FRCR 2B Viva: A Case-Based Approach

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A Case-Based Approach

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Dedications

Thanks must go to my wife, Monica, and children, Francesca and Gianluca, for their remarkable tolerance.

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Foreword

This book is an excellent collection of cases ideal for the preparation of FRCR 2B Viva examinations. There is a great variety of cases with a wonderful section on tips for candidates appearing for the examination. This has been constructed from a continuum of knowledge gained over three decades from consultants who have experience of examining candidates and specialist registrars who have passed the examination. The cases span the spectrum of diseases commonly brought to the examination by generations of examiners. When I read the book it became obvious

that it is a vital link in the teaching portfolio because it demonstrates how to approach each case, pick up the radiological signs, and formulate an answer. There are notes related to the disease and a short bibliography. The contents cover gastrointestinal, chest, musculoskeletal, neuroradiology, urogynae-cological, paediatrics, and radionuclide imaging. This book is modern, up to date, and is based on organ imaging. I believe it will also prove to be an excellent revision book that will not become obsolete and is well worth owning.

Professor Philip Gishen, MB, BCh, DMRD, FRCR Imperial College Healthcare NHS Trust

Preface

There are several Fellowship of the Royal College of Radiologists (FRCR) Part 2B examination books on the market at present, detailing cases in a manner that will test the candidate. These books are of course an invaluable source of information and allow for self-testing. Here, we have designed a book for the FRCR 2B Viva examination not to test the candidate's knowledge, but to demonstrate the approach to the case and formulation of the ideal answer. The purpose of this book is not to test but to allow construction of the most suitable response in the examination.

The vast majority of candidates for the FRCR 2B examination are well prepared, with an extensive and comprehensive knowledge of the subject. The expression of this knowledge in the formal setting of the examination may, however, be a problem for some of the candidates. There is no point in knowing everything if you cannot demonstrate this to the examiner. Our aim is to establish a formula to

allow the candidate to achieve this; we have detailed the best approach to an image, and the response to further questions, in a manner that is succinct and clearly responds to the situation. Each case has a model answer, with all the relevant background information needed to confidently assess and detail an appropriate explanation of a film. The cases presented are classics in each section; the answers are models to build on; the background knowledge and bibliography are to be read and understood.

Each section is authored by a specialist registrar who has recently passed the FRCR 2B examination, supervised by an experienced consultant with a subspecialty interest, all of whom have at some time been teachers on the King's College Hospital FRCR 2B course. To this effect, we believe we have produced an ideal approach to assimilating the response desired by the examiner and providing success in the examination.

Paul S. Sidhu Suzanne M. Ryan Phillip F. C. Lung

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Paul S. Sidhu Suzanne M. Ryan Phillip F. C. Lung

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Introduction to the FRCR 2B Viva

A medical career is littered with numerous examinations, a necessity for climbing further up the ladder of specialisation and for the eventual attainment of a goal, in this case attainment of a postgraduate qualification and career in radiology. Very few people enjoy an examination, particularly an examination that involves "face-to-face" confrontation with two examiners; the "viva voce" beloved by purveyors of examinations. However, there is no substitute for this type of examination, stressful as it is for the candidate; the viva examination has from time immemorial been the best method of assessing skills of processing information and formulating an opinion. It is here to stay.

The candidate for the FRCR 2B examination is subject to a viva and will need to be fully prepared for this process. There is no reason why the candidate should be anything but fully confident in this process and be ultimately successful. But candidates will not pass the examination if they have not put in years of toil, reviewing all manner of images and building up a "databank" of knowledge. There is no substitute for hard work. Examinations are also a game, played out to achieve a result. Play the game right and you will pass. Once the knowledge has been attained by candidates, displaying all they have learned in a meaningful manner is the winning game.

A breakdown of the components will give a better understanding of the process.

The Candidate. After many years as a medical student, postqualification ward work, and often 3 years of basic grounding in radiology, the examination is tackled. At this stage, the candidate should have reviewed thousands of "examination" style films, in all imaging modalities, and have read around the subject every time a new disease or abnormality is encountered. Very often the presentation of a disease process is faced while "on call," when there is pressure to come to a diagnosis, a process that will reinforce any knowledge gained—do not underestimate the importance of emergency work.

The entire working life of a candidate up to the examination is a learning process, and the ability or desire to accumulate knowledge is the candidate's

own responsibility. If the hours of work and accumulation of knowledge have not been achieved, the candidate will not pass the examination. Nobody can teach this; this is an attitude that should be acquired from early in the candidate's career. Poor knowledge is very quickly spotted by the examiner. Do not blame others in your department for your failure—it is your responsibility to seek out, acquire, and consolidate knowledge. Teaching guides and directs you to the correct manner in which to use the knowledge you acquire by reading and experience.

Nevertheless, even the most prepared candidate will be a nervous wreck on examination day. The ability to process the knowledge and formulate an intelligent answer needs to be implicit—this is the "game." The game is not won without knowledge, but the presentation of the self-acquired knowledge in a meaningful manner can be taught. This is where pre-examination teaching is important; sit with your senior colleagues and practise the viva technique. Never go into an examination without being fully prepared as you will not succeed. Candidates need to have a rigid formula to present their knowledge in a manner that cannot be faulted.

