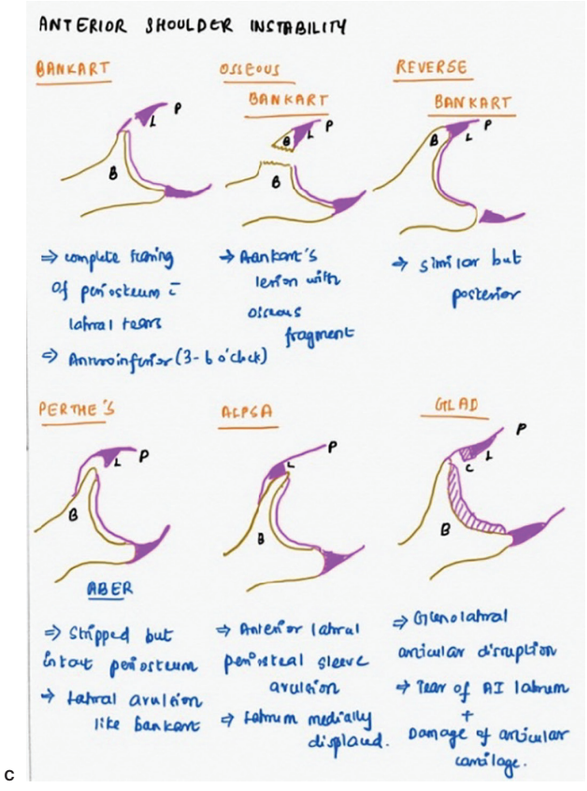
- Generalized osteopenia
- **Cortical thinning:** "Pencil-point" cortex.
- Periosteal reaction due to subperiosteal hemorrhage
- **Scorbutic rosary:** Expansion of the costochondral junctions, may relate to the fracturing of the zone of provisional calcification during normal respiration. Similar to the rachitic rosary appearance seen in rickets.
- Haemarthrosis
- **Wimberger's ring sign:** Circular, opaque radiologic shadow surrounding epiphyseal centers of ossification, which may result from bleeding.
- **Frankel's line:** Dense zone of provisional calcification
- **Trümmerfeld zone:** Lucent metaphyseal band underlying Frankel's line.



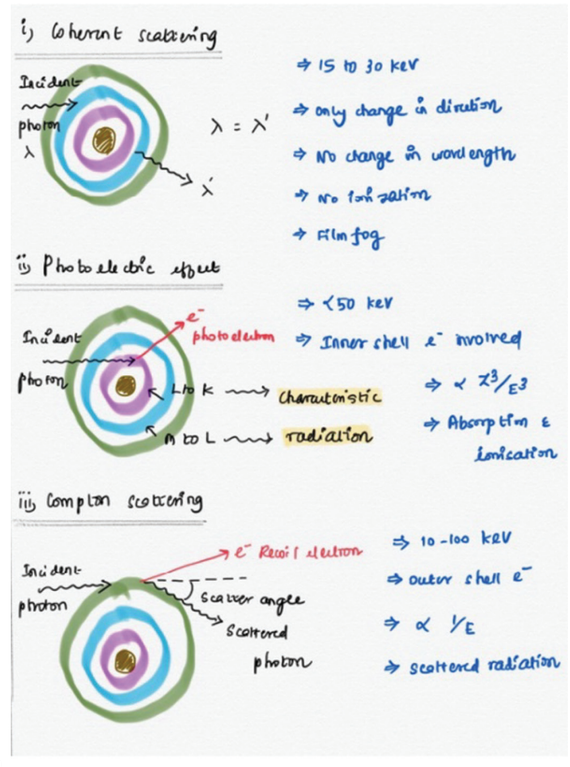
A



B



C



D

Fig. 1.1A to D: Sample answer sheets

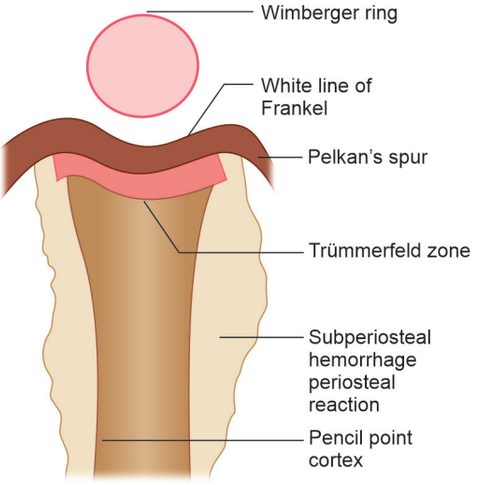


Fig. 1.2: Schematic diagram of scurvy findings

- **Pelkan spur:** Metaphyseal spurs which result in cupping of the metaphysis.

Diagrams are very important, in theory answersheets where we can show the important points in easy and efficient way (Fig. 1.2).

CHAPTER 2

Radiology Case Presentation—General Guidelines

Radiology case presentation guidelines followed by the Stanley Medical College is similar to case presentations in various exams and FRCR Viva. It includes short cases and long cases. These guidelines will aid in the structuring of the radiological report and also its evaluation by the examiners. The report should be succinct and relevant. The examination includes X-ray, CT, ultrasound, radionuclide and MR scans. Candidates will be provided with a double viewing box/monitor, a magnifying glass and a ruler. A brief case history and other relevant clinical data will be provided. Each case may comprise up to four modalities in films or dicom images depending on spotter, short or long case. These vary in complexity and difficulty; some require more time for analysis and reporting than others. It should be ensured that sufficient time is allocated to report each case adequately.

Candidates are expected to start reading the films with a little delay as soon as the films are mounted on the viewing box. There is no need to list dates of investigations or to list all sequences unless these factors are of direct relevance. Similarly, there is no need to repeat the clinical information that has been given, but should use the clinical information in interpreting the observations.

A better performance will be expected in the interpretation of common and routine investigations rather than highly specialised investigations. Examiners mostly will show examples from the major clinical radiology sub-specialties. Candidate's power of observation and deduction will be assessed. A logical and informed approach to film interpretation, as well as a clear ability to debate the merits, relevance and role of further investigative modalities, will be expected. Examiners may ask supplementary questions to assess the understanding of the given clinical problem.

In reaching a conclusion, diagnoses should be given in the order of probability. Long lists regurgitated indiscriminately make it difficult for the faculty to assess the true acumen of candidate. In some cases, the correct diagnosis can be directly arrived at. In others, further views or investigations will be helpful and it is important that the candidate clearly substantiates the reasons for wanting the additional data.

During presentation, the faculty will attempt to assist the candidate to perform to the best of his/her ability. Do not stop the discussion unless the examiner interrupts in between. The candidate has to discuss all the positive findings and points relevant to his/her diagnosis. The candidate should not stop presenting a case unless he does not get the answer for a **"What Next"** question. Listen carefully for any clues provided and must not be afraid to ask for clarification regarding questions asked and doubts regarding the patient's clinical presentation. The examiner will point out any incorrect statements, inappropriate further investigations and management made by the candidate.

The amount of discussion that takes place on each case will vary and is at the discretion of the individual examiner. The actual number of cases shown is immaterial. It is more important that a meaningful interaction is taking place with the faculty. Patient confidentiality must be respected.

It is strongly advised to follow a standard format (Stanley Medical College Radiology resident reporting format), i.e. approach to **diagnosis first, followed by management and discussion** about the condition (Table 2.1).

Table 2.1: Sample presentation template	
Stanley Medical College—radiology resident presentation template–**what next?**	
1	**Name:** **Age/Sex:** **Occupation:** **Date and time:** **Interval imaging:** **Referred from:** Emergency/OPD/IPD (if relevant and provided)
2	**Clinical presentation:** **Clinical examination:** **Biochemical results:** (if relevant)
3	**Radiological techniques, part in the study, and observations (positive and negative findings, associated findings, complications related to the condition, other organs)** In this section, the candidate should record his/her observations of all the imaging studies available on the films/monitor, including detailed description on positive findings as well as relevant negative findings and brief note on remaining organ systems. Image description of lesion includes site, size, shape, margins, extent, characterisation, skip lesions, nodal involvement/ metastasis and enhancement pattern as appropriate.
4	**Structured analysis and interpretation through logical approach (A-C-B-D-E-F approach)**—there is interpretation of the observed findings which can provide explanation regarding the etiology and complications. For example, description whether lesion being observed is benign or malignant, infective rather than neoplastic, giving your reasons for the same. There is reanalysis with patient data, clinical findings, previous images and biochemical values to arrive at the final diagnosis. **A-C-B-D-E-F approach:** **A**natomy (epicenter) and age **C**haracteristics (size, shape, margins, extents, diffusivity, necrosis, vascularity) **B**ehavior and extension (skip lesions, adjacent organs, enhancement pattern, regional and distal spread) **D**ifferential work up and diagnosis: Substantiate with positive and negative findings. (Further elaborated in the latter part of the section) The steps A-C-B-D should happen in the Radiologist's thought process (Not a part of Reporting template) **E**valuation further and **F**ollow-up imaging
5	**Principal diagnosis (substantiate with positive and negative findings):** Based on the candidate's interpretation he/she should attempt to come to a single diagnosis especially in cases where the findings are classical (e.g. tuberous sclerosis). **Relevant differential diagnosis—balance of probability:** In classical cases, there is no differential diagnosis. If the case is complex, then he should state which diagnosis he/she feels is most likely and then list other possibilities in order of likelihood, in the differential diagnosis section which should be limited to relevant 2–3 conditions. However, in this book we have also added some rarer differential diagnosis for the purpose of elaborate discussion and to enlighten students.
6	**Management (recommended further investigations—treatment goals)/applied radiology in treatment** A. *Critical cases*–immediately inform the referring/appropriate clinician (e.g. acute infarct, EDH with brain herniation, pulmonary embolism, encephalitis, pneumothorax, cardiac tamponade, pseudoaneurysm, acute abdomen) B. *Noncritical cases:* Relevant further investigations—laboratory, radiological and nuclear medicine, HPE (reasons for wanting additional data) to confirm the diagnosis. C. *For tumours:* Confirmatory HPE examinations followed by local and locoregional staging by MRI/CT scan. PET scan for systemic staging. Treatment depends on the stages/cellular type of disease. Final decision on treatment is based on discussion in multidisciplinary tumour board. Similarly, if the candidate makes a diagnosis of an abscess or tumour, he should indicate if a drainage or biopsy is appropriate.
7	**Brief discussion about the condition in viva:** Here the candidate should discuss about the diagnosis of his case and briefly discuss the etiopathology, clinical presentation, treatment options and some note on differential diagnoses.

SPECIMEN REPORTS

Case 1: A six months old child seen unconscious in the emergency department.

Case history: Age—6 months/female child

Clinical presentation: Unconscious/no history of trauma.

