RADIOLOGY EVENTS
READ
AND DISCREPANCIES

ISSUE 14

THE ROYAL COLLEGE OF RADIOLOGISTS
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INTRODUCTION

Welcome to the 14th edition of READ and many thanks to those of you who have contributed cases of radiological discrepancy with useful learning points to share with your radiology colleagues.

Error or discrepancy occurs in about 4% of all imaging examinations. Fortunately, due to all the safeguards that hospitals and departments have in place, it is rare for a patient to be harmed by a single error. As radiologists, we tend to obsess about discrepancy in image interpretation, particularly the 'missed finding' or perceptual error. This is understandable as, unlike other doctors, every clinical act we perform is available for reinterpretation any time in the future thanks to picture archiving and communications systems (PACS)! The shared learning that takes place in local learning from discrepancy meetings (LDMs) and through reading this newsletter is one of the safeguards that alerts us to potential imaging pitfalls. We should, however, also be aware of all the other safeguards in our practice and make sure that these are as robust as possible.

Two cases in this newsletter highlight system rather than human failure. READ 057, submitted by a trainee, is a good example of a scenario where a diagnostic error made by an inexperienced radiologist was corrected swiftly by excellent and accessible consultant supervision. The trainee learned a valuable lesson by having to make a preliminary report on their own but in a supportive environment. Are you sure that trainees are as well-supported in your own department? READ 058 is a salutary tale of system failure where a liver lesion, correctly identified and recommended for further work-up, was not investigated further by the clinical team resulting in delayed diagnosis for the patient. Do you have a robust system in place in your own department to ensure that important findings are acted on by the clinical team?

Well-run LDMs should highlight system failures. Having identified examples of system failure at your local LDM, what steps have you taken to prevent such a discrepancy happening again? If you have found a local solution (other than locking the reporting room door!), please share it with other radiologists through the READ newsletter.

We are delighted to announce that the next edition will feature an invited review by the RCR Human Factors Adviser, Paul McCoubrie, who will add his insight into the published cases

READ on …

Submit your case at
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READ 055

IS IT REALLY THE URINARY BLADDER?

Case 1: An elderly female with intermittent right iliac fossa pain was referred by her general practitioner (GP) for a computed tomography (CT) kidneys, ureters, bladder (KUB). The CT study was reported negative for renal tract calculi or other cause for the patient's symptoms. Pelvic ultrasound subsequently demonstrated an 8 cm simple right ovarian cyst. On review of the CT the cyst could be seen deep in the pelvis in the midline, separate from the empty urinary bladder (Figure 1). The patient continued to have symptoms that were attributed to intermittent right adnexal torsion and subsequently underwent right salpingo-oophorectomy.

Case 2: A middle-aged female with intermittent lower abdominal pain was referred by her GP for a CT KUB. The CT was reported as negative for renal tract calculi or other abnormality. Pelvic ultrasound subsequently demonstrated a 10 cm right ovarian cyst with several mural nodules. On review of the CT, this cyst could be seen in the pelvis, slightly to the right of the midline, superior to the empty urinary bladder (Figure 2). After oophorectomy the lesion was histologically reported as a serous cystadenofibroma of the right ovary.

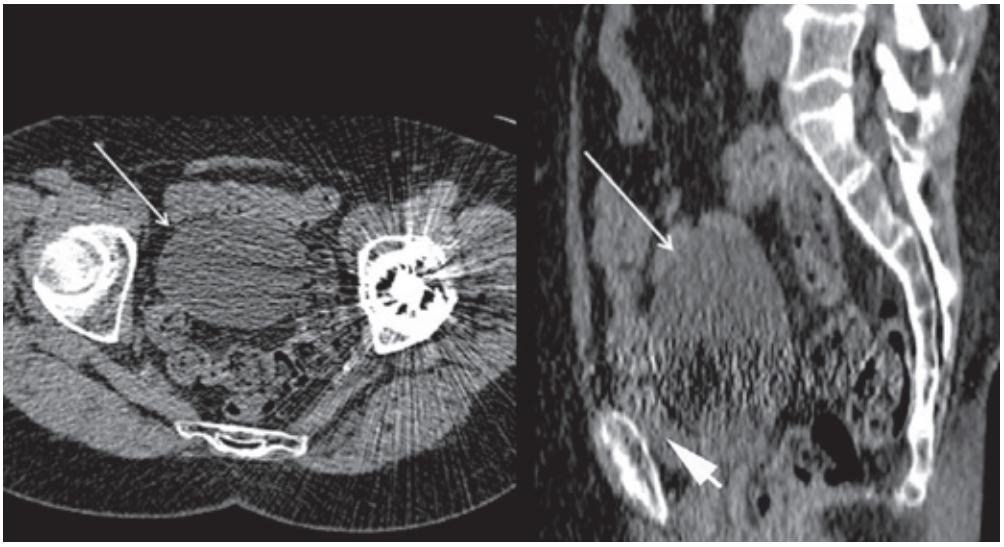

Figure 1. Axial and sagittal CT images from the patient in Case 1 demonstrating the ovarian cyst (thin arrow) and the collapsed urinary bladder (thick arrow).