The Examiner. Spare a thought for this poor soul—unpaid, unloved, and subject to 5 or more gruelling days of listening to anxious, nervous individuals who are often incoherent, all in a room 6 foot (or less) square, with no natural light. If this sounds cruel, it is, but the examiners keep coming back. The examiner has previously been a candidate and will understand the anxiety. The examiner will not only be knowledgeable, but most importantly will know exactly what is on the film being shown and will know the final diagnosis. The candidate will not. This is often the only differentiating factor between candidate and examiner, as often the breadth of knowledge is greater in the candidate.

The examiner is not out to fail the candidate. All the examiners are selected and trained to be decent, respectable examiners; they will do their utmost to settle the candidate, tease out knowledge, and try to get the candidate through the examination. No examiner will lead the candidate in the wrong direction or try to humiliate the candidate. But if candidates have poor knowledge, they will not succeed.

Two examiners are present, and both will assess the candidate and each other—if one examiner has been harsh in judging the candidate, the other will note this. The examiners are not out to fail a candidate—candidates fail because they were not good enough. The examiners have homes, families, and work places to go to after the examination period is over; they will be courteous and fair to all the candidates and will not single anyone out for "rough" treatment. The capability of the candidate is usually obvious at the end of the viva, as is whether the candidate has been successful. Rarely is a candidate subject to a review at the end of the process and there is disagreement.

The Examination. Confined in the small room are two examiners and a candidate, so pay attention to your personal hygiene. Dress in a presentable and nonprovocative manner. Be pleasant but not overly friendly; do not address the examiners by their first name. Do not rush to the examination hall but arrive with plenty of time to spare. Sit and have a cup of tea or coffee with colleagues, and go to the bathroom before the viva! Do not try to learn something new before going into the examination, as this is a waste of time and will push up your state of anxiety.

The examiner is likely to start off with a simple and straightforward film to ease you into the examination and to calm you down. You will not be given the "prizewinning" set of films of the rarest case for your first set of examination questions. Look at the film carefully, look at review areas, and think about the film. If you have worked hard, listened to your senior colleagues, read around the subject, been "on call," you will have encountered a similar film in the past. Do not blurt out an answer you cannot retract. Count to 5, and then describe what you see. Give the diagnosis if it is an "Aunt Minnie" (often this will be the case with the first film) and wait for the examiner to reply. This is not a dinner party or social occasion where you as the host need to keep a conversation going. Shut up when there is nothing more to say and let the examiner take you on to the next stage with a question. If you are correct, the film will be removed; if more is needed, a question will be asked. The examiner not involved in asking questions is the one doing the marking. No examiner is supposed to show films in their own subspecialty, and the other examiner will note this.

The number of films viewed in the course of the examination is no indication of your success: if you have reviewed only one complex case and have got

it correct, you have passed. Normally, there is plenty of time to show many cases. Never argue with the examiner, as it will get you nowhere and you will be likely to fail. All the films are vetted before the examination, and there are no films that are "wrong"—you do not have more insight than the panel of examiners. If the examiner tells you something that you disagree with, move on with the case or move to the next case. At the end of the examination, thank both examiners in a brief and professional manner. There is no place for a hug if you think you have done well.

After the Examination. There is a gap between the end of the examinations and the availability of the results; this is the time to "brag." Every candidate has been shown the worse possible combination of "sneaky" films designed to "trip up" innocent candidates and fail them outright. Nevertheless, the candidate, with incredible foresight and intelligence, has spotted this trap set by the evil examiners, designed to foil career progression, and spun out an incredible answer that could not be faulted, has undoubtedly shown the examiners to be clowns, and has secured a pass. It was even said that the President of the College had heard about this incredible candidate and had asked to personally meet to offer congratulations; a standing ovation among the examiners would precede this meeting.

It is true that candidates pass because they are good enough—candidates fail because they were not good enough. The tendency is often to dwell on the "missed" aspects of the films and forget the correct diagnosis made. This is a natural tendency, and it is best to wait for the result rather than agonise over the possibilities. Discussion after the examination is good for all; it lets off all that accumulated anxiety, so do it and embellish all you like as it does no harm.

So, in summary:

- 1. Knowledge is gained only by hard work.
- Teaching is desirable but is no substitute for reading and working.
- 3. A well-prepared candidate will not fail.
- 4. Teaching of examination technique is mandatory prior to the examination.
- 5. The examiners are not out to fail the candidate—lack of knowledge is easily evident.
- Think before you speak in the examination—you cannot retract what you have said.

The ability to formulate a suitable response forms the basis of the chapters of this book.

1 Introduction to Gastrointestinal Imaging

Phillip F. C. Lung and Suzanne M. Ryan

Good knowledge and understanding of gastrointestinal imaging is essential for success at radiological examinations. However, this can be difficult at a time when fluoroscopic techniques are on the wane and use of MRI on the rise; examinees are caught in the middle of changing clinical practice. Examiners tend to display barium studies (prized films kept as important teaching examples) that many candidates may not have experienced outside of educational examination courses.