Radiological Techniques and Observations

1. Non-contrast CT scan brain (Fig. 2.1)
2. X-ray chest (Fig. 2.2)/X-ray thigh (Fig. 2.3)/X-ray knee (Fig. 2.4)

Observations (*see* Figs 2.1 to 2.4)**:** A non-contrast head CT shows chronic subdural hematoma in bilateral frontal convexity and new subdural haemorrhage in right frontal convexity and posterior interhemispheric SDH. Minimal midline shift is seen to the left side. No skull fracture is seen on these images. X-ray thigh AP view shows displaced spiral midshaft fracture in right femur. X-ray left knee shows corner fracture in upper end of tibia. Chest X-ray shows fractures of the right first to eighth ribs in posterior aspect with evidence of callus formation indicating healing. There are also fractures of the left fourth to ninth ribs posteriorly but no associated

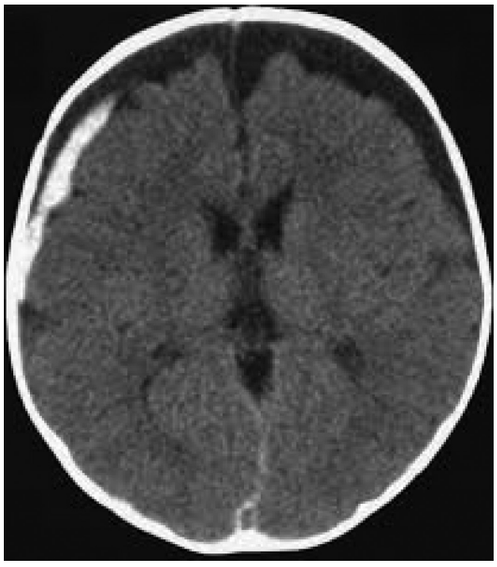


Fig. 2.1

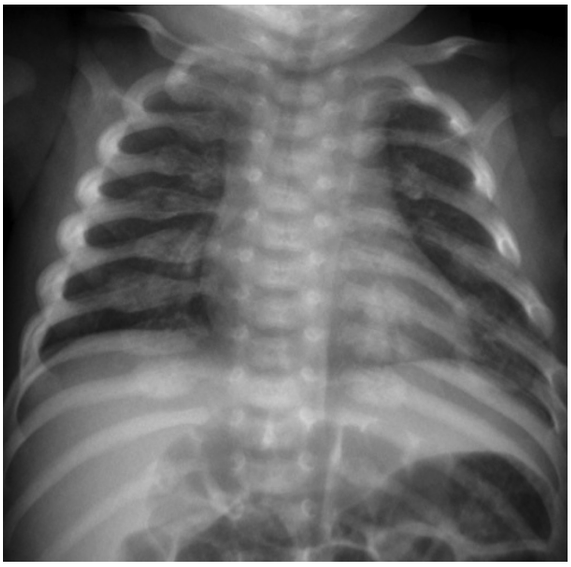


Fig. 2.2

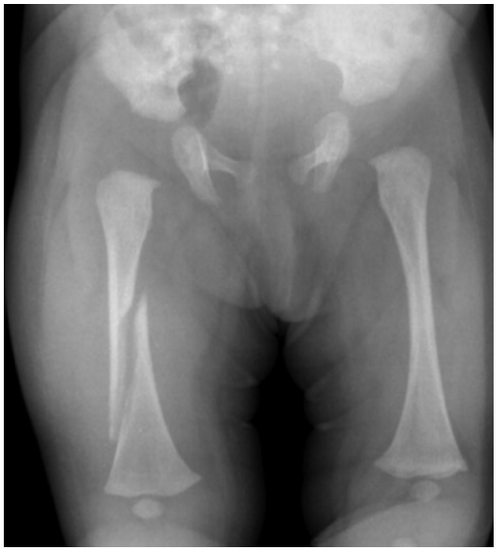


Fig. 2.3

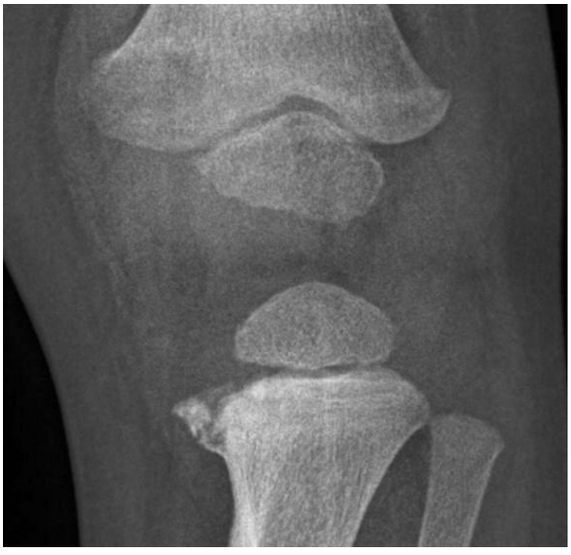


Fig. 2.4

callus or periosteal new bone, suggesting that these fractures have occurred very recently. The lung fields are clear.

Analysis and Interpretation

CT brain: Extra axial fluid collection lies within the sub-dural rather than the sub-arachnoid space. It contains both high and low attenuation material indicating that it is likely to represent an acute or chronic sub-dural haematoma.

Chest X-ray: Shows multiple rib fractures in different stages of healing.

X-ray thigh: Spiral midshaft fracture in right femur

X-ray knee: Corner fracture in upper end of left tibia.

Principal diagnosis: Non-accidental trauma, since the fractures are at different stages of healing and some new fractures also seen and acute on chronic subdural hematoma in brain in absence of skull fracture adds to the diagnosis of non-accidental trauma.

Differential diagnosis: Consider accidental trauma. This appears unlikely in view of the posterior rib fractures of different ages.

Management: The patient needs an urgent neurosurgical opinion and the child protection service must be alerted. A skeletal survey should be performed to look for other fractures and to ensure that there is no evidence of any other skeletal abnormality, such as osteogenesis imperfecta.

Brief discussion about the condition: Non-accidental trauma is a complex injury in infants and young children as a result of abuse. Diagnosis of non-accidental trauma has direct impact on medical, social and legal outcomes of children and families. The classical metaphyseal corner or bucket handle fracture is virtually pathognomonic for abuse. Lateral and posterior rib fractures are highly specific for abuse. Patterns of skull fracture that suggest child abuse are multiple 'eggshell' fractures, occipital impression fractures, fractures crossing sutures. CNS injury especially subdural hematoma is common in child abuse. Visceral injuries include liver, pancreatic laceration and adrenal bleeding are specific for child abuse.

Case 2: A 12 months old child with abdominal distension and seizure.

Case history: 12 months/male child

Clinical presentation: Complaints of abdominal distension for 2 months with 1 episode of seizure.

Radiological investigations and observations: CT brain, MRI brain and CT abdomen.

Observations on CT brain (Fig. 2.5)**:** Hyperdense extra-axial mass lesion noted in right fronto-parietal convexity and left parietal convexity.

Observations on MRI brain (Fig. 2.6): TI post-contrast image shows heterogeneously enhancing extra-axial dural mass noted in bilateral parietal convexity.

Observations on CECT abdomen (Fig. 2.7): Large heterogeneous mass soft-tissue dense mass in left suprarenal fossa extending into midline with coarse stippled calcifications.

Analysis and Interpretation

CT/MRI brain: Diffuse dural mass lesion involving bilateral parietal and right frontal convexity in a 12 months old child.

CT abdomen: Soft-tissue mass in left suprarenal fossa with coarse stippled **calcifications,** this is likely to relate to a malignancy in the adrenal gland.

Principal diagnosis: Neuroblastoma in left adrenal region with cranial metastasis as my principal diagnosis considering the age of the child.

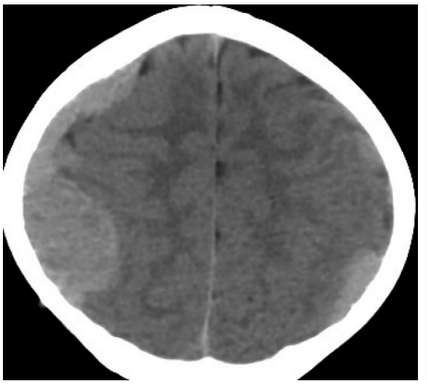


Fig. 2.5

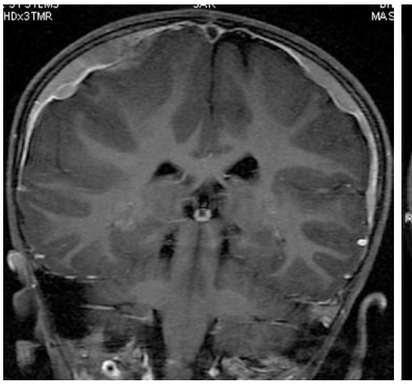


Fig. 2.6

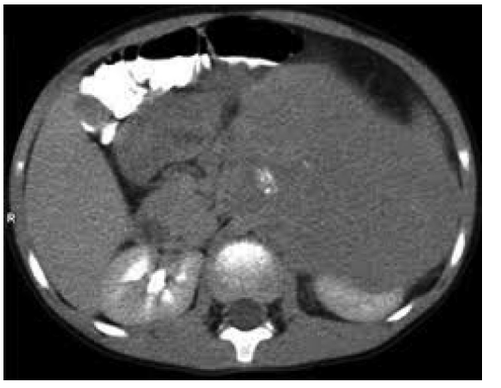


Fig. 2.7

Differential diagnosis: Adrenal carcinoma can rarely occur in this age group and may calcify but is unlikely to extend intraspinally. Wilms' tumor is intrarenal, only occasionally calcifies and rarely metastases to bone so should not give these appearances.

Management: **Further investigations required**—ultrasound, MRI, bone/MIBG scan, PET.

An ultrasound scan would confirm whether the calcified mass lies within the adrenal gland.

Bone scan/I^{131} MIBG scan: Nuclear scintigraphy with areas of increased activity suggests skeletal metastases. An MRI scan and PET scan would be required for staging and monitoring response to treatment. Bone marrow aspiration and catecholamine estimation are usually also performed. A biopsy may also be required.

Brief discussion about the condition: Neuroblastomas are tumors of neuroblastic origin. They represent the most common extracranial solid childhood malignancy. Common metastatic sites are bone, liver, lung, pleura and brain.

SAMPLE REPORTING TEMPLATES

There is no specific standardized reporting template available, here we provided some sample pathological reporting templates used in our department, and reporting templates need constant revision based on recent updates.

Central Nervous System

Arachnoid cyst: Left anterior temporal arachnoid cyst compressing adjacent anterior and medial temporal lobe with adjacent bony scalloping. It measures 40 (TR) × 39 (CC) × 47 (AP) mm. It elevates and displaces left middle cerebral artery medially. There is significant mass effect on the left lateral ventricle with midline shift to right (7 mm)—Galassi type-III.

Medulloblastoma: Evidence of well-defined large heterogeneously enhancing mass lesion measuring about 3.5 × 4.5 × 5.3 cm (AP × TR × CC) arising from the vermis (roof of fourth ventricle) within the 4th ventricle causing moderate obstructive hydrocephalus and transependymal seepage of CSF and cerebellar tonsil herniating 6 mm below the foramen magnum. Fourth ventricle CSF is seen anteriorly as cleft. There is significant compression on the brain stem. No intra-axial edema. The lesion is highly cellular showing T2 hypo/isointense signal with restricted diffusion, areas of necrosis and calcifications. MR spectroscopy shows reduction of NAA, elevation of choline, elevated lipid lactate peak and taurine peak. Entire neuraxis imaging shows no leptomeningeal enhancement or drop metastasis. Mass is extending into the foramen of Luschka on left side and foramen of Magendie (in cases of ependymoma)–medulloblastoma–group 4.