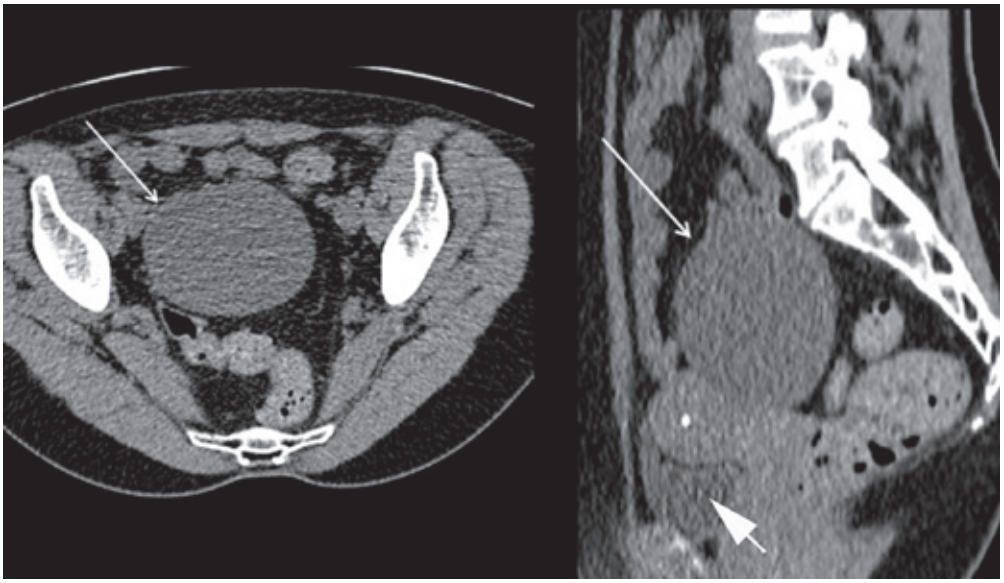

Figure 2. Axial and sagittal CT images from the patient in Case 2 demonstrating the ovarian cyst (thin arrow) and the collapsed urinary bladder (thick arrow).

REPORTER'S COMMENTS

Main causes

- In both cases the reporting radiologist had mistaken an ovarian cyst for the urinary bladder.
- The urinary bladder was collapsed in both cases, making it more difficult to identify.
- Both cases were performed without intra venous (IV) contrast administration (CT KUB). In Case 1, artefact from a left hip prosthesis further compromised radiological assessment of the pelvis.

LESSONS LEARNED

Midline ovarian cysts are easy to mistake for the urinary bladder, particularly if the bladder is empty. Careful inspection of multiplanar reconstructions, especially the sagittal view, will reduce the likelihood of making such an error.

In 38% of females, flank pain has a gynaecological cause so alternative diagnoses should be sought when reporting CT KUB in female patients.[1]

CT KUB should be reserved for patients with a high index of suspicion of renal tract calculi. If protocolled appropriately, calculi should be detected in at least 44% of CT KUB with alternate diagnoses made in at least a further 6% as discussed in an RCR audit for the investigation of renal colic with CT-KUB.[2] Consider IV contrast-enhanced CT for patients presenting with symptoms not not typical of ureteric or renal colic.

References

1. Nadeem M, Ather MH, Jamshaid A, Zaigham S, Mirza R, Salam B. Rational use of unenhanced multi-detector CT (CT KUB) in evaluation of suspected renal colic. *Int J Surg* 2012; **10**(10): 634–637.
2. **www.rcr.ac.uk/audit/radiological-investigation-renal-colic-following-introduction-ctkub** (last accessed 02/02/17)

READ 056

A DIFFUSE BRAIN ABNORMALITY

A middle-aged patient came to the accident and emergency (A&E) department following cardiac arrest. The patient was unresponsive and ventilated. Initial CT of the brain was reported as 'no intracranial haemorrhage or any acute intracranial pathology'. The referring clinician requested a neuroradiology opinion as he suspected hypoxic ischemic injury. He also provided the additional history of difficult intubation. Review of the initial CT (Figure 1) revealed diffuse cerebral swelling with widespread loss of parenchymal grey-white matter differentiation, in keeping with global hypoxic ischemic injury. Follow-up CT showed further increase in the extent of cerebral swelling and coning (Figures 2 and 3). The patient died two days later.

REPORTER'S COMMENTS

Main causes

- Lack of appreciation of the signs of widespread loss of grey-white matter differentiation, basal ganglia low density and generalised cerebral swelling.
- Despite the history of cardiac arrest, the reporting radiologist did not assess specifically for signs of hypoxic ischaemic injury.
- Perceptual error as symmetrical or more diffuse change may be harder to discern.
- Even though the history of difficult intubation was not given to the original reporter the diagnosis of hypoxic ischaemic injury should have been considered in the context of unconsciousness after cardiac arrest.

LESSONS LEARNED

Always look for signs of hypoxic ischemic injury in cases with cardiac arrest, difficult intubation and clinically unresponsive patients.[1]

The cortical grey and white matter interface should be inspected carefully on all brain CTs. More focal, asymmetrical loss may indicate territorial infarction. Diffuse loss of the grey-white matter interface, however, is a sign of severe global hypoxic ischemic injury and should be specifically looked for, particularly if it is suspected clinically. Switching between normal brain window setting and a 'stroke window' that narrows the window width to accentuate grey/white matter contrast can help to make such an abnormality more conspicuous.[2] Changes may be subtle in the early stages of the process and a follow-up study may be helpful.