While there is no substitute for experience, there are pointers that may make the interpretation easier.

- Plain abdominal films may be difficult to read, but can be broken down into "bite-size" chunks:
 - Bowel. The study has usually been performed to look at the bowel, in particular the bowel gas pattern. Unless there is an obvious abnormality elsewhere that immediately catches your eye, you should first assess the bowel; gaseous distension, constipation, absence of gas, dilatation, and abnormal wall are all important.
 - Intra-abdominal free gas. This should be excluded in all films in spite of whatever clinical details you have. Rigler's sign and retroperitoneal free gas outlining the kidney must be specifically checked for.
 - Solid organs. Are they enlarged or atrophic? Is there any associated calcification? Are they outlined by gas? Is there any gas within the structure (biliary or portal venous in the liver, gas within the bladder)?
 - Bone and joint degenerative disease is common, and bony destruction from tumour may be a pointer to the reason the film was shown.
 - Review areas. Look at the lung bases and hernial orifices.
- ② Be methodical and you will pick up the vast majority of lesions. Difficult films are there to identify candidates who are not methodical.
- 3 Barium studies remain a staple of the examination, despite the fact that fewer such examinations take place in routine clinical practice. As a result, it is unlikely that the candidate will be shown a subtle case, but the candidate should have good knowledge of the following types of cases:
 - Cancers of any part of the gastrointestinal tract must be searched for and excluded.

- Emergencies include volvulus and megacolon.
- Inflammatory bowel disease: Always look out for gallstones, sacroiliitis, and malignancy.
- Target lesions may be subtle and multiple.
- Changes may be seen in the overall fold pattern, as in coeliac disease and scleroderma.
- "Aunt Minnies": Achalasia, candidiasis, linitis plastica, and so on may all occur.
- Take your time to look at all of the opacified lumen. Always check the background chest or abdomen, as well as the bones. At the end of every review of a barium study, you must say that you would like to view the entire series as an important detail may be on a view that you have not yet seen.
- © CT and MRI are currently viewed on hard copies in the viva. As a result, there will be multiple images on each film, and the technique for viewing these is different from that of scrolling on a computer. This will alter in due course, with computer-based examinations being introduced; however, accumulation of sufficient examination cases will take time.
 - Have a quick look at the film to see if an obvious abnormality is apparent. Use the clinical information that is available to direct this.
 - If no overt abnormality is seen on the initial survey, concentrate on a single organ before moving to another organ. This will allow you to build up a picture and exclude abnormalities as you progress. If you try to look at every organ on one image before moving to another one, you will spend overall more time and may miss details.
 - There will often be several abnormalities on the film, and you need to pick them all up. Once you have picked up one abnormality, think about the clinical context to direct your search. For example, if the liver looks cirrhotic, make sure you exclude hepatocellular carcinoma, and look for portal vein thrombosis, varices, and ascites.
- (5) All your cases should end by discussing the management options. Try to remember what you do at your institution. Local multidisciplinary clinical meetings are the ideal environment to pick up these details. In your preparation, you should always know what the management is for any particular case. If you do not know, ask.

Achalasia 1 1

Clinical History

A 42-year-old woman presents with a history of dysphagia (Fig. 1.1.1).



Fig. 1.1.1

Ideal Summary

This is a frontal chest radiograph of a woman. There is a double right cardiac contour with a right paravertebral opacity that extends below the level of the hemidiaphragm (Fig. 1.1.1, arrow). No air-fluid level is seen. There is no bony destruction or loss of disc space in the adjacent thoracic spine. The lungs and pleural spaces are clear. The hila appear unremarkable, and the aortic knuckle is to the left of the midline. I would like to compare this with any previous films. Given the clinical history, the opacity is most likely due to a dilated oesophagus. Causes for this include achalasia and malignancy. I would confirm this with a barium swallow examination.

These are two images from a barium study on the same patient (Fig. 1.1.2).

These are selected images from a contrast swallow study. A grossly distended oesophagus is seen overlying the mediastinum. There is a transition point in the region of the gastro-oesophageal junction (Fig. 1.1.2, arrow) with smooth narrowing into a "bird's beak" (also known as a "rat's tail"). Contrast is seen distally within the stomach. No mucosal abnormalities are identified. I would like to review the remainder of the series. The most likely diagnosis is achalasia, and I would recommend that this patient be referred to the gastroenterology team.