Ectopic posterior pituitary gland: Ectopic posterior pituitary gland along the median eminence of hypothalamus with absent pituitary stalk. Thin anterior pituitary—hypoplastic.

Choroidal fissural cyst: Choroidal fissural cyst measuring about 15 × 7 mm causing mild mass effect over the left hippocampus.

High grade glioma: 2.8 (TRANS) × 5.7 (AP) × 3.6 (CC) cm size hypointense lesion in T1w, hyperintense in T2W sequences seen at the peritrigonal, left parieto-occipital lobe compressing the trigone and posterior horn of left lateral ventricles. There is surrounding perilesional oedema extending to the left optic tract. The lesion extending along the splenium of corpus callosum to the right peritrigonal parietal white matter. It shows peripheral diffusion restriction. There is mild midline shift to the right (5.6 mm). The lesion shows increased vascularity with areas of hemorrhage. MR spectroscopy shows significant elevation of choline, lactate and lipid. On contrast administration the lesion shows heterogeneous enhancement with increased regional CBF and CBV in perfusion imaging. On tractography no significant reduction in the density of the corticospinal tracts and other white matter fibers. Left arcuate fibers are compressed and displaced medially. On finger tapping in the right hand, cortical activation was observed along the superior and lateral aspect of left precentral gyrus. The lesion is well away from the hand motor area.

On word generation (language) fMRI, activation noted in left inferior frontal gyrus and left superior temporal gyrus, left supramarginal gyrus along antero-superolateral aspect of the lesion and parietal opercula anterior to the lesion. Minimal activation also noted in the right supramarginal gyrus and right angular gyrus.

Low grade glioma: Well-defined heterogeneous mass lesion in the left supramarginal gyrus, left angular gyrus and indenting on the posterosuperior aspect of left superior temporal gyrus. The lesion shows areas of hemorrhage. No evidence of mass effect/prominent arterial feeder/contrast enhancement. MR spectroscopy shows mild elevation of choline, lactate and lipid.

DNET: Well-defined T2 hyperintense lobulated expansile, cortical based 42 mm (AP) × 35 mm (transverse) × 38 mm (craniocaudal) size lesion in the left frontal parasagittal region (left paracentral lobule) with facilitated diffusion and partial suppression in FLAIR. No evidence of

surrounding perilesional vasogenic oedema/midline shift. On spectroscopy the lesion shows elevated choline, lactate and decreased NAA. No evidence of any other T2/FLAIR grey matter hyperintensity. Above MR features favours dysembryoplastic neuroepithelial tumor (DNET) than low grade glioma.

Functional MRI: Activation of right motor cortex (right precentral gyrus) observed, during finger tapping with left hand. On finger tapping with right hand, cortical activation was observed along the superior and medial aspect of left precentral gyrus and adjacent paracentral lobule and also along the posterior rim of the left paracentral lobule lesion. On bilateral finger tapping, bilateral motor cortex, supplementary motor cortex activation was noted (left > right). No evidence of plasticity.

Tractography: Shows displacement of the corticospinal tract posteriorly. No evidence of infiltration/destruction of corticospinal tract. Corticobulbar fibres are displaced anteriorly and inferiorly. A few posterior fibres were destroyed. Superior longitudinal fasciculus is displaced laterally. Superior fronto-occipital fasciculus appears normal.

Atypical teratoid rhabdoid tumor (ATRT): Evidence of large solid and cystic mass lesion measuring about 7.1 × 6.4 × 7.5 cm (AP × TR × CC) in left side of posterior fossa with significant mass effect over the cerebellum and brainstem. Enhancing solid nodules with hemorrhagic areas seen along the medial aspect of mass. There is compression and narrowing of the 4th ventricle and aqueduct with moderate obstructive triventricular hydrocephalus and periventricular seepage of CSF. Mass is pushing the brainstem and cerebellum to right side. No extension to IAC. Superomedially cystic component is extending up to the pineal region/posterior 3rd ventricular region. Features favour the possibility of atypical teratoid rhabdoid tumor (ATRT) in left side posterior fossa with significant mass effect.

Brainstem glioma: 29 × 28 × 27 mm (AP × TR × CC) size expansile exophytic lesion in the left side of pontomedullary junction and left middle cerebellar peduncle with areas of hemorrhage in the inferior aspect. There is expansion of left middle cerebellar peduncle with compression on the left side of fourth ventricle. There is significant surrounding edema in the medulla, left cerebellum, bilateral thalamus and posterior capsular regions. MR spectroscopy shows marked elevation of choline, marked reduction of NAA peak and presence of lactate. Features favour brainstem glioma. DTI shows displacement of pontocerebellar fibers laterally and corticospinal tract fibres medially. Mass effect noted in the left corticospinal tract without significant white matter loss. Leptomeningeal enhancement seen in/around the spinal cord in the cervicodorsolumbar region—**suggestive of drop metastasis.**

Pineoblastoma: 22 mm (AP) × 19 mm (TR) × 23 mm (CC) size well-defined heterogeneously enhancing lobulated mass lesion with peripherally dispersed calcification in the pineal region extending to the upper part of the aqueduct with areas of haemorrhage causing dilatation of third and lateral ventricles. Tectal plate and suprasellar region appear intact. No evidence of leptomeningeal metastasis. On spectroscopy the lesion shows mildly elevated choline and decrease NAA. The lesion shows diffusion restriction—above features favours pineoblastoma than germinoma.

***Choroid plexus papilloma*:** Large T2 heterogeneous mass with frond-like pattern in the trigone of left lateral ventricle. It measures 6.8 × 4.8 × 6.7 cm. No demonstrable calcification or hemorrhage is seen at present. Both lateral, third and fourth ventricle are dilated. No evidence of acute infarct. Mild midline shift to the right measuring 4 mm. There is moderate dilation of right lateral ventricle and gross dilatation of left lateral ventricle. The lesion shows homogeneous enhancement with cauliflower-like appearance. No obvious evidence of intraparenchymal extension/parenchymal edema. There is prominent arterial supply from the lateral posterior choroidal artery, anterior choroidal artery and the prominent choroidal vein drains into the thalamostriate vein. No obvious evidence of leptomeningeal/drop metastasis. MR spectroscopy shows elevated choline significantly decreased NAA and creatine.

Hypothalamic hamartoma: Well-defined non-enhancing T1 hypointense long TR mild hyperintense lesion in the tuber cinereum/hypothalamus region causing partial narrowing of the third ventricle and abutting the optic tract. MR spectroscopy of the lesion shows mildly decreased NAA peak.

Diffuse cerebral atrophy (children)—Grey matter neurodegeneration: GM gangliosidosis and neuronal ceroid lipofuscinosis. Suggested clinical/biochemical correlation. Other possibilities include: Glycogen storage disorder. Mucolipidosis, multiple sulfatase deficiency, post-encephalitis sequelae.

Cerebellar atrophy (children): Moderate to severe cerebellar atrophy with ex-vacuo dilatation of 4th ventricle. FLAIR hyperintense cerebellar cortex (INAD, Marinesco-Sjögren, late onset GM2 gangliosidosis, late infantile NCL).

Moderate to severe **cerebral and cerebellar atrophy** with increased T2W signal intensity in the white

matter and excess iron deposition in the basal ganglia. Possibilities include: Neuro degeneration with brain iron accumulation–pantothenate kinase associated neurodegeneration (Hallervorden-Spatz disease), oculodigital dental dysplasia, Kufor-Rakeb syndrome (pallidopyramidal degeneration), neuronal ceroid lipofuscinosis, glycogen storage disease.

Severe cerebellar atrophy (children): Severe cerebellar atrophy with T2W/FLAIR increased cerebellar cortex signal intensity. Evidence of flat pons. Clava hypertrophy noted. No evidence of basal ganglia atrophy/basal ganglia T2 hyperintensity/supratentorial hypomyelination. Mild increased iron deposition in globus pallidus. MR spectroscopy of the brain parenchyma shows normal spectral pattern. No significant reduction of NAA/elevation of choline. No abnormal metabolites seen. Possibilities include: Infantile neuraxonal dystrophy, congenital disorder of glycosylation (Pmm2-CDG)—no evidence of flat pons seen, Marinesco-Sjögren SIL1, ceroid lipofuscinosis (late), juvenile GM2 (late).

Arteriovenous malformation (AVM): Abnormal cluster of vessels (arteriovenous malformation nidus) measuring about 27 × 19 mm in the inferolateral aspect of the left inferior parietal gyrus in the angular gyrus and adjacent parieto-occipital region. No acute hemorrhage/surrounding edema. No obvious evidence of infarct/atrophy. Lesion is fed by cortical branches of left MCA and left PCA. Venous drainage to superior sagittal sinus. No adjacent gliosis. Spetzler-Martin grade-I.

Aneurysm: 7.3 × 6 × 8.5 mm size multilobulated saccular aneurysm seen arising from the M1 bifurcation of right middle cerebral artery/anterior communicating artery. The aneurysm is arising from the posterior aspect and pointing posterosuperiorly. Neck of the aneurysm measures 2.3 mm. No obvious evidence of any other aneurysm, wall irregularity/thrombus/vasospasm/subarachnoid hemorrhage/infarct/bleb/lobulation.

Giant cavernous malformation: 37 (TR) × 22 (AP) × 28 (CC) mm size T2W multilobulated lesion in the right inferior frontal gyrus, frontal central white matter extending near the anterior body of right lateral ventricle with minimal surrounding oedema. It shows hyperintense signal with popcorn appearance. The lesion blooms in SWI sequence in the periphery. Mild midline shift to left (2 mm). No obvious dilated feeding artery/draining vein seen. Multiple cavernomas—**familial multiple cavernous malformation syndrome.**

Developmental venous anomaly: Abnormal dilated venous channels draining into the collector subependymal vein in the right frontal region.

Molar tooth malformation—Joubert syndrome: Abnormal configuration of the midbrain with typical "molar tooth" appearance. Enlarged 4th ventricle with "bat-wing" configuration and enlarged cisterna magna. Absent cerebellar primary fissure and fastigial point. Hypoplasia of the vermis with apposition (not fusion) of cerebellar hemispheres and clefting of the superior cerebellar vermis. High riding 4th ventricle with deep pontomesencephalic junction.

Dorsal tegmental tract hyperintensity: Abnormal T2W/FLAIR hyperintensity in the midbrain and upper pons and dorsal tegmental tract—usually seen in cerebral palsy.

Creatine deficiency syndromes: Minimal hyperintensity noted in the dentate nucleus, medial portion of middle cerebellar peduncle and nucleus accumbens. MR spectroscopy of the brain shows markedly decreased creatine—**creatine deficiency syndromes** can be considered (guanidinoacetate methyltransferase (GAMT) deficiency, deficiency of L-arginine: Glycine amidinotransferase (AGAT), defect in a creatine transporter protein).