Careful inspection to assess the convexity sulcal spaces is also essential. As swelling progresses the conspicuity of the convexity sulcal pattern becomes reduced. 'Pseudo-subarachnoid haemorrhage' may be seen due to relative hyperattenuation of the sulcal spaces mimicking true subarachnoid haemorrhage in cases of diffuse cerebral oedema.[3]

If the scan findings are not compatible with the clinical picture, further discussion with the referring clinician is often helpful.

References

1. Huang BY, Castillo M. Hypoxic-ischemic brain injury: imaging findings from birth to adulthood. *Radiographics* 2008; **28**(2): 417–439.
2. Lev MH, Farkas J, Gemmete JJ *et al*. Acute stroke: improved non-enhanced CT detection – benefits of soft copy interpretation by using variable window width and centre level settings. *Radiology* 1999; **213**(1): 150–155.
3. Given CA, Burdette JH, Elster AD, Williams DW 3rd. Pseudo-subarachnoid hemorrhage: a potential imaging pitfall associated with diffuse cerebral edema. *AJNR Am J Neuroradiol* 2003; **24**(2): 254–256.

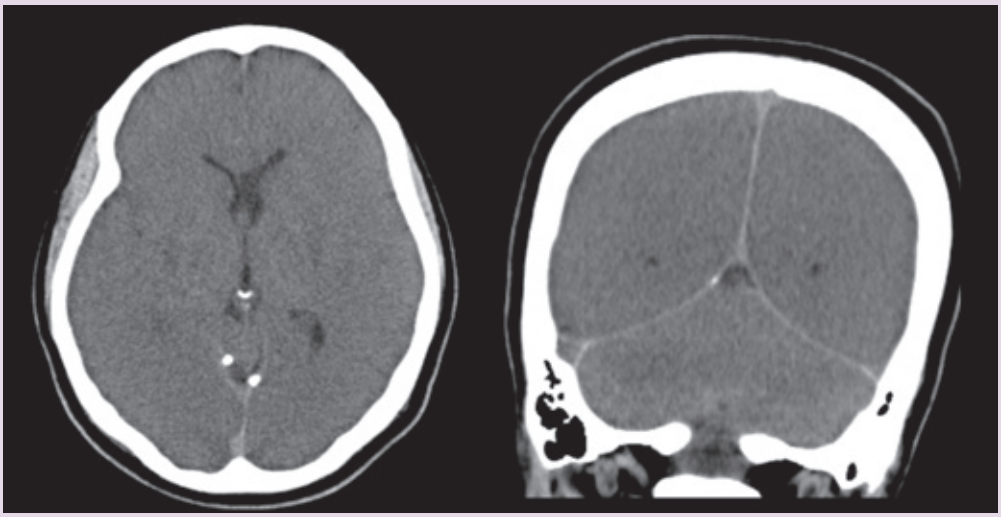

Figure 1. Axial and coronal sections from the initial CT show diffuse cerebral oedema with diffuse loss of grey-white matter differentiation. In addition, there are hypodensities in the basal ganglia bilaterally. The coronal view demonstrates the cerebellar involvement and relative hyperdensity of the falx and tentorium.