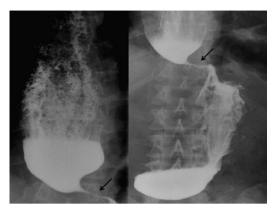


Fig. 1.1.2

Examination Tips

- Always ask for the full series of films if it is available.
- Check the outline of the oesophagus, and if there is massive dilatation of the oesophagus, consider either achalasia or previous oesophageal resection and colonic bypass.
- Comment on the outline of the oesophagus
 - If it is smooth, consider achalasia.
 - If there is focal irregularity, consider oesophageal malignancy.
 - If diffuse irregularity is present, candidiasis may produce oesophageal dilatation with aperistalsis.
- Always check the stomach as primary gastric carcinoma with gastro-oesophageal stricture may produce a similar appearance.
- O Comment on the gastro-oesophageal junction. If it is patulous, this may be from scleroderma or drugs; if it is narrowed, this may indicate achalasia, malignancy, postinflammatory or extrinsic compression.
- Look at the lung fields for evidence of aspiration.

Differential Diagnosis

The imaging appearances shown in Fig. 1.1.2 are classic for achalasia. However, important differential diagnoses to consider in a case of oesophageal narrowing include:

- Oesophageal malignancy:
 - There is likely to be an irregular contour of the distal oesophagus with possible shouldering present.

- The narrowed distal segment may produce a rat's tail appearance.
- It may occasionally produce imaging findings similar to those of achalasia with a smooth symmetrical narrowing and aperistaltic dilated proximal oesophagus.
- Inflammatory stricture at the gastro-oesophageal junction:
 - Typically, there is smooth narrowing of a short segment of distal oesophagus.
 - Peristalsis is maintained, and the degree of oesophageal dilatation is less than that found in achalasia.
 - Ulcers and thickened mucosal folds may suggest an inflammatory component.

Notes

 Achalasia is a motility disorder with failure of relaxation of the lower oesophageal sphincter.

- Primary achalasia occurs in the 30- to 50-year age group.
- Primary achalasia is an idiopathic process, while secondary achalasia may be caused by malignancy, inflammatory strictures, and scleroderma. The oesophageal dilatation in secondary achalasia (< 4 cm) is typically less than that found in primary achalasia (> 4 cm). The narrowing of the distal oesophagus in secondary achalasia may be irregular, reflecting the underlying cause.
- Primary peristalsis is absent in classic achalasia.
- There is an increased risk of squamous cell carcinoma of the order of 2 to 7%.

Bibliography

Cole TJ, Turner MA. Manifestations of gastrointestinal disease on chest radiographs. Radiographics 1993;13(5):1013–1034

Jang KM, Lee KS, Lee SJ, et al. The spectrum of benign esophageal lesions: imaging findings. Korean J Radiol 2002;3(3):199–210

Acute Appendicitis

Clinical History

A 23-year-old man presents with right-sided abdominal pain (Fig. 1.2.1).



Fig. 1.2.1

Ideal Summary

This is a plain film abdominal radiograph of an adult. Two calcific densities can be seen in the right hemipelvis (Fig. 1.2.1, arrow). They do not have the central lucency typical of phleboliths. There are no dilated bowel loops or any evidence of free intraperitoneal gas. The appearances are nonspecific, but, given the clinical history, an important differential diagnosis to consider would be an appendicolith. I would perform an ultrasound examination to assess the right iliac fossa to see if an abnormal appendix could be identified.

These are two images from the ultrasound examination on the same patient (Fig. 1.2.2).

These are ultrasound images of a blind-ending viscus, likely the appendix, with high reflectivity

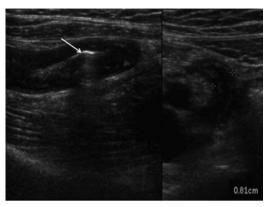


Fig. 1.2.2

centrally (Fig. 1.2.2, arrow) and posterior acoustic shadowing that may represent an appendicolith or gas. There is thickening of the appendix wall, with a combined wall thickness greater than 7 mm in diameter (0.81 cm between the callipers). No free fluid is seen. The imaging findings are in keeping with acute appendicitis, and I would urgently refer the patient to the surgical team for consideration for an appendicectomy.

These are further images from the same patient; a CT examination was performed (Figs. 1.2.3 and 1.2.4).

These are selected axial images of a contrast-enhanced CT examination. There is a calcified appendicolith (Fig. 1.2.3, arrow) seen at the base of the appendix, and the appendix itself is distended with fluid



Fig. 1.2.3



Fig. 1.2.4

(**Fig. 1.2.4**, arrow). There is fat stranding around the appendix with free fluid in the pelvis. There is no evidence of a mass at the appendix level or free gas to suggest a perforation.

Examination Tips

If you are shown a case of appendicitis, be aware of the specific findings within and the limitations of each modality:

- Plain film:
 - Only 5 to 10% of appendicoliths are visible on a plain abdominal film.
 - Appendicitis may result in small bowel obstruction.
 - An appendiceal mass may displace bowel loops away from it and appear as an ill-defined area of increased density.

Ultrasound:

- The appendix should be seen as blind-ending and arising from the caecum. It is best seen using a high-frequency linear transducer, especially in children.
- An appendicolith may be seen as intraluminal high reflectivity with posterior acoustic shadowing, although gas may produce a similar finding.
- An inflamed appendix is noncompressible, with a combined wall thickness measuring greater than 7 mm; associated free fluid may also be seen.
- CT scanning:
 - Fat stranding and free fluid are often the first clues to locating the abnormality.
 - Always check for adjacent extraluminal free gas.