Parecho virus encephalitis: Evidence of restricted diffusion in bilateral frontoparietal and temporal subcortical white matter and corpus callosum. Punctate T1W hyperintensities seen in bilateral frontoparietal subcortical white matter. MR spectroscopy of the brain parenchyma shows normal spectral pattern. No abnormal metabolites seen. Features favour the possibility of Parecho virus/dengue/chikungunya encephalitis.

Craniosynostosis: Altered shape of the skull. Premature fusion of bilateral coronal suture leading to craniosynostosis with increased convolutional markings in the frontal bone (anterior plagiocephaly). Increased convolutional markings secondary to craniosynostosis. Sagittal, metopic, lambdoid and squamosal sutures are normal.

Microcephaly: Microcephaly with simplified gyral pattern in frontal and temporal region.

CT Cisternogram: **CSF leak:** 1.6 × 3.2 mm size bony defect in the left cribrifrom plate of ethmoid bone with CSF leak into the left nasal cavity.

Pontine tegmental cap dysplasia: Abnormal brainstem with hypoplastic pons and flattened ventral surface. Shrunken cerebellum and focal bulging of the pontine tegmentum projecting into the fourth ventricle—pontine tegmental cap dysplasia.

Hippocampal malrotation/vertical hippocampus/hippocampal inversion: Vertical orientation and medial

positioning of head and body of left hippocampus with round hippocampal head—hippocampal malrotation, above features show association with malformations of cortical development and temporal lobe epilepsy.

van der Knaap disease (megalencephalic leukoencephalopathy with subcortical cysts): Diffuse T2 W and FLAIR hyperintensities, not showing restricted diffusion in bilateral cerebral deep and subcortical white matter and external capsule. Multiple subcortical cysts in bilateral temporal lobes and high frontal regions.

Metachromatic leukodystrophy: Confluent bilateral symmetrical periventricular white matter hyperintensity with gliosis and mild ex-vacuo dilatation of both lateral ventricles. Bilateral symmetrical white matter hyperintensity also involves subcortical U fibers, external capsule, posterior limb of internal capsule, dorsal aspect of pons, middle cerebellar peduncles and bilateral cerebellar white matter.

Metachromatic leukodystrophy (early changes): Confluent periventricular white matter hyperintensities on T2 W/FLAIR sequence with no significant restricted diffusion. Both thalami and basal ganglia are preserved. No significant involvement of brainstem/subcortical U fibers. MRS shows mild reduction of NAA, rise in choline with no significant changes in other metabolite peaks.

Krabbe disease: Diffuse T2 W/Flair hyperintensity involving the periventricular, subcortical cerebral white matter, internal capsule, corpus callosum, dorsal brain stem, deep cerebellar white matter with high myelin edema. T2 hypointense signal noted in the bilateral thalamus which is hyperdense in CT. MR spectroscopy shows no significant abnormal metabolite—above features favour Krabbe disease. Suggested clinical/lab parameter correlation.

Hypomyelination: Severe hypomyelination/demyelination (myelination of only perirolandic white matter and central white matter).

Hypomyelination/leukodystrophy with diffuse thinning of corpus callosum: Possibilities include: 1. Hypomyelinating leukodystrophy–III, 2. hypomyelination and congenital cataract, 3. HSP60 chapernopathy, 4. Salla disease.

Canavan's disease: Bilateral cerebral subcortical and periventricular white matter, internal capsule, external capsule, globus pallidus, dentate nucleus, extreme capsule, cerebral peduncle, dorsal brainstem, deep cerebellar white matter shows T2W diffuse hyperintensities and hypointense in T1W FLAIR images. These changes appear bright in diffusion weighted images. Caudate and putamen appear normal. On spectroscopy, the lesion shows increased NAA compounds. Suggested urinary and plasma N-Aspartyl acetic acid/clinical correlation.

Alexander disease: T2W/FLAIR hyperintensity in the bilateral frontal, temporal white matter, external capsule, extreme capsule, basal ganglia, thalamus, brainstem and periventricular white matter garlands in the peritrigonal occipital region without diffusion restriction. On contrast administration mild enhancement noted along the ventricular lining and periventricular white matter in the frontal white matter. On spectroscopy, the lesion shows mildly decreased NAA peak.

Glutaric aciduria type I: Arachnoid cyst in bilateral anterior temporal regions (middle cranial fossa) with hypoplasia of the underlying temporal lobes—Bat wing shape with widened sylvian fissures. Hyperintensity involving caudate and lentiform nucleus, white matter, dorsal brain stem with diffusion restriction.

Anterior temporal pole cyst with calcification: Leukoencephalopathy with calcifications and cysts (LCC), TORCH (congenital CMV infection)/pseudo-TORCH (Aicardi–Goutières syndrome), cystic leukoencephalopathy without megalencephaly (RNASET 2-deficient cystic leukoencephalopathy), Cockaye syndrome (no basal ganglia calcification/cerebral atrophy).

Anterior temporal pole cyst without calcification: Abnormal T2W/FLAIR hyperintensities in bilateral peritrigonal and perilateral white matter, which does not show diffusion restriction. Bilateral anterior temporal pole cyst seen. No obvious evidence of basal ganglia and cerebral calcification seen. MR spectroscopy of the brain parenchyma shows normal spectral pattern. No significant reduction of NAA/elevation of choline. No abnormal metabolites seen. Possibilities include, TORCH (congenital CMV infection)/pseudo-TORCH (Aicardi–Goutières syndrome)—no evidence of calcification seen. Megaloencephalic leucoencephalopathy with subcortical cysts (no obvious megalencephaly). Leukoencephalopathy with calcifications and cyst—no evidence of calcification seen. Congenital muscular dystrophy.

Excessive T2W hyperintense changes noted in the bilateral deep white matter region. No evidence of PVL. No abnormal diffusion restriction. Diffuse and excessive high signal intensity (DEHSI)—**suggestive of normal finding in preterm infant. Mild elevated lactate peak—normal for age.**

Hypoglycemic (hypoxic) ischemic brain injury sequelae: Bilateral parieto-occipital gliosis with ex-vacuo dilatation of occipital horn of lateral ventricles.

***Hypoxic ischemic encephalopathy* (HIE):** Diffuse bilateral periventricular white matter hyperintensity

with white matter loss, ex-vacuo dilatation of both lateral ventricles with undulated margins. Mild diffuse thinning of the corpus callosum. Features favour the possibility of **periventricular leucomalacia** (hypoxic ischemic encephalopathy).

Abnormal T2W/FLAIR hyperintensity in the bilateral thalamus and posterior putamen—**profound hypoxic ischemic brain injury sequelae.**

Diffusion restricting T2 hypointense lesion seen throughout the cerebral parenchyma involving the deep grey matter and thalamus. Diffusion restricting lesion noted in the entire corpus callosum and internal capsule and extension along the corticospinal tract. Cerebellum and posterior brainstem relatively appear normal. MR spectroscopy of the brain parenchyma shows significantly elevated lactate peak seen throughout the brain parenchyma. **Features are suggestive of severe hypoxic ischemic encephalopathy changes.**

T2W hyperintensity in bilateral posterior lentiform nucleus, thalami, perirolandic frontoparietal regions, bilateral periventricular and deep white matter. Diffuse thinning of corpus callosum. Mild ex-vacuo dilatation of both lateral ventricles. MR spectroscopy shows reduction of NAA and presence of lactate in affected areas: **Features are suggestive of sequelae of perinatal hypoxic ischemic encephalopathy.**

Gliosis in left frontal, temporal, parietal and occipital cortex and white matter with ex-vacuo dilatation of left lateral ventricle, secondary hypoplasia of brainstem on left side and reduced volume of skull. Milder gliosis in right frontal and parietal cortex and white matter. Features are suggestive of sequelae of hypoxic ischemic encephalopathy (**multicystic encephalomalacia).**

Benign enlargement of subarachnoid space: Prominent bilateral frontotemporal arachnoid spaces—benign enlargement of subarachnoid spaces (normal variant). **Or** Widening of bifrontal subarachnoid spaces (craniocortical diameter >5 mm)—suggestive of benign physiologic enlargement of subarachnoid spaces (otherwise known as benign macrocephaly of infancy/external hydrocephalus/physiologic extraventricular obstructive hydrocephalus). It is a self-limited condition with spontaneous resolution of CSF spaces between 12–24 months.

Iron deposition (children): Abnormal T2W/FLAIR hyperintensity in the caudate nucleus, anterior aspect of putamen with bilateral globus pallidus. MR spectroscopy of the basal ganglia shows decreased NAA and mildly increased lactate. Bilateral optic atrophy. Moderate to severe atrophy of cerebellum and pons. Mild diffuse cerebral atrophy. Above feature favour metabolic/neurodegeneration. Possibilities include: Mitochondrial disorder/fatty acid associated neurodegeneration suggested clinical/lab parameter correlation.

Methylmalonic acidemia: Bilateral symmetric altered signal intensity appearing hypointense on T1W and hyperintense on T2W noted in globus pallidus without diffusion restriction/contrast enhancement. MR spectroscopy shows no significant abnormal metabolite. Possibilities include: Methylmalonic Acidemia, Kernicterus (less likely—no history of neonatal jaundice), Succinate semialdehyde dehydrogenase deficiency, L-2-hydroxy glutaric aciduria, isovaleric acidemia, neonatal hypoxia.

Bilateral putaminal T2W/FLAIR hyperintensity (putaminal necrosis): With diffusion hyperintensity and elevated lactate in MR spectroscopy. SWI shows no increased iron deposition. Possibilities include: Mitochondrial disease—including G14459A mutation, SUCLA-2 gene mutation. Pediatric autoimmune neuropsychiatric disorder associated with streptococcal infection (PANDAS). Propionic acidemia. Neuroacanthocytosis—additional features like cerebellar atrophy is absent. No evidence of thalamus, midbrain, pons involvement to suggest Wilson disease. No evidence of T1 hyperintensity in globus pallidus. Methanol intoxication/hypoxic ischemic changes—no supportive clinical history. Juvenile Huntington's disease—no evidence of caudate nucleus involvement. Molybdenum cofactor deficiency—no evidence of caudate nucleus, white matter involvement. Glutaric aciduria—no other white matter/bilateral sylvian arachnoid cyst **(refer to case 4.28).**

Bilateral globus pallidus T2W/FLAIR hyperintensity (adult): Bilateral globus pallidus T2W/FLAIR hyperintensity with diffusion restriction—possibilities include: 1. Poisoning (carbon monoxide), 2. Hypoglycemia/hypoxia, 3. Cocaine/heroine abuse. Neuro degeneration with brain iron accumulation less likely.