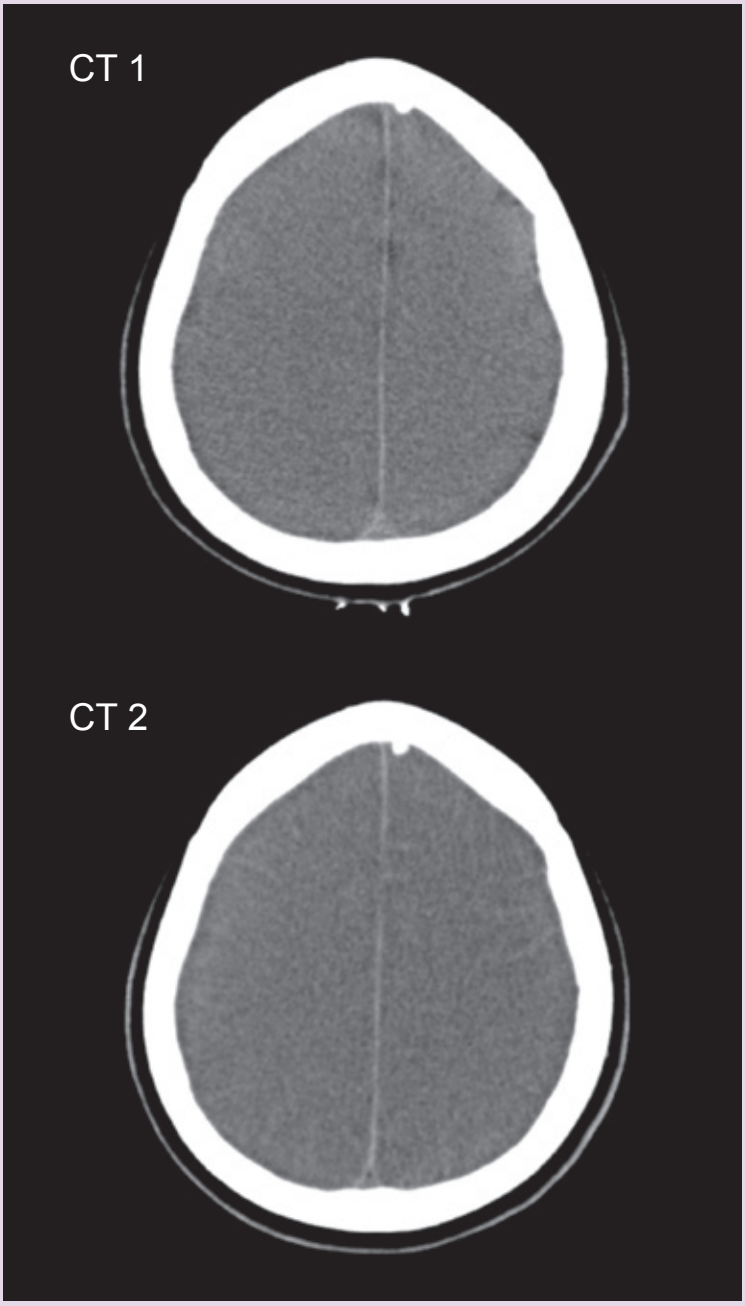

Figure 2. Axial CT sections from the initial and subsequent CT studies demonstrating progression of the cerebral oedema and development of hyperdense sulcal spaces ('pseudo-subarachnoid haemorrhage' sign).

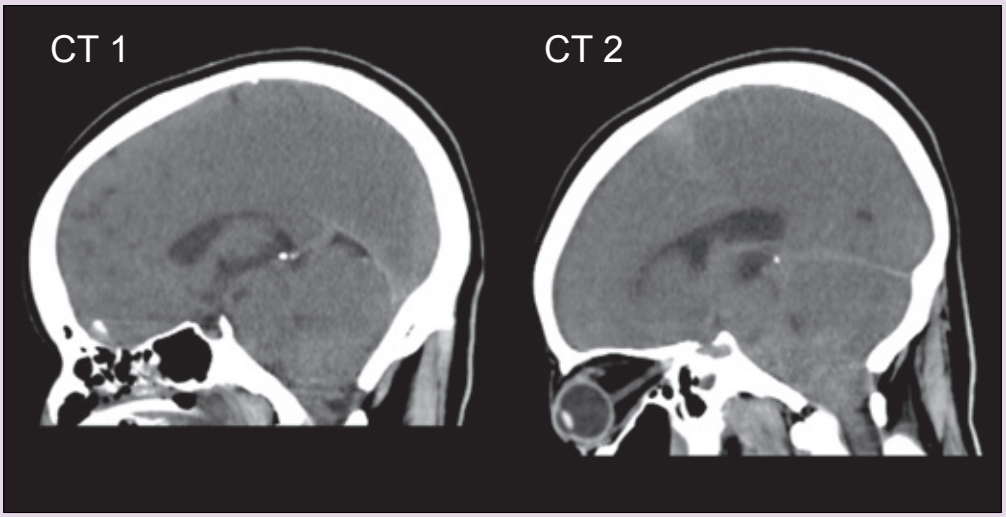

Figure 3. Midline sagittal sections from the initial and subsequent CT studies showing development of tonsillar herniation.

A BUCKET OF FLOWERS FOR THE RADIOLOGY TRAINEE

An elderly hypertensive female on warfarin presented to her local accident and emergency (A&E) department in the evening with severe headache and vomiting. The registrar in A&E suspected subarachnoid haemorrhage and requested an urgent brain CT.

The reporter was a junior radiology trainee who saw no acute abnormality on initial review. The trainee observed that the choroid plexus looked prominent and extended towards the base of the occipital horn of the lateral ventricles, especially on the right (Figure 1) and noticed hyperdensities within the fourth ventricle extending inferiorly and laterally, particularly well seen on axial (Figure 2a) and sagittal planes (Figure 2b).

The referring physician contacted the reporting radiologist several times for an urgent report as the patient was very unwell and the trainee radiologist provisionally communicated that the hyperdense findings might represent subarachnoid hemorrhage. Meanwhile, the radiology trainee contacted the on-call consultant radiologist for advice. The consultant told the trainee that the findings were in keeping with prominent choroid plexus and the changes in the fourth ventricle and foramen of Luschka were due to a normal variant called Bochdalek's flower basket rather than subarachnoid haemorrhage.[1] The trainee radiologist immediately communicated the amendment to the referring team. Understandably, the whole process was a stressful experience for the junior trainee who had not seen this normal variant before and misinterpreted it as subarachnoid haemorrhage.

REPORTER'S COMMENTS

Main causes

- Unfamiliarity with this normal anatomical variant resulted in misinterpretation of the findings as subarachnoid hemorrhage.
- The CT was reviewed on call under pressure with a clinical history compatible with a diagnosis of subarachnoid hemorrhage.

LESSONS LEARNED

The case emphasises the importance of knowledge of normal variants and the risk of misinterpreting these as abnormal.

Pressure from the referring team and clinical information should not influence the reporter to misinterpret the imaging findings to fit into the clinical scenario.