Differential Diagnosis

In a patient with the features demonstrated above, no differential diagnoses need to be offered.

Notes

- Classic clinical symptoms are periumbilical pain moving to the right iliac fossa. However, atypical signs are seen in one-third of patients.
- Around 95% of patients with acute appendicitis have a raised white blood cell count.

Bibliography

Gaitini D. Imaging acute appendicitis: state of the art. J Clin Imaging Sci 2011;1:49

Carcinoid Tumour 13

Clinical History

A 55-year-old man presents with a history of watery diarrhoea (Figs. 1.3.1 and 1.3.2).

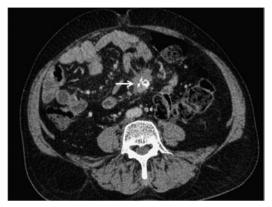


Fig. 1.3.1



Fig. 1.3.2

Ideal Summary

These are axial CT images through the abdomen with intravenous contrast enhancement. There is a spiculated soft tissue mass, lying centrally, seen within the mesentery and containing focal areas of calcification (Fig. 1.3.1, arrow). A desmoplastic reaction is centred on the mesenteric soft tissue (Fig. 1.3.2, arrow) with tethering of several small bowel loops. The bowel appears unremarkable. Hazy fat stranding of the adjacent mesentery is also present. The most likely diagnosis is a carcinoid tumour. Given the clinical history, I would like to examine the liver to assess the

patient for liver metastases and also look for a possible primary tumour in the bowel.

Here is further imaging performed on the patient (Fig. 1.3.3).

This is an octreotide nuclear medicine study taken at 24 hours. Several focal areas of increased uptake are seen within the centre of the abdomen, corresponding to the mesenteric mass seen on the CT examination. A smaller focus of increased uptake is seen within the anterior liver. No other sites of abnormal increased uptake are seen. The findings are consistent with a mesenteric carcinoid tumour and liver metastasis. I would like to assess the liver on a biphasic CT.

Examination Tips

When dealing with a mesenteric mass, comment on the following:

- Is the lesion well-defined or irregular?
- Is there spiculation to suggest a desmoplastic reaction?
- When multiple mesenteric masses are present, necrotic change is more in keeping with infection with tuberculosis.
- Calcification may represent treated disease and may be either lymphoma or tuberculosis.
- Look for complications associated with a carcinoid tumour:
 - Tethered small bowel loops, which may cause adhesional small bowel obstruction.
 - Small bowel ischaemia related to mesenteric vessel infiltration.
 - Intussusception with a carcinoid tumour as the lead-point.

Differential Diagnosis

There is no differential diagnosis for the above imaging appearances.

Notes

Carcinoid tumours are most common in patients aged 50-60 years, and are twice as common in men.

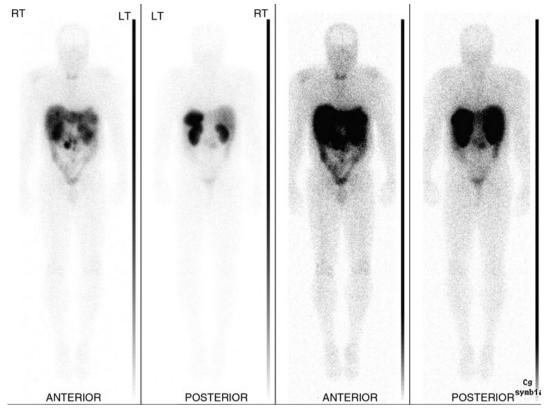


Fig. 1.3.3

- Patients with carcinoid tumours are most commonly asymptomatic.
- Carcinoid syndrome occurs in 10% of patients, in the presence of liver metastases, and the patient may experience flushing, watery diarrhoea, and bronchial constriction.
- Carcinoid syndrome is associated with cardiac abnormalities in two-thirds of patients, including pulmonary/tricuspid stenosis or regurgitation; there may be right heart enlargement.
- A total of 30% of carcinoid tumours are multiple.
- The primary gastrointestinal carcinoid tumour is submucosal, and a barium follow-through examination was historically used to detect this, although this was associated with a poor sensitivity when the primary was less than 2 cm in size
 - Carcinoid tumours may appear as smooth intraluminal defects and may appear as a "target" lesion if there is ulceration.
 - There may be thickening of the mucosal folds caused by the primary tumour.

- With mesenteric involvement and a desmoplastic reaction, there may be tethering and angulation of the small bowel loops.
- A small bowel carcinoid tumour is better depicted on triphasic CT enterography with negative (or neutral) oral contrast, as the submucosal masses are highly vascular.
- Liver metastases are also highly vascular and must be assessed on both arterial and portovenous phase CT imaging.
- Iodine 131-MIBG may be used to detect neuroendocrine tumours such as carcinoid tumours, pheochromocytoma, and neuroblastoma.
- Indium 111-octreotide has a sensitivity of 75% and a specificity of 100% in the detection of carcinoid tumours.