Caudate and putamen: Abnormal T2W/FLAIR/diffusion weighted hyperintensities in the caudate and putamen. MR spectroscopy shows elevated lactate—features favours Leigh's disease. Other possibilities include: Propionic academia, molybdenum cofactor deficiency, Wilson's disease, juvenile Huntington's disease, osmotic myelinolysis, hemolytic uremic syndrome, hypoxia—less likely, carbon monoxide poisoning **(refer to case 4.27).**

Prominent Virchow-Robin spaces: In bilateral cerebral hemisphere—possibilities include Lowe syndrome, mucopolysachridoses, hypomelanosis of Ito and velocardiofacial syndrome.

Bilateral thalamus without haemorrhage: Non-diffusion restricting long TR hyperintense lesions noted in the both

thalamus, bilateral outer putamen and caudate nucleus and bilateral patchy supratentorial subcortical and deep white matter and pons, midbrain central white matter. Possibilities include: Japanese encephalitis. Cerebral malaria/acute disseminated encephalomyelitis (rare).

Bilateral thalamus with haemorrhage: Bilateral thalamic hyperintensities with/without diffusion restriction and haemorrhages. Normal deep venous system noted. Possibilities to be considered: Flavi virus encephalitis like Japanese encephalitis, cerebral malaria, H3N2 influenza encephalitis with vasculitis (acute necrotizing encephalitis), herpes simplex encephalitis (no involvement of temporal lobe in this patient), dengue fever (haemorrhage less likely).

Reversible splenial lesion: Focal lesion in the splenium of corpus callosum which shows diffusion restriction without hemorrhage—reversible splenial lesion syndrome (RESLES) probably due to encephalitis/encephalopathy of varied etiology.

Tubulinopathy: Diffuse thinning of corpus callosum. Thin anterior commissure. Irregular prominent bilateral lateral ventricles—moderate ventriculomegaly. Enlarged caudate nucleus and fused striatum (absent diminutive internal capsule dividing the caudate from putamen): Dysmorphic basal ganglia—above features favour tubulinopathy (DYNC1H1 mutation).

Arnold-Chiari malformation Type 1: Small posterior fossa with cerebellar tonsilar herniation 8 mm below the foramen magnum line causing crowding of the foramen magnum. Evidence of syringomyelia in the cervical cord from C2 to C5 vertebrae level—Arnold-Chiari malformation Type 1. CSF flow shows pulsatile flow in the syringomyelia. No flow noted in the posterior subarachnoid space. Anterior cervical subarachnoid space and pre-pontine and pre-medullary cisterns show normal CSF flow.

Posterior fossa is smaller than normal. Herniation of the cerebellar tonsil below the foramen magnum up to the lower C2 level. 4th ventricle is elongated and slit-like with moderate triventricular hydrocephalus. There is medullary kink, beaking of tectum, large massa intermedia, scalloped posterior petrous pyramids and clivus, large funnel-shaped foramen magnum. Cerebellum wraps around the medulla. Low lying tentorium and torcula.

Pantothenate kinase associated neurodegeneration (Hallervorden-Spatz disease): Mild diffuse cerebral and cerebellar atrophy with increased signal intensity in the globus pallidus giving eye of the tiger appearance. Bilateral basal ganglia calcification seen. MR spectroscopy shows no abnormal metabolite seen. No evidence of altered signal intensity in the cerebellum. Possibilities include: Neurodegeneration with brain iron accumulation—pantothenate kinase associated neurodegeneration (Hallervorden-Spatz disease). Kearns-Sayre syndrome and other mitochondrial disorder NARP (neuropathy, ataxia and retitis pigmentosa).

Table 2.2: MR parkinsonism index-2 (MRPI 2)

	AP diameter	*Width*	*Volume (mm^3)*	*Interpretation*
Midbrain	14 mm			
Superior colliculus	3 mm			
Midbrain (midline) M			1.17	
Pons (midline) P			5.46	
Pons /midbrain area ratio P/M			5.46/1.17 = 4.67	
Superior cerebellar peduncle in coronal plane SCP		4.8 mm		
Middle cerebellar peduncle in sagittal plane MCP		11.1 mm		
MCP/SCP ratio			2.31	
MRI parkinsonism index = (P/M) × (MCP/SCP)		10.79		PSP >13.55
Third ventricular width (V3): Average width (from three measurements) of the 3rd ventricle on axial image at the level of anterior and posterior commissures	9.5 mm			
Frontal horn (FH) (maximal left to right frontal horn width on axial image in AC-PC plane)		37.7 mm		
MRPI 2.0 = MRPI × (V3/FH)		10.79 × 0.251 = 2.70		PSP-P ≥2.18 PSP-R ≥2.5 PD <2

Fukuyama's congenital muscular dystrophy: Cerebellar hypoplasia noted with tiny cortical cysts. Pontine hypoplasia with Z-shaped deformity. Bilateral lissencephaly with cortical polymicrogyria. Periventricular white matter long TR hyperintense lesion. Absent septum pellusidum noted. Bilateral lateral ventricles dilatation. Thickened tectal plate noted. Basal ganglia and thalamus appear normal.

Progressive supranuclear palsy: Diffuse cerebral and cerebellar atrophy. Disproportionate atrophy of midbrain. AP diameter of midbrain measures 11.9 mm, width of middle cerebellar peduncle 6.6 mm, width of superior cerebellar peduncle 1.7 mm on right side and 2.7 mm on left side. Area of midbrain measures 0.68 sq. cm and area of pons measures 3.4 sq cm.

Pituitary macroadenoma: Dumbbell-shaped well-defined enhancing mass lesion with cystic areas of size 36 (CC) × 26 (AP) × 28 (Trans) mm in the sella-suprasellar region compressing and elevating the distal intracranial optic nerves and optic chiasma. Expansion of sella noted. There is extension to right cavernous sinus, reaching up to the lateral venous compartment. Cavernous segment of right internal carotid artery is mildly pushed laterally without stenosis. Sphenoid sinus is well pneumatised. Normal pituitary gland and infundibulum are not made out separately. Tubercinerium, mamillary body, floor of the IIIrd ventricle are normal: Features are suggestive of pituitary macroadenoma. Post-sellar pneumatisation of the sphenoid sinus with prominent sellae bulge and plannum sphenoidale, dorsum sella pneumatisation. Sphenoid sinus show single midline septum anteriorly and multiple septae posteriorly. Inter-carotid distance—20 mm. Optic chiasma is seen over the pituitary and dorsal sellae—normal/post-fixed chiasma.

Viral encephalitis: Diffusion restricting long TR hyperintense lesion in the both putamen, right thalamus, right posterior parietal cortex and right inferior frontal gyri and right amygdala. Possible viral encephalitis.

Rasmussen's encephalitis: Diffuse gyral thickening and T2 hyperintensity noted involving the left cerebral hemisphere (left temporal, frontal and parietal regions). T2 hyperintensity noted in the left thalamus and caudate part of left basal ganglia and left substantia nigra. Focal T2 hyperintensity noted in the subcortical region with left frontal region. No evidence of restricted diffusion. Mass effect noted on the left cerebral sulci and left lateral ventricle with mild effacement. Mild midline shift to right of 2.0 mm noted. Mild leptomeningeal enhancement noted in the left cerebral hemisphere. The different diagnoses are Rasmussen's encephalitis/viral encephalitis/postictal change associated with status epilepticus.

Acoustic schwannoma: Evidence of a large heterointense extra-axial avidly enhancing mass lesion measuring 5.2 (AP) × 3.6 (TR) × 4.1 (CC) cm noted in the left CP angle region. There is extension into the left internal auditory meatus with intrameatal component measuring 5.1 × 5 mm. Distal 3.4 mm of the internal auditory canal appear normal. Fundus of internal auditory canal and cochlear aperture appear normal. Areas of multiple microhaemorrhages within the mass. The lesion is highly vascular with multiple feeders from the left vertebral artery which is hypoplastic. Left VII-VIII nerve complex in the CP angle cistern and proximal IAM are not visualised separately from the mass. There is compression of the pons, lower midbrain, left middle cerebellar peduncle and adjacent cerebellar hemisphere without intra-axial edema. Effacement of aqueduct and fourth ventricle with early obstructive hydrocephalus (dilatation of third and both lateral ventricles). Increased intracranial tension with bilateral prominent perioptic CSF sheath and tortuous optic nerves, flattening of posterior sclera and partial empty sella. Evidence of tonsillar herniation of 12 mm below the foramen magnum.

Eosinophilic granuloma: Evidence of well-defined lytic lesion in the right parietal bone measuring 37 mm (AP) × 15 mm (TR) × 53 mm (CC) with extra-axial and subgaleal soft tissue components. There is destruction of both inner and outer tables. The lesion intracranially abuts the dura which is thickened and demonstrates increased enhancement. No other calvarial lesions are seen. No definite intra-axial abnormality. The infundibulum and cavernous sinuses have a normal appearance.

Tuberous sclerosis: Multiple subependymal nodules along the lateral ventricles predominantly along the caudothalamic groove, cortical tubers in the left postcentral gyrus and right superior frontal gyrus. No evidence of subependymal giant cell astrocytoma/ocular lesions/white matter changes/vascular lesions.

PRES (posterior reversible encephalopathy syndrome): Bilateral frontoparietal, parietal, occipital and left cerebellar subcortical and cortical T2W FLAIR hyperintensity with/without diffusion restriction.

Colloid cyst: Well-defined T1 hyperintense lesion measuring 12.5 mm (AP) × 10.5 mm (TR) × 10 mm (CC) in anterior third ventricle at the foramen of Monro, more towards left side causing mild asymmetric prominence of left lateral ventricle.

Normal pressure hydrocephalus: Age related diffuse cerebral atrophy with prominent cortical sulci and both

lateral ventricles. There is mild disproportionate dilatation of both lateral ventricles compared to sulcal prominence with minimal periventricular hyperintensity along the frontal and occipital horns of both lateral ventricles. Accentuated flow void noted across the aqueduct and adjacent posterior third ventricular region and proximal 4th ventricle. MR spectroscopy of the ventricle shows lactate peak—finding favouring the possibility of normal pressure hydrocephalus over other dementia. No dilatation of IIIrd and IVth ventricle. No ballooning of optic and infundibular recess of third ventricle. No disproportionate dilatation of sylvian fissure.

Alzheimer's disease: Cerebral sulcal spaces, basal cisterns, cerebellar foliae and ventricular system appear prominent. Suggestive of moderate diffuse parenchymal atrophy. Global cortical atrophy scale: score 2. The hippocampus volumes measures 1.30 cm^3 on right side and 1.29 cm^3 on left side—decreased. MTA-scale for Medial Temporal lobe Atrophy: score 3. The interuncal distance at the level of anterior commissure is 31 mm—more than 30 mm is considered as abnormal.

Wilson disease: Bilateral symmetrical FLAIR/T2 hyperintensities in the putamen, thalami, pallidum and tegmentum of midbrain and periaqueductal grey matter. T1 hyperintensity noted in the globius pallidus. Midbrain lesion shows characteristic 'face of the giant panda'—Wilson disease.

Tolosa-Hunt syndrome: Ill-defined hypointense lesion in the right orbital apex and right cavernous sinus, which shows homogeneous enhancement. The lesion measures 20 (AP) × 10 (CC) × 6 (TR) mm. No obvious evidence of dural tail.