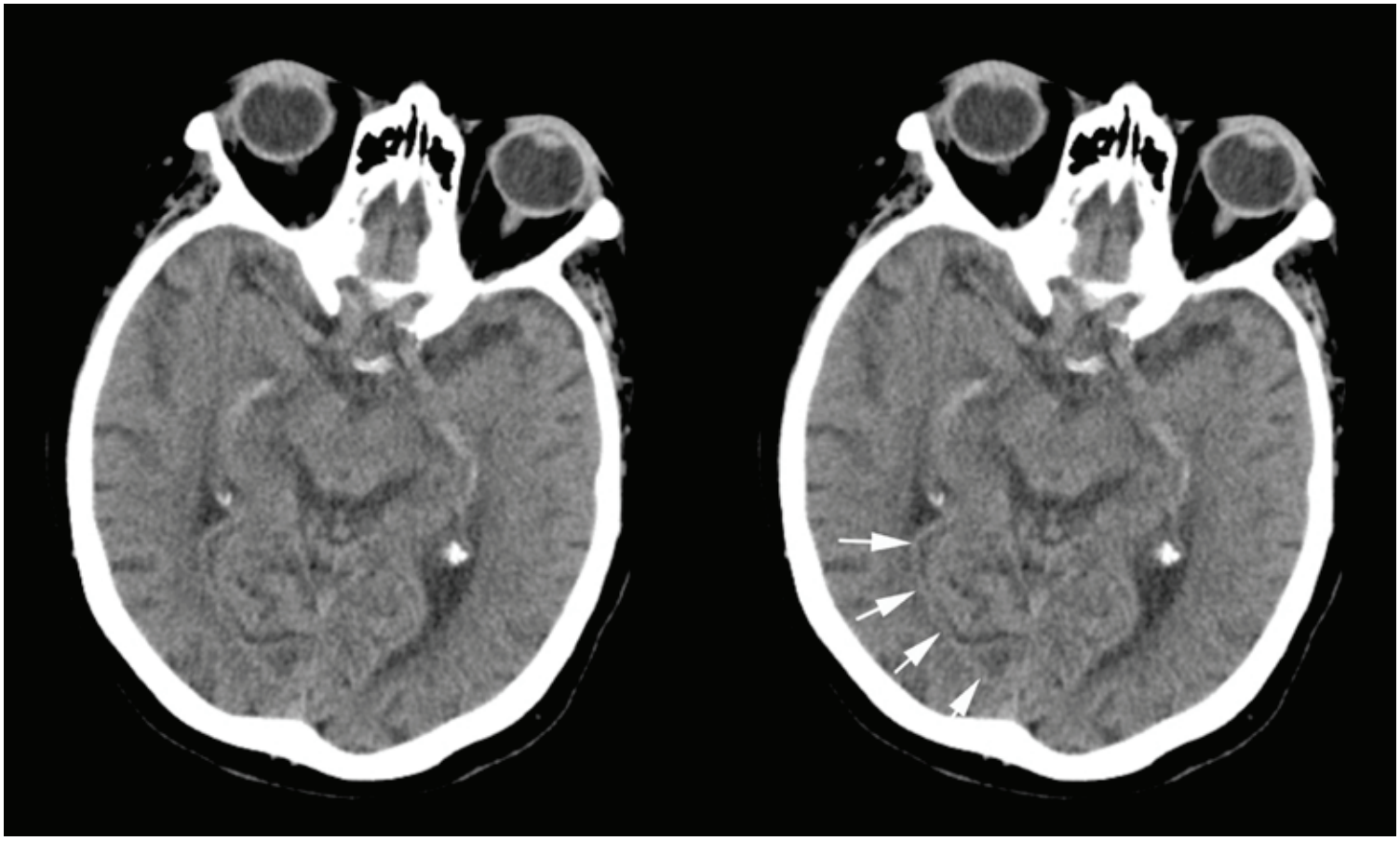

Figure 1. Unenhanced CT brain axial images through cerebral cisterns demonstrating linear high densities in the occipital horn of the lateral ventricle (white arrows).