Bibliography

Horton KM, Kamel I, Hofmann L, Fishman EK. Carcinoid tumors of the small bowel: a multitechnique imaging approach. AJR Am J Roentgenol 2004;182(3): 559–567

Cirrhosis 1 4

Clinical History

A 54-year-old man presents with a history of abdominal pain (Figs. 1.4.1 and 1.4.2).



Fig. 1.4.1



Fig. 1.4.2

Ideal Summary

These are selected axial portal venous phase images through the upper abdomen. The contour of the liver is irregular throughout, and the parenchyma is heterogeneous, but there are no focal intrahepatic lesions. I can see a filling defect within the main portal vein (Fig. 1.4.2, arrow) in keeping with a portal vein thrombus. The spleen appears enlarged, but I would like to see a coronal reformat to confirm this. A small volume of ascites is present in a perihepatic distribution. I can see no evidence of variceal formation. The features are in keeping with decompensated liver cirrhosis with portal vein thrombosis. I would refer the patient to the hepatologists for further management.

Examination Tips

In any patient with suspected cirrhosis, look for the following:

- Irregular hepatic contour
- Enlargement of the caudate lobe
- The presence of biliary duct dilatation, which raises the possibility of primary sclerosing cholangitis
- Focal hepatic lesions: hepatocellular carcinoma is a recognised complication
- O Portal vein thrombus: also look for a portal vein cavernoma following thrombosis
- Sequelae of portal vein hypertension:
 - Left gastric and oesophageal varices; recanalisation of the umbilical vein within the falciform ligament: venous collaterals in the anterior abdominal wall
 - Mesenteric oedema due to raised venous pressure
 - Splenomegaly
- The presence of ascites, which is suggestive of liver function compromise and decompensation

Differential Diagnosis

There is no differential diagnosis for the above imaging appearances.

Notes

- The liver margin may be smooth, nodular, or lobular; however, this does not correspond to the underlying cause of the cirrhosis.
- The presence of a mass, focal increased arterial enhancement, with portal venous washout and arterio-portal shunt are all suggestive of a hepatocellular carcinoma.

A transjugular intrahepatic portosystemic stentshunt will divert flow from the portal venous circulation and into the hepatic vein. This will improve portal hypertension and has a role in the treatment of variceal bleeding.

Bibliography

Brancatelli G, Federle MP, Ambrosini R, et al. Cirrhosis: CT and MR imaging evaluation. Eur J Radiol 2007; 61(1):57–69

Colorectal Cancer

Clinical History

A 70-year-old man presents with a history of weight loss (Figs. 1.5.1 and 1.5.2).



Fig. 1.5.1 **Ideal Summary**

These are selected images from a barium enema series. There is a short segment of irregular circumferential narrowing in the mid-transverse colon with an "apple core" appearance (Figs. 1.5.1 and 1.5.2, arrow). Multiple diverticula are seen throughout the colon. No other lesions are seen in the remainder of the visualised colon, and there is no proximal bowel dilatation present to suggest bowel obstruction. The most likely diagnosis is colorectal carcinoma. I would urgently refer the patient to the surgical team and arrange a staging CT examination.

This is another case from a different patient, with CT colonoscopy images (Figs. 1.5.3 and 1.5.4).

This is an axial CT colonography image through the abdomen with a three-dimensional intraluminal view. There is a polypoid mass in the right colon (Fig. 1.5.3,

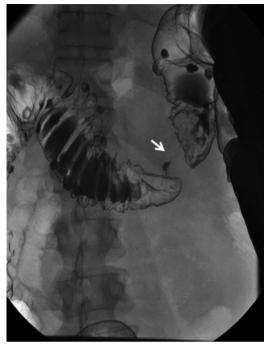


Fig. 1.5.2

arrow), which exhibits a "saddle-shape" morphology on the three-dimensional image (Fig. 1.5.4, arrow). I cannot see any soft tissue extending beyond the muscularis propria or enlarged mesenteric or retroperitoneal lymph nodes. I would like to review the remainder of the images. The findings are suspicious for a polyp malignancy. I would ensure a staging CT was performed. At my institution, I would refer the patient for a same-day endoscopy examination and discuss this further at the multidisciplinary meeting.

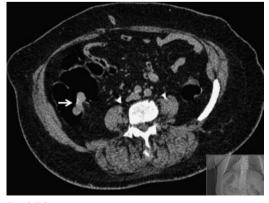


Fig. 1.5.3



Fig. 1.5.4

Examination Tips

- When assessing a stricture on a barium enema series, look at:
 - The site of the lesion.
 - The configuration of the colon. Has there been previous surgery? Is the site of stricture at an anastomosis?
 - Is it in a watershed area? This raises possibility of ischaemia.
 - Length. Malignant strictures tend to be shorter in length. A longer length stricture may be secondary to colitis, diverticulitis, or ischaemia.
 - Is the stricture smooth or irregular?
 - Are the ends of the stricture tapered or shouldered?
 - Assess for the presence of diverticula.
- In a primary colonic malignancy, look specifically for a synchronous lesion (5%) and polyps (30%).
- Always check for and comment on complications: obstruction, perforation, or invasion into adjacent organs.
- Assess the bones (on the correct window settings) for metastases.
- On CT colonography, polyps that demonstrate the following morphology are suspicious for malignancy:
 - Saddle-shaped lesions
 - Polyps with a central depression
 - Flat polyps: you are unlikely to have this in the examination as it is a difficult lesion to identify.