Aberrant course of the right anterior inferior cerebellar artery indenting on the right facial nerve at the root entry zone with mild deviation of 7th nerve course. No evidence of **neurovascular conflict at the nerve root entry zone** of left trigeminal nerve.

Mesial temporal sclerosis: Body of the right hippocampus appears small with loss of internal architecture (striations) and loss of hippocampal digitation with increased signal intensity and dilated temporal horn of right lateral ventricle. Right fornix and mamillary body atrophy—right mesial temporal sclerosis. Right hippocampus volume ~1.89 cc. Left hippocampus volume ~2.09 cc.

Acute intracerebral hemorrhage: [60 mm (AP) × 39 mm (TR) × 38 mm (CC)] in the left para sagittal frontal region inter-hemispheric fissure and adjacent sulcal spaces. There is extension to all ventricle more in the left lateral ventricle. Significant mass effect on left lateral ventricle with midline shift to the right (14 mm) anterior body of the corpus callosum is displaced downwards. Mildly prominent right lateral ventricle. Haemorrhage also noted in the sylvian fissure and basal cisterns.

Extra dural haemorrhage: Maximum thickness measuring 18 mm in the left lateral convexity along the parietotemporal region, causing mild mass effect with local sulcal effacement and midline shift of 4 mm to the right. No evidence of hydrocephalus. Basal cisterns appears normal. Linear undisplaced Fracture noted in the squamous part of left temporal bone.

Comment about space of the haemorrhage, size, intraventricular extension, obstructive hydrocephalus, edema, midline shift, herniation, any area of infarction, vessel injury, old gliosis, if any.

Small vessel ischemic changes: Multiple focal discrete and confluence T2W/FLAIR hyperintensities in the periventricular and central white matter which does not show diffusion restriction—small vessel ischemic changes. Fazekas scale for WM lesions—score 2.

Acute disseminated encephalomyelitis (ADEM): Altered signal intensity appearing hyperintense on T2 and FLAIR, not showing restriction of diffusion in bilateral basal ganglia, thalamus, brain stem and deep cerebellar white matter. No abnormal enhancement on contrast administration—features favours acute disseminated encephalomyelitis (ADEM).

Acute infarct in the left middle frontal gyrus, inferior frontal gyrus, pre central gyrus, postcentral gyrus, left centrum semiovale, left superior, inferior parietal gyrus, insula, head of left caudate nucleus, left temporoparietal region. Minimal haemorrhagic transformation in the left parietal region. No evidence of significant mass effect**.**

Demyelination: Abnormal T2W/FLAIR hyperintensity in the right superior cerebellar subcortical white matter which does not show diffusion restriction/contrast enhancement—demyelination.

Vasculitis/chronic demyelination plaque: Multiple small T2W and FLAIR hyperintensities, not showing restricted diffusion seen scattered in bilateral fronto parietal subcortical white matter and parieto-occipital periventricular white matter. Normal callososeptal interface. No evidence of Dawson's fingers. No evidence of haemorrhage. No optic nerve demyelination.

Multiple sclerosis: Multiple nondiffusion restricting nonenhancing linear, ovoid and globular long TR hypointensity lesions involving the callososeptal interface, perilateral ventricular regions, bilateral frontal, temporal, parietal, subcortical regions and right internal capsule, left cerebellar white matter, superior cerebellar

Table 2.3: Neuroimaging radiological interpretation system (NIRIS)		
Category	*Definition*	*Patient management/actions*
NIRIS 0	No abnormal finding	Discharge from the ED
NIRIS 1	Fracture ±	Follow-up neuroimaging and/or admit for observation.
	Extra-axial hematoma, intraparenchymal hematoma/contusion <0.5 mL±	
	Subarachnoid haemorrhage.	
NIRIS 2	Extra-axial hematoma, intraparenchymal hematoma/contusion >0.5 mL±	Admit to a more advanced care unit.
	Diffusion axonal injury ±	
	Intraventricular haemorrhage ±	
	Mild hydrocephalus ±	
	Midline shift 0–5 mm	
NIRIS 3	Extra-axial hematoma, intraparenchymal hematoma/contusion >5 mL±	Consider neurosurgical procedure (ventricular drain, burr hole, craniotomy/ craniectomy, surgical drainage/evacuation of hematoma)
	Moderate hydrocephalus ±	
	Midline shift >5 mm ±	
	Focal herniation	
NIRIS 4	Extra-axial hematoma, intraparenchymal hematoma/contusion >25 mL±	High risk traumatic brain injury-related death
	Severe hydrocephalus ±	
	Diffuse herniation/duret haemorrhage	

vermis, pons—right middle cerebellar peduncle junction. Multiple patchy focal lesions in the entire spinal cord involving lateral, central and posterior columns at variable levels. Features favour multiple sclerosis—suggested clinical/CSF of analysis correlation. **Restricted diffusion noted in bilateral central part of the centrum semiovale more on the right side. Patchy peripheral diffusion restriction in the left centrum semiovale lesions (Table 2.4).**

Bilateral T1 hyperintensity in globus pallidus: Bilateral T1 hyperintensity in globus pallidus—possible basal ganglia calcification/hepatocellular—renal dysfunction, manganese deposition (in patients with chronic renal disease on hemodialysis).

Idiopathic intracranial hypertension: Partial empty sella seen. Prominent bilateral perioptic sheath fluid with posterior scleral flattening. Moderate luminal narrowing in bilateral distal transverse sinuses.

Hydrocephalus with post-op status: Post-operative burr hole defect in the frontal and right parietal bone. Moderate dilatation of third and both lateral ventricles with collapsed fourth ventricle–obstructive hydrocephalus. VP shunt tube *in situ* one through the right parieto-occipital and other through the right

Table 2.4: Multiple sclerosis sample reporting						
Region	*Location*	*No of lesions*	*Size*	*Diffusion restriction (+/–)*	*Contrast enhancement (+/–) type of enhancement*	*Comparison with previous MRI New lesions/T1 Black Holes*
Periventricular and callososeptal interface						
Juxtacortical/ cortical						
Infratentorial						
Spinal cord						
Lobar-vloume						Volume loss
Corpus callosum-volume						
Other findings (optic nerve)						

frontal lobe with tip in the body of right lateral ventricle. Bifronto-temporal encephalomalacic gliosis. Paucity of subcortical white matter in the frontotemporal region. Right parieto-occipital loculated fluid collection measuring 46 × 18 mm along the shunt tract near the reservoir.

Head and Neck

CA tongue: 19 (AP) × 10 (TR) × 18 (CC) mm size ill-defined mass lesion in the right anterolateral border of the anterior third of tongue with minimal infiltration of the intrinsic muscles. Mylohyoid, hyoglossus and genioglossus, stylopharyngeus appears normal. No evidence of extension into the mandible/retromolar trigone/lingual septum/floor of mouth/midline/posterior third of tongue—features suggestive of carcinoma tongue. **(T2M0N0)**.

Ca tongue reporting checklist: **Laterality**: **Right or left, Size: ….× …. × … cm.** Depth of invasion: (Previous depth of invasion:)

T Stage: To comment on whether the tumor crosses the midline or abuts lingual raphe, involvement of extrinsic muscles like genioglossus, hyoglossus, geniohyoid, involvement of lingual neurovascular bundle (grade: 0/I/II/III) and uni/bilateral involvement.

To comment on whether the tumor extends to posterior one-third of the tongue, RMT, tonsillo-lingual sulcus and tonsil extension of tumor to sublingual space/submandibular space, infiltration of mylohyoid and involvement of floor of mouth, comment on extension to masticator space, infratemporal fossa (ITF), high infratemporal fossa. Inferior extent: Look up to vallecular/epiglottis/PFS, also comment on involvement of hyoid, if not involved—comment on distance from hyoid bone. Look for any bony erosion—mandibular involvement, cortical breach, marrow signal abnormality.

N: Presence of nodal disease: Metastatic/benign (reactive)/indeterminate, if indeterminate/suspicious: Need for additional imaging, laterality: Ipsilateral/contralateral/bilateral levels: Level: IA and IB/II, III, IV, V, VI and retropharyngeal. Size of the largest node: Right side: ….. mm and level, Left side: ….. mm and level, presence of necrosis if any, perinodal extension/extracapsular spread: Absent/present, vascular involvement by the nodes: IJV: involved/compressed; CCA, ICA, ECA abutment: Absent/ Present, If present angle of contact for CCA and ICA: <90, 90–179, 180–269; >270. Strap muscles involvement, Prevertebral fascia invasion. M: Lung, hepatic, adrenal and skeletal metastasis.

Juvenile nasopharyngeal angiofibroma (JNA): Large well-defined lobulated highly vascular enhancing mass lesion measuring about 5 × 4.1 × 4.4 cm (AP × TR × CC) noted in the right spheno palatine foramen, posterior nasal cavity, posterior ethmoidal sinus and right sphenoid sinus. Lesion is highly vascular with multiple flow voids within. Main arterial supply of the mass is seen from the hypertrophied right internal maxillary artery. There is widening of the right pterygopalatine fossa and extension of the soft tissue into the infratemporal fossa. Erosion of the medial and lateral pterygoid plate on right side. There is involvement of right foramen rotundum and vidian canal. There is erosion of the anterior wall of the foramen ovale. Erosion of floor of right middle cranial fossa (greater wing of sphenoid) without intracranial extension. No involvement of the ICA. There is extension into the inferior aspect of the orbital apex on right side through the widened inferior orbital fissure. No involvement of the extraocular muscles/optic nerve. Nasal septum is pushed to left. The mass is extending into the nasopharynx—features are consistent with juvenile nasopharyngeal angiofibroma–stage IIc: (staging system proposed by Sessions, modified by Radkowski).

Multinodular goiter: Thyroid appears diffusely enlarged showing heterogeneous enhancement, right lobe measures ……left lobe measures …….with mild mass effect over trachea, no retrosternal extension of thyroid lobes noted. No significant cervical lymph nodes noted. TIRADS-II.

Post-cricoid carcinoma: An ill-defined soft tissue dense lesion measuring 2 × 1 cm noted in post-cricoid region at the level of C5–C6, the lesion does not extend to bilateral pyriform fossa, the lesion appears to erode cricoid and cricoarytenoid joint, arytenoid appears sclerosed, the lesion infiltrates posterior aspect of larynx with marked narrowing of airways, lesion appears to involve the anterior wall of proximal cervical esophagus for a length of 1 cm causing luminal narrowing of esophagus, recurrent laryngeal nerve appears normal. The parapharyngeal fat planes are infiltrated, no cervical lymphadenopathy.

Glottic growth: Ill-defined soft tissue density lesion measuring about 19 × 11 mm in the right vocal cord. No subglottic extension of the lesion. Minimal infiltration of the right supraglottic fat. No obvious extra laryngeal extension. There is sclerosis of the right arytenoid cartilage - ? Infiltrated.