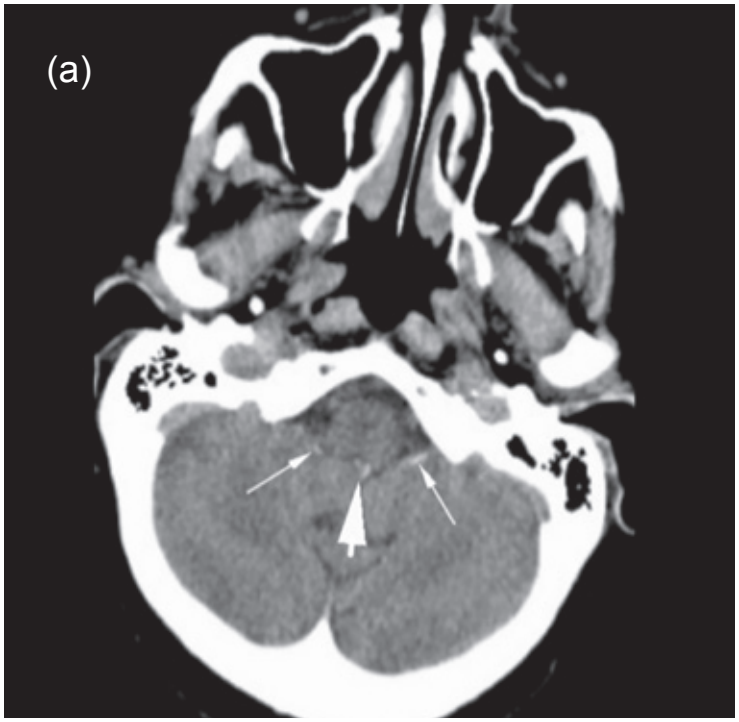

Figure 2a. Unenhanced CT brain axial image showing hyper densities within the fourth ventricle (large arrow) and in the foramen of Luschka on either side (small arrows).

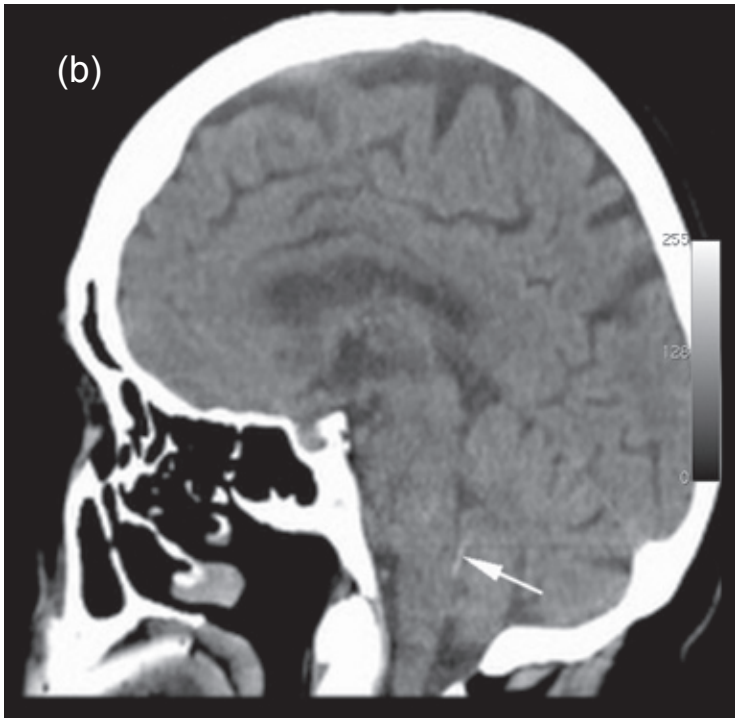

Figure 2b. Unenhanced CT brain sagittal image demonstrating hyper densities within the fourth ventricle (arrows).

Trainees should not hesitate to contact the on-call radiology consultant for advice as demonstrated in this case, which resulted in early correction of the error. Trainees should refer to the document published by The Royal College of Radiologists regarding support while on call.[2]

References

1. Horsburgh A, Kirollos RW, Massoud TF. Bochdalek's flower basket: applied neuroimaging morphometry and variants of choroid plexus in the cerebellopontine angles. *Neuroradiology* 2012; **54**(12): 1,341–1,346.
2. **www.rcr.ac.uk/sites/default/files/handover_on_call_guidance_march_2016.pdf** (last accessed 02/02/17)

READ 058

DELAYED DIAGNOSIS

An adult diabetic male in his 60s was referred for an ultrasound because of abnormal liver function tests including elevated alanine aminotransferase (ALT). The liver was reported to be fatty with a smooth outline but no focal lesion was identified.

Two years later the patient underwent CT angiography (CTA) of abdomen and pelvis and lower limbs due to ischaemia of his right leg at which time a 3 cm hypervascular lesion within segment VIII of the liver was seen (Figure 1a). The reporting radiologist recommended further characterisation but no further imaging was organised by the referring team.

Three years later he underwent CT pulmonary angiography for acute dyspnoea. This was negative for pulmonary embolus but demonstrated a pulmonary nodule reported as suspicious for a lung cancer. In hindsight, reflux of contrast into the hepatic veins appears to course around an isodense lesion (Figure 1b) occupying the position of the previously seen hypervascular hepatic lesion on the previous CTA. The previous CT study was not reviewed when the computed tomography pulmonary angiography (CTPA) was reported.

Biopsy of the lung nodule revealed adenocarcinoma and a diagnosis of primary bronchogenic carcinoma was made.

A staging positron emission tomography (PET) CT showed increased metabolic activity in the lung nodule but also in segment VIII of the liver without an obvious unenhanced CT abnormality.

Magnetic resonance imaging (MRI) liver was contraindicated due to the incompatibility of a permanent cardiac pacemaker.

Repeat liver ultrasound targeted at examination of the liver reported that the liver was normal in appearance and echotexture with no focal hepatic lesions.

Contrast-enhanced ultrasound of the liver showed no focal liver lesion.

A restaging CT in portal venous phase revealed an ill-defined mass in segment VIII (Figure 1c) of the liver. Tumour thrombus was observed in the right portal vein.

Eventual diagnosis of hepatocellular carcinoma (HCC) with portal vein invasion and pulmonary metastasis was made three years after the liver lesion was first detected and after numerous different radiological examinations.