Differential Diagnosis

There are no differential diagnoses for the above appearances. However, the differentials for a colonic

stricture include the following:

- Ischaemic colitis
 - This usually occurs in watershed areas, and there may be evidence of "thumb-printing."
 - A smooth stricture on barium enema with tapered margins may be seen in chronic cases.
- Diverticulitis
 - This is most often seen in the sigmoid colon and is usually a long (> 5 cm) segment of luminal narrowing on a background of multiple diverticula.
- Extrinsic pathology
 - There is smooth narrowing making obtuse angles with bowel wall, usually on the antimesenteric border of the colon.
 - Causes for this include endometriosis and ovarian malignancy.

Notes

Around 25% of colorectal carcinoma occurs in the sigmoid colon, 20% in the rectum, and 15% in the transverse and ascending colon.

Regarding colorectal cancer screening in the UK:

- Offered to men and women aged 60 to 69 years every 2 years (extending up to 75 years in some areas).
- If there is a positive faecal occult blood test, the individual is referred for colonoscopy.
- CT colonography is offered when:
 - Colonoscopy has been incomplete
 - The patient is unfit or unsuitable for colonoscopy
 - The patient refuses colonoscopy.

CT Colonography

- Look for a rectal insufflation tube to confirm the type of study.
- Depending on the institution, the study may involve a low radiation dose with or without intravenous contrast.
- Oral contrast produces a "faecally tagged" study.
- Reconstruction of the CT data may be used to produce a three-dimensional volume-rendered intraluminal image – "virtual endoscopy."

Bibliography

Dighe S, Swift I, Brown G. CT staging of colon cancer. Clin Radiol 2008;63(12):1372–1379

http://www.cancerscreening.nhs.uk/bowel/index.html (accessed Nov 2012)

1.6 Crohn's Disease

Clinical History

A 33-year-old man presents with abdominal bloating and pain (Fig. 1.6.1).



Fia. 1.6.1

Ideal Summary

This is a selected image from a barium followthrough study. There are several loops of abnormal distal small bowel that are narrowed with "cobblestoning" and linear ulceration (Fig. 1.6.1, arrow) separated by shorter distended loops of ileum. The distal ileal loops show separation. No definite fistulas are seen. The appearances of the right colon are suggestive of previous resection with a short segment of narrowing at the ileocolonic anastomosis, although contrast is seen within the colon. The features are consistent with active Crohn's disease. I would like to assess the remainder of the series and any old films.

Examination Tips

In a case with Crohn's disease, comment on:

 Location, Although Crohn's disease can occur anywhere along the gastrointestinal tract, there is usually involvement of the terminal ileum with

- small bowel involvement. Describe where exactly the abnormalities are: proximal, mid, or distal small bowel, terminal ileum, or colon
- Distribution, Are there skip lesions, and are the abnormalities aymmetrical?
- Luminal narrowing. Is there any contrast downstream of the narrowing; is there any pre-stenotic bowel dilatation?
- Ulceration. Are there aphthous ulcers that appear as target lesions, linear ulcers that cross perpendicular to the folds, fissuring, or cobble-stoning?
- Fistula formation: enteroenteric, enterocolonic, or enterocutaneous
- Bowel loop separation
- Obstruction: upstream bowel dilatation
- Evidence of previous operations
- Associations with Crohn's disease: gallstones or sacroiliitis: comment if these are present on the barium image.

Differential Diagnosis

No differential diagnoses should be given in a case of classic Crohn's disease. However, if there is only isolated terminal ileal abnormality, the following should be considered:

- Lymphoma
- Tuberculosis
- "Backwash" ileitis with ulcerative colitis
- Ischaemia

Notes

- There is an equal male to female distribution.
- Two peaks in incidence occur at ages 15 to 25 and 60 to 70 years.
- Small bowel follow-through was historically frequently used in the diagnosis of Crohn's disease, but this is now being replaced by CT and MR enterography
 - A small bowel follow-through and CT enterography are able to detect mucosal abnormalities.
 - A small bowel follow-through and MRI cine loops help provide functional and dynamic information such as peristalsis and the presence of small bowel adhesions.
 - It may help estimate the extent of the disease and the length of remaining bowel.

• It may be used to map small bowel fistulas associated with Crohn's disease.

Bibliography

Nolan DJ, Gourtsoyiannis NC. Crohn's disease of the small intestine: a review of the radiological appearances in

100 consecutive patients examined by a barium infusion technique. Clin Radiol 1980;31(5):597–603
Sinha R, Murphy P, Hawker P, Sanders S, Rajesh A,
Verma R. Role of MRI in Crohn's disease. Clin Radiol 2009;64(4):341–352

Focal Nodular Hyperplasia

Clinical History

A 35-year-old asymptomatic woman (Fig. 1.7.1).