Supraglottic growth: Soft tissue density lesion measuring about 2.2 × 1.5 cm in the right side of the larynx involving

Table 2.5: Lymphoma reporting tool			
No.	Size (mm) (long-axis diameter) × (short-axis diameter)	FDG uptake (SUVmax)	Location (lymph node location/location within the affected organ)
1.			
2.			

the supraglottic and glottic space. No subglottic extension. There is mild sclerosis of the right aretynoid cartilage and thickening of the right aryepiglottic fold. No involvement of the left vocal cord. A few small subcentimetric lymph nodes in bilateral cervical region.

Lymphoma: Multiple lobulated enlarged lymph nodes noted in the bilateral preauricular, cervical infra-clavicular, hilar, mediastinal lymph nodes. There is no evidence of splenomegaly. With PET scan avid uptake **(SUV 3.0)** noted in the lymph nodes and in the right iliac bone (recommended site for biopsy). No evidence of other lesions in the stomach, liver, lung/pleura, kidney, CNS and skin (Table 2.5).

Vocal cord palsy: Paramedian position of the right vocal cord. Prominent right pyriform sinus with thickened aryepiglottic fold—right vocal cord palsy. No obvious mass lesion in the neck/visualised mediastinum.

***Post-radiation status of larynx*:** Partially abducted left vocal cord with mild effacement of left pyriform fossa and vallecula with thickened (17 × 9 × 17 mm) false cord, supraglottic and glottic region extending to involving the posterior aspect of the pharynx with narrowing of laryngeal inlet. Diffuse non-enhancing edema of the pyriform fossa, Aryepiglottic folds and post-cricoid pharynx. Mild irregularity in the right lateral border of tongue-radiation induced changes

***Vagal schwannoma*:** Evidence of a large extraaxial intra and extracranial dumb bell-shaped heterointense enhancing mass lesion with cystic areas and hemorrhage noted in left jugular foramen, cerebello medullary cistern and CP angle. Extracranially, lesion extends to the upper carotid space. Intracranial component measures 3.9 (AP) × 2.8 (trans) × 3.4 (CC) cm, the component in jugular foramen measures 2 × 1.2 cm, extracranial component in upper carotid space measures 4.5 (trans) × 2.69 (AP) × 3.9 (CC) cm. The left internal carotid artery is displaced anteromedially by the mass and internal jugular vein is compressed posterolaterally and not visualised in the region the mass in carotid space. Medulla and left middle cerebellar peduncle are indented by the mass. No intra-axial edema. No obstructive hydrocephalus. Smooth widening of left jugular foramen. No extension to internal auditory meatus. No extension to hypoglossal canal. Left vocal cord palsy changes seen.

Branchial fistula: 6 mm length linear fistulous tract extending from skin in the left paramedian lower neck extending up to the sternocleidomastoid muscle. No evidence of extension into the left lobe of thyroid/pyriform fossa.

***Lower cranial nerve/jugular schwannoma*:** Large intra- and extra-cranial dumb bell-shaped heterointense enhancing mass lesion with cystic areas and hemorrhage in left upper carotid space, jugular foramen, cerebello medullary cistern and CP angle.

Suppurative lymphadenitis: Conglomerate enlarged left upper and mid cervical lymph nodes and upper posterior triangle lymph nodes with areas of necrosis. No evidence of vertebral, bone or deep neck space involvement.

Carotid body tumor: Well-defined intensely enhancing highly vascular soft tissue density mass lesion measuring 3.6 × 3.7 × 5.2 cm (AP × TR × CC) in the left carotid space at CCA bifurcation level with splaying of the internal carotid and external carotid arteries. The lesion shows salt and pepper appearance in T1/T2 images. Mass is extending superomedially to lower para pharyngeal space. Left internal jugular vein is compressed and pushed laterally without thrombus. There is no encasement of ICA and ECA. No e/o luminal narrowing/thrombus formation. Multiple arterial feeders seen arising directly from proximal external carotid artery, ascending pharyngeal artery and occipital artery. No supply from the ICA.

Chronic otomastoiditis without cholestatoma: Soft tissue noted filling the right middle ear cavity, right external auditory canal, mastoid antrum and mastoid air cells. Soft tissue noted in right facial nerve recess and sinus

Table 2.6: Cochlear implant workup on HRCT	
1	Mastoid cellularity—non-pneumatised / hypo/ normal
2	Middle ear cavity—ossicles integrity and round window
3	Position of genu and descending mastoid segment of facial nerve—any aberrant course
4	Jugular bulb (if high riding)
5	Bony labyrinth structure with special focus on cochlea size, turns, interscalar septum, modiolus, cochlear aperture, cochlear duct length
6	Internal auditory canal
7	Cochlear and vestibular aqueduct

Table 2.7: Cochlear implant workup in MRI	
Signal of fluid in labyrinth	Hypointense in case of labyrinthitis: Balkany classification of labyrinthitis
Inner ear structures with special focus on cochlea. Scalar anatomy, osseous spiral lamina, outlines of modiolus, cochlear aperture size	Inner ear anomalies
Internal auditory canal with 7–8th nerve complex evaluation	Absence of 8th nerve/ undivided 8th nerve, hyoplastic 8th nerve
Lateral bulbopontine and cerebellopontine angle	For evaluation of any mass lesion
Brain stem (fascicular segment of 8th nerve, cochlear nerve nuclei)	For evaluation of any mass lesion in brain stem
Fascicular segment of 8th nerve-course along lateral aspect of pons, cochlear nuclei-hypointense structure along dorsolateral aspect of inferior cerebellar peduncle	
Auditory pathway and brain survey	Any lesion/ hypoxic changes, demyelination in superior olivary nucleus, lateral leminiscus, medial geniculate body, superior temporal gyrus, Heschl gyrus

Table 2.8: Cochlear implantation work up (measurements)		
Cochlea	2.5–2.75 turns, 29.5–32 mm in length, 8–10 mm height	Cochlear anomalies—aplasia/ hypoplasia
Cochlear aperture	Length: 0.8–1.2 mm, diameter: 1.8–2.5 mm	Diameter <1.4 mm—indicative of cochlear nerve deficiency
Cochlear duct length	CDL = (4.16 * A) – 3.98, A: Largest distance from round window to the lateral wall of cochlea	Electrode array of optimal length to be chosen
IAC diameter		<2.5 mm—absent/ hypoplastic 8th nerve
Cochlear aqueduct		>3 mm—dilated, risk of transmission of ear infection to meninges
Vestibular aqueduct	At midpoint between crus and external aperture	>1.5 mm/calibre is more than half of posterior semicircular canal

Table 2.9: CT reporting and staging system of acquired cholesteatoma		
Tympanic cavity involvement (T)	*Mastoid cavity involvement (M)*	*Complications (C)*
T1: Attic cholesteatoma	M0: No mastoid cavity involvement	C0: Uncomplicated cholesteatoma
T2: Tympanic cholesteatoma	M1: Cholesteatoma extending into mastoid antrum	C1: Temporal complications
T3: Attico-tympanic cholesteatoma	M2: Cholesteatoma extending to mastoid cavity and cells	C2: Cranial and intracranial complications
T4: Holotympanic cholesteatoma		

tympani. No evidence of erosion of scutum/widening of Prussak's space, erosion of malleus and incus on both sides. No erosion of tegmen tympani, lateral semicircular canal or facial nerve canal/sinus plate/inner ear.

Chronic left otomastoiditis with cholesteatoma: Soft tissue noted filling the left middle ear cavity, mastoid antrum and mastoid air cells. Soft tissue noted in left facial nerve recess and sinus tympani. There is erosion of scutum, malleus, incus with widening of Prussak's space. Middle ear soft tissue component measures 12 × 9 mm with extension of soft tissue into the left external auditory canal. No erosion of tegmen tympani, lateral semicircular canal or facial nerve canal/sinus plate/inner ear. No evidence of widening/block of left eustachian tube. Partial sclerosis of left mastoid air cells. No obvious evidence of jugular vein, left sigmoid sinus thrombosis. No obvious evidence of intracranial abscess/ meningeal component. Nonecho planar diffusion shows restricted diffusion.

Temporomandibular joint dislocation: Abnormal anterior displacement of articular disc of left temporomandibular joint with inter position of the posterior band between the condyle and the eminence. It remains anteriorly displaced at all times during jaw opening—**anterior displacement without reduction.**

Abnormal anterior displacement of articular disc of right temporomandibular joint with interposition of the posterior band between the condyle and the eminence. It reduces during jaw opening. **Anterior displacement with reduction.**

Right temporomandibular joint articular disc posterior band is located at 12 o'clock position related to condyle in closed mouth and 'bowtie' appearance in between articular eminence and condylar head in open mouth—**normal**.

***Obstructive sleep apnoea in dynamic sleep MRI*:** Severe dynamic airway narrowing with complete obliteration of the lumen at the naso-oropharyngeal junction due to apposition of the posterior margin of the soft plate and the posterior pharyngeal wall during sleep.

Eagle's syndrome: Mildly elongated bilateral styloid process (36 mm in length).

Sinonasalpolyposis: Extensive polypoidal mucosal thickening completely filling the left maxillary, ethmoid, frontal and sphenoid sinuses and nasal cavity. OMU complex is completely blocked on left side. There is extension of the polyp into the nasopharynx through the choana. There is mild sclerosis of the walls of the sinuses—secondary to chronic inflammation. Small mucosal polyp in the right maxillary sinus. Deviation of the nasal septum to right, mild mucosal thickening in right frontal, ethmoid and sphenoidal sinuses. DD: Allergic fungal sinusitis.

Sinusitis: Minimal mucosal thickening in left maxillary sinus. Kero's type I skull base. Bilateral partial concha bullosa. Hypertrophy of both inferior turbinate.

Os terminale–Os odontoideum: Hypoplastic dens with wide gap between the os terminale–os odontoideum complex, which is seen near the basi occiput (dystrophic). Enlarged anterior arch of atlas. Evidence of atlanto-axial instability along with prominent retro os odontoideum soft tissue causing myelomalacic changes in the cervical cord at C1–C2 vertebrae level.

Anteriorly displaced C2 body and posteriorly pointing os odontoideum compromising the spinal canal. T2 W hyperintense/T1 W isointense lesions noted in upper cervical cord at C2 level measuring about 8 mm in length—suggestive of cord edema/contusion.

Os odontoideum: A well-corticated round area of ossification measuring 7 × 7.5 mm articulating with the anterior arch of atlas. Os odontoideum is seen subluxated to right side in relation to body of the C2 with left lateral angulation at C1–C2 level. No canal compromise. Clivus is short. Evidence of mild basilar invagination without canal compromise. Tip of the dens is just below the McRae line, 5 mm above the Chamberlain and 8 mm above the McGregor line.

Evidence of mild **basilar invagination** with tip of the dens minimally indenting the cervico medullary junction without intra-axial edema. Tip of the dens is 2 mm below the McRae line, 4 mm above the Chamberlain and 8 mm above the McGregor line.

***Persistent hyperplastic vitreous*:** Small right eyeball with retrolental soft tissue lesion and hypoplastic vitreous. Hyaloid remnant of cloquets canal visualised. No evidence of calcifications—features favour persistent hypoplastic primary vitreous. Evidence of vitreous hemorrhage with thin subchoroidal collection.