REPORTER'S COMMENTS

Main causes

- The hypervascular liver lesion on the initial CT was not followed up with complete characterisation despite the radiological recommendation.
- Although the CTPA identified a lung malignancy, the previous CT was not reviewed.
- The liver lesion could not be seen on normal or contrast-enhanced ultrasound.
- A cardiac pacemaker precluded MRI characterisation of the liver lesion.
- The patient had no radiological features to suggest cirrhosis

LESSONS LEARNED

Radiological recommendations for further investigation may not happen for a variety of reasons. The reporter should ensure that important unexpected findings are flagged to the referring team in accordance with local protocol.

The diagnosis of HCC can be challenging but might have been made earlier if the original CTA had been reviewed when the CTPA was reported.[1,2] Due to increasing numbers of CTs being performed, reporters are under pressure to report faster and this may lead to overlooking the review of previous imaging, particularly if the past images are not obviously related to the current clinical problem.

Although hypervascular liver lesions can be benign, HCC should always be considered even if the liver is not obviously cirrhotic.

In a patient who is unable to undergo MRI, a triple phase CT can be helpful in demonstrating the hypervascular nature of a lesion and portal vein thrombus which in this case strongly favoured the diagnosis of HCC.

References

1. Thng CH, Kuo YT. Hepatocellular carcinoma – issues in imaging. *Cancer Imaging* 2004; **4**(2): 174–180.
2. Choi JY, Lee JM, Sirlin CB. CT and MR imaging diagnosis and staging of hepatocellular carcinoma: part 1. Development, growth and spread: key pathologic and imaging aspects. *Radiology* 2014; **272**(3): 635–654.

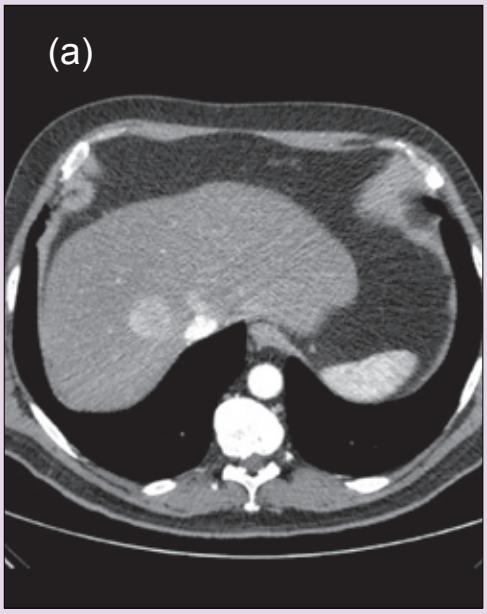

Figure 1a. CT angiogram demonstrates a hypervascular lesion in segment VIII of the liver.

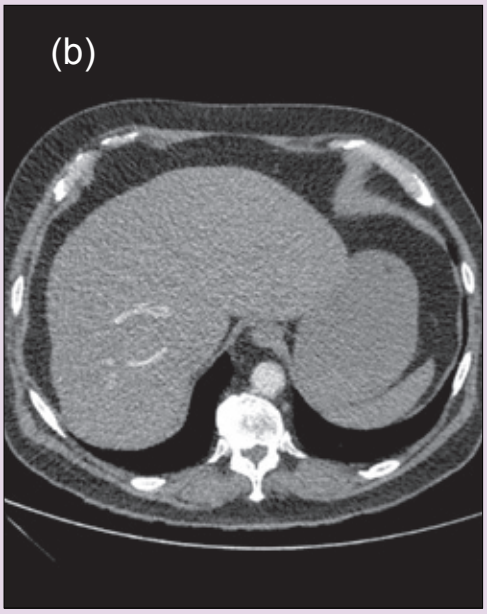

Figure 1b. Reflux of contrast agent into the inferior vena cava on CTPA appears to course around an isodense lesion in segment VIII of the liver.

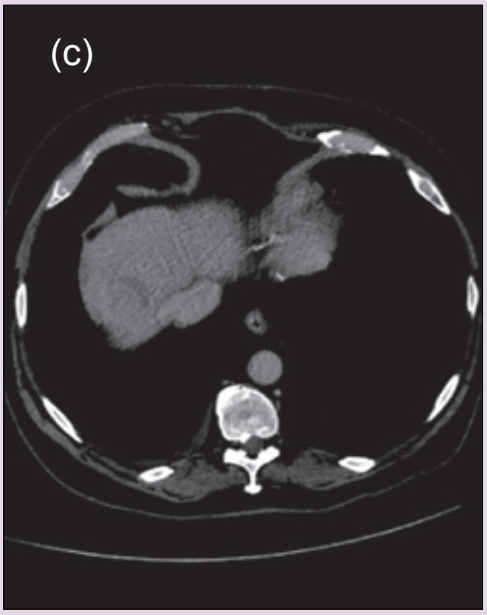

Figure 1c. Portal venous phase imaging of liver lesion.

READ 059

A COMPLICATION OF THERAPY

A child with acute lymphoblastic leukaemia (ALL) in consolidation phase presented with confusion and non-focal neurology. She had received methotrexate six days before the scan. The patient was referred for a MRI brain, initially reported as normal. The imaging was referred to a specialist paediatric neuroradiology centre for a second opinion.