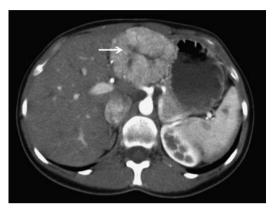


Fig. 1.7.1

Ideal Summary

This is a selected axial arterial-phase CT image through the upper abdomen. There is an enhancing lesion in the left lobe of the liver, with a central lowdensity scar and radiating septa (Fig. 1.7.1, arrow). There are no other liver lesions. There are no features to suggest liver cirrhosis. The most likely diagnosis is focal nodular hyperplasia (FNH), and other differential diagnoses would include hepatic adenoma and hepatic malignancy. I would like to ask if there is any venous-phase imaging to confirm my diagnosis.

This is the venous-phase CT image at the same level (Fig. 1.7.2).

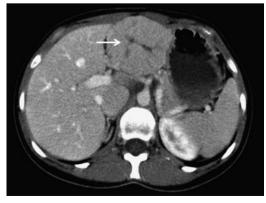


Fig. 1.7.2

The venous-phase CT image demonstrates an enhancement pattern within the lesion, which is isodense or slightly hypodense to the liver. The central scar remains of low density with no evidence of enhancement (Fig. 1.7.2, arrow). The features are consistent with a diagnosis of FNH.

Examination Tips

When dealing with liver lesions, ask yourself the following questions:

- Is it single or multiple? Multiple lesions will favour metastatic disease, while a single lesion is more likely hepatic adenoma, FNH, or fibrolamellar hepatocellular carcinoma (HCC).
- Is there homogeneous or heterogeneous enhancement? Homogeneous lesions include FNH or smaller hepatic adenoma, while malignancy must be excluded in heterogeneous enhancing lesions.
- Is there a scar? If so, it is more likely to be FNH or fibrolamellar HCC.
- Is there background hepatic cirrhosis? If yes, HCC should be the primary diagnosis, unless there is strong evidence to suggest otherwise.
- What is the enhancement pattern?
 - Arterial phase: FNH, hepatic adenoma, HCC, and neuroendocrine metastases are hyperenhancing. Other metastases are usually hypodense to liver.
 - Portal venous phase: hepatic adenoma and FNH are usually isodense compared with liver; malignancies are usually hypodense to liver.
 - Delayed phase: FNH and fibrolamellar HCC are isodense to liver; hepatic adenoma, HCC, and metastases are hypodense compared with
 - Is there disruption to (haemorrhage of) the lesion? This favours hepatic adenoma or HCC.
 - Extrahepatic primary tumour: look specifically for this.

Differential Diagnosis

The diagnostic possibilities for an arterial-phase enhancing lesion include:

- Hepatic adenoma:
 - Heterogeneous enhancement on arterial phase
 - Variable enhancement on portal venous phase
 - May be associated with disruption (haemorrhage)

- Fibrolamellar HCC:
 - Usually very large and heterogeneous
 - Presence of metastatic deposits in 70% of cases
- Metastases:
 - Usually multiple and in an older population
 - Washout during the portal venous phase
- "Flash" haemangioma: a lesion that fills rapidly and not visibly from the periphery:
 - Should continue to enhance on delayed-phase imaging

Notes

- Focal nodular hyperplasia is the second most common benign liver tumour.
- There is usually only a single lesion in 80% of cases.
- Oral contraceptives do not cause FNH but have a positive effect on its growth.
- The lesion contains both hepatocytes and noncommunicating biliary structures.
 - Gadolinium BOPTA (gadobenate dimeglumine) is excreted by the biliary epithelium.
 - It produces delayed and persistent enhancement on MRI.
 - This may help differentiate FNH from hepatic adenoma, which will typically not enhance on a hepatocyte-specific phase with gadolinium BOPTA.

- Approximately 20% of FNHs are atypical.
 - This includes telangiectatic FNH, which does not have a central scar, demonstrates persistent enhancement, and exhibits a high T1 and T2 signal on MRI.
 - However, atypical FNH still comprises biliary epithelium, and gadolinium BOPTA scanning may help in differentiating.
- Ultrasound findings:
 - It is homogeneous and isoechoic to liver, with a hypoechoic central scar.
 - It may demonstrate a "spoke-wheel" pattern on colour Doppler imaging.
 - Contrast-enhanced ultrasound demonstrates the classical "spoke-wheel" pattern and persisting enhancement into the late portovenous phase.
- MRI findings:
 - Tumour parenchyma: T1 ↓ /↔, T2 ↑ /↔
 - Central scar: T1 ↓, T2↔
 - Postcontrast: hyperintense on the arterial phase, and then isointense on the venous phase.

Bibliography

Kehagias D, Moulopoulos L, Antoniou A, et al. Focal nodular hyperplasia: imaging findings. Eur Radiol 2001;11(2):202–212