Spine

Scoliosis: Dorsolumbar scoliosis with convexity to the left. Apical vertebrae is L1. Upper end vertebra is D8. Lower end vertebra is L3. Cobb's angle measures 22°. No structural abnormality in the vertebrae**.**

Sacral agenesis: High bulbous ending cord at upper border of D12 vertebrae level (distal spinal cord hypoplasia). Sacral agenesis involving the lower sacral vertebrae–group I caudal regression syndrome.

Sacroiliitis: Bilateral symmetric sacroiliitis with subchondral erosions, marrow oedema and sclerosis. No evidence of ankylosis—features favour seronegative spondyloarthropathy. Suggested clinical/lab parameter correlation.

Sacroiliitis with ankylosis: Bilateral symmetric sacroiliitis with subchondral erosions, marrow oedema and sclerosis, partial ankylosis in the both sacroiliac joint—features favour seronegative spondyloarthropathy—possible ankylosing spondylitis. Suggested clinical/lab parameter correlation.

Osteitis condensans ilii: Sclerosis of iliac side of bilateral sacroiliac joint without edema/erosion–osteitis condensans ilii.

Dorsal dermal sinus with cutaneous opening in the S1–S2 region coursing anteroinferiorly and extends into the spinal canal at upper part of S1 vertebrae level. Intradural sinus ascends superiorly within the spinal canal through an heterogeneous peripherally enhancing ill-defined intraspinal canal lesion from S1 to L2 levels and attached posterior to the conus at L2 vertebrae level. The intraspinal lesion measures 45 (CC) × 4 (TR) × 6.4 (AP) mm. There is areas of well-defined lobulated cyst (12 × 6 mm) at L5 vertebrae level within the lesion and shows areas of diffusion restriction—suggestive of epidermoid. The lesion does not show peripheral enhancement and leptomeningeal enhancement. 24 × 3 mm size peripherally enhancing infected subcutaneous dorsal dermal sinus at L5, S1 level. Conus ends at L2 vertebrae level.

Myelomeningocele: Evidence of large lumbosacral myelomeningocele with herniation of meninges and

the neural elements through the wide posterior osseous spinal defect at L3 to lower sacrum. Myelomeningocele sac measures 9.5 × 9 cm. Neural elements are seen dangling from the myelomeningocele sac. Mild prominence of the central canal of the cord. There is tethering of the cord. No filum terminale lipoma. No sacral hypoplasia. No vertebral segmentation anomalies. No evidence of cerebellar tonsillar herniation.

Meningocele/terminal myelocystocele: Evidence of large lumbosacral meningocele with herniation of meninges through the wide posterior osseous spinal defect at L3 to lower sacrum. Meningocele sac measures 9.5 × 9 cm. Mild prominence of the central canal of the cord. There is tethering of the cord. No filum terminale lipoma. No sacral hypoplasia. No vertebral segmentation anomalies. Terminal end of cord noted at L3 vertebral level. The sac is covered with skin. Cord ends above the L3 vertebra.

Ventral cord herniation: Anterior herniation of the cord at D5 vertebral level with prominent subarachnoid space in the posterior aspect—suggestive of idiopathic ventral cord herniation.

Diastematomyelia: Evidence of diastematomyelia from D9 to upper border of L3 vertebra and tethering of the cord, with conus ending at L3-L4 level. Bony spur seen at D12–L1 vertebrae level measuring 79 mm (AP) × 6.4 mm (width) × 7.8 mm (CC). Two hemicords unite inferiorly at L3 vertebrae level. Focal syringomyelia of the left hemicord at D12 to L2 vertebrae level.

Lipomyelocele: 41 mm (CC) × 20 mm (TRANS) × 13 mm (AP) size transitional lipoma in the dorsal aspect of the conus extending from L1 to S1 vertebrae level with tethering of cord. Conus ends at lower border of L5 vertebrae level. Transitional lipoma continuous up to the subcutaneous fat through the posterior spinal defect at L4, L5 vertebrae level. Focal syringomyelia (16 × 7 mm) at D12, L1 vertebrae level. No evidence of cerebellar tonsilar herniation.

LDM (limited dorsal myeloschisis): Linear fibro neural tract extending from the dorsal aspect of the conus at upper L2 level and connected to the skin through the small posterior spinal defect at L3–L4 level. There is herniation of the CSF through the same posterior spinal defect with CSF sac surrounding the fibro neural tract in the subcutaneous plane. CSF sac measuring about 20 × 10 mm (saccular type).

Mucopolysaccharidosis: J-shaped sella seen. Multiple prominent VR space noted along the corpus callosum and white matter. Acute kyphosis (Gibbus) at the thoracolumbar junction due to wedging of L1 vertebral body. Platyspondyly. Prominent—postdens soft tissue thickening and posterior ligamentous thickening (thickened dural ring at the foramen magnum) causing narrowing of postdens space and cervical cord compression. No evidence of altered signal intensity in the cervical cord.

Spinal dural arteriovenous fistula (Type I spinal AVM): Prominent dilated serpiginous vessels in the posterior spinal canal of thoracic region, more prominent in the lower dorsal region from D8 to D12 and in the anterior spinal canal from D5 to D11 level. MR angiography shows feeding vessel from D9 to D10 left radicular artery. Cord hyperintensity noted from D8 vertebral level to conus with mild expansion of cord and heterogeneous enhancement—features favour cord edema/ischemia (venous hypertensive congestive myelopathy).

Grade I spondylolisthesis of L5 over S1 with bilateral spondylolysis of L5 pars interarticularis.

Spondylolysis: Defect in the bilateral pars articularis of the L5 vertebra causing grade 1 anterolisthesis of L5 over the S1 vertebra. Sclerosis and degenerative changes noted in the posterior elements of the L5 vertebra. Associated with pseudodisc bulge and mild bilateral neural foramen narrowing.

Hirayama disease: Moderate localized lower cervical cord atrophy at C3 to C6–C7 with cord hyperintensity predominantly involving central, anterior gray matter (C3 on right hemicord and C4 on the left hemicord to C6–C7). Cervical abnormal curvature and symmetric cord flattening noted. Loss of attachment of dorsal dural sac, anterior displacement of the dorsal dura on flexion (from C3 to D1) and a prominent posterior epidural space with a prominent epidural veins which appears as flow void in space sequence. On contrast administration contrast enhancement noted in the posterior epidural component.

Burst fracture: Burst fracture noted involving body of D9 vertebra with fracture fragment causing mild narrowing of spinal canal and mild injury to the posterior ligamentous complex (PLC). No evidence of any injury to nerve roots and spinal cord compression—**TLICS-4** (Table 2.10).

Spine trauma: Jefferson fracture: Linear fracture noted on the left and right sides of the anterior and posterior arches of the atlas. Minimal displacement of the posterior arch noted. Atlantodental interval is increased measuring 6 mm.

Locked facet: Dislocation of the left facet joint of C6 behind C7 noted resulting in a unilateral locked facet causing grade 2 anterolisthesis of C6 on C7. Associated rotatory subluxation without dislocation of the right facet joint. No associated fracture is seen on the right side.

Table 2.10: Thoracolumbar injury classification and severity score (TLICS) calculation

Injury morphology	*Point value*	*PLC status*	*Point value*	*Neurological status*	*Point value*
Compression	1	Intact	0	Intact	0
Burst	2	Injury suspected or intermediate	2	Nerve root involvement	2
Translation or rotation	3	Injured	3	Spinal cord or conus medullaris injury Incomplete Complete	 2 2
Distraction	4			Cauda equina syndrome	3

Alignment and articulation of the rest of the cervical vertebra appears normal.

Spinal TB: Altered T2 signal intensity noted involving the body of D6 to D8 vertebrae with collapse and anterior wedging of the D7 vertebra. Loss of intervertebral disc height noted. There is a prevertebral, paravertebral and epidural collection extending from the D5 to L3 vertebral levels. Epidural collection causes narrowing of the spinal canal with compression of the spinal cord at the D6 level. Anterior subligamentous spread noted. Left psoas appears enlarged with a T2 hyperintense collection noted from L2 to L4 levels. On contrast, the collection shows thick, peripheral enhancement. Visualized lung fields show multiple miliary nodules.

Infective spondylodiscitis: D5–D6 disc signal alteration adjacent vertebral marrow signal changes with epidural soft tissue collection causing mild cord compression without significant cord signal abnormality. Lytic destruction of D11 and D12 vertebral bodies with partial collapse of D12. There is involvement of the D11–D12 disc space. Anterior epidural soft tissue thickening at D11–D12 level indenting the cord and extending to the right D11–D12 neural foramina. There is adjacent pre and paravertebral abscess and large right psoas abscess (7 × 6.6 × 20 cm). Right kidney is pushed laterally by the psoas abscess.There is involvement of the adjacent D10 and L1 vertebral bodies. There is marrow edema of pedicles of D11 and D12. Mild scoliosis of lower dorsal spine with convexity to left.

Transverse myelitis: Long segment T2 hyperintensity noted in the cord extending from the C5 to D2 levels. Predominantly central cord involvement noted in the T2 axial images. Cord appears mildly expanded with associated oedema. The rest of the cord appears normal. There is no enhancement on contrast. There is no associated syrinx.

Disc bulge: Disc dessication with diffuse disc bulge noted at the L4–L5 level causing spinal canal narrowing with compression of the ventral thecal sac without causing any significant compression of the nerve roots.

Disc protrusion: Disc dessication with focal right paracentral disc protrusion noted at the L4–L5 level along with facetal arthrosis causing spinal canal narrowing and compression of the right exiting L5 nerve root. No evidence of synovial cyst.

Intramedullary ependymoma: Evidence of intramedullary heterogeneously enhancing mass lesion with cystic areas and hemosiderin rim measuring about 10 × 14 × 62 mm (AP × TR × CC) at lower C2 to upper C7 level. Expanding the cord with cord edema extending up to the lower medulla superiorly and D3 level inferiorly.

Chest

COVID-19: Multiple focal patchy ground-glass opacities with interstitial thickening in bilateral lung fields predominately lower lobe and peripheral involvement—likely viral pneumonitis (CT chest severity score: 10/25), lung involvement 40% (moderate).

Percentage of lobe involved:

1. <5% involvement,
2. 5–25% involvement,
3. 26 to 50% involvement,
4. 51 to 75% involvement,
5. 76 to 100% involvement.

CT severity score: Severe: 16–25, moderate: 9–15, mild: 0–8.

Interstitial pneumonitis: Extensive intralobular and interlobular septal thickening, ground glass opacities in both lungs and minimal traction bronchiolectasis noted in the subpleural interstitium more severe in the basal segments of both lower lobes. Features favour interstitial pneumonitis.

***Usual interstitial pneumonia pattern*:** Subpleural interstitial thickening with early honey combing in both lungs with predominant involvement of the bases.

Hypersensitive pneumonitis: Crazy paving pattern of ground glass opacities with interstitial thickening in bilateral lower lobe lung with mosaic attenuation in bilateral upper lobe lung with sparing of basal segment—possible **hypersensitive pneumonitis.**