On further review of the MRI, bilateral signal abnormality was noted in the deep cerebral white matter and splenium of the corpus callosum (Figures 1 and 2) with associated restricted diffusion (Figure 3). Methotrexate toxicity was diagnosed.

REPORTER'S COMMENTS

Main causes

- Perceptual error:
  - The signal abnormality on the T2-weighted, T1-weighted and diffusion-weighted imaging (DWI) was not appreciated
  - A symmetrical abnormality is easy to overlook.
- Unfamiliarity with the neurological complications of methotrexate therapy.

LESSONS LEARNED

When interpreting imaging studies, it is important always to consider the clinical context including a thorough drug history.

Be vigilant to check all MRI sequences for abnormalities. In this case the diffusion-weighted sequence showed the abnormality most clearly.

When symmetrical abnormalities are present consider systemic and metabolic causes. Other causes of splenial restricted diffusion include hypoglycaemia, electrolyte abnormalities, seizure activity, diffuse axonal injury (DAI, see READ 057), Marchiafava-Bignami disease, demyelination and acute demyelinating encephalomyelitis (ADEM). The latter should especially be considered in children with other white matter abnormalities.

Central nervous system complications of leukaemia and its treatment are well-documented causes of neurotoxicity. Methotrexate, in particular, is a major cause of neurotoxicity in children undergoing ALL treatment with a reported incidence of between 9–53%.[1] The associated white matter damage (leukoencephalopathy) causes T2/FLAIR dependent signal abnormality, typically in the periventricular white matter. DWI changes are often seen and may be found earlier in the neurotoxic process. Early recognition is key to alert oncologists and allow therapy to be altered and, despite the presence of imaging changes, patients may recover without persisting neurological deficit. For further information on this subject we direct our READers to an excellent pictorial review of medication neurotoxicity in children.[2]

References

1. Fisher MJ, Khademian ZP, Simon EM, Zimmerman RA, Bilaniuk LT. Diffusion-weighted MR imaging of early methotrexate-related neurotoxicity in children. *Am J Neuroradiol* 2005; **26**(7): 1,686–1,689.

2. Iyer RS, Chaturvedi A, Pruthi S, Khanna PC, Ishak GE. Medication neurotoxicity in children. *Pediatr Radiol 2011*; **41**(11): 1,455–1,464.

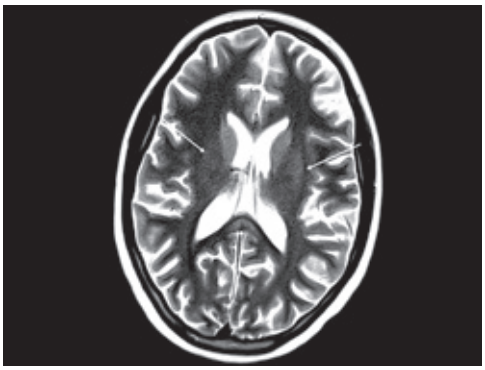

Figure 1. Axial T2-weighted image shows patchy but symmetrical T2 hyperintensity involving the deep white matter and splenium of the corpus callosum.

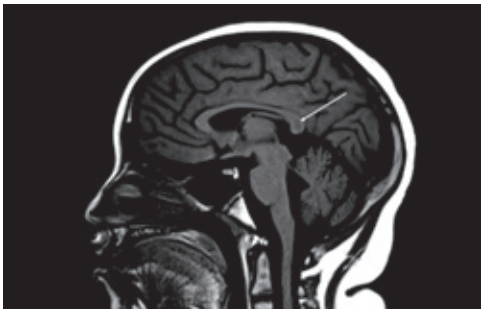

Figure 2. Sagittal T1-weighted image demonstrates corresponding splenial hypointensity.

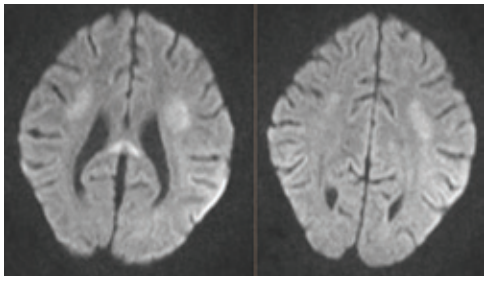

Figure 3. Restricted diffusion (apparent diffusion coefficient [ADC] maps not shown but were correspondingly dark) is noted in the deep white matter of both cerebral hemispheres and within the splenium of the corpus callosum. Overall these features are consistent with methotrexate toxicity.

Continue READing

READ supports our capability to learn and as such is now an important part of a radiologists commitment to improving the safety of patients in England and across the UK.[1] It has to be driven by radiologists for radiologists. The local learning from discrepancies meeting is a rich source of learning that can also be shared with the wider radiology community. Every case discussed should be considered for submission to READ and don't forget, each story submitted will attract continuing professional development (CPD) points.[2]

1. National Advisory Group on the Safety of Patients in England. *A promise to learn – a commitment to act*. London: Williams Lea, 2013.

2. The Royal College of Radiologists. *Continuing professional development (CPD) scheme, third edition*. London: The Royal College of Radiologists, 2014.

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