Case 17728

Eurorad

Atypical amoebic liver abscess mimicking liver malignancy – the importance of seeing the full picture

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Section: Abdominal imaging
Area of Interest: Abdomen Gastrointestinal tract Liver
Imaging Technique: CT
Imaging Technique: MR
Special Focus: Abscess Infection Tropical diseases
Case Type: Clinical Cases
Authors: Eugene Cho1, Benjamin P T Loveday2,3,4, Benjamin Teh5,6, Owen W J Prall6, Hyun Soo Ko1,7
Patient: 62 years, female

Clinical History:

A 62-year-old female presented to the GP with several weeks of fever, diarrhoea, vague right-upper-quadrant pain and raised inflammatory markers (CRP, WCC). Given the fairly non-specific symptoms, a CT was performed demonstrating a liver and osseous lesion that were deemed suspicious. This resulted in referral to a quaternary cancer centre.

Imaging Findings:

CT showed a hypoenhancing 28mm segment 8 liver lesion with an ill-defined perilesional hyperenhancement on arterial phase. It was heterogeneously hypoenhancing on portal-venous phase and demonstrated mild hyperenhancement with central washout on delayed phase. L4 vertebral body contained a well-defined lytic lesion (Figure 1).

Multidisciplinary meeting 2 weeks post CT deemed imaging findings indeterminate, prompting further MRI and laboratory work-up. Liver function tests and tumour markers were normal. MRI liver with hepato-specific contrast (Gd-EOB-DTPA) was performed 3 weeks after CT. The lesion was T1-hypointense with a T2-hyperintense rim, showed no diffusion-restriction, similar contrast enhancement pattern to CT, and no hepatobiliary contrast uptake (Figure 2). L4 lesion had typical benign features (Figure 3).

Additionally, subtle caecal wall thickening was noted on CT and MRI (not reported on prior CT) (Figure 4). Subsequent colonoscopy biopsy confirmed amoebic colitis (Figure 5) resulting in the liver lesion diagnosed as atypical amoebic liver abscess (ALA).

Discussion:

Amoebiasis is a faecal-orally transmitted parasitic E. Histolytica infection that commonly affects travellers from (sub)tropical regions and people subject to poor sanitation, malnutrition, and immunosuppression [1-3]. Developed East-Asian countries have also seen an emergence in coinfection with HIV-1 due to oral-anal sexual contact [4].

E. Histolytica infection most commonly causes amoebic colitis, which usually occurs within three weeks of exposure. Diagnosis is made by stool microscopy, antigen testing, or PCR [2]. Radiological findings show typical inflammatory colonic wall changes (e.g., thickening, ulcerative lesions) and most affected sites are the caecum and rectum with sparing of the ileum [5].

ALA is the most common extraintestinal manifestation of amoebiasis. Usual symptoms are as described in our case; however, they commonly evolve within 20-weeks post-exposure [6]. On US, ALA may be seen as a rounded, well-defined hypoechoic mass. It can show thin peripheral calcification on US and CT. ALA CT findings demonstrate features similar to a bacterial liver abscess with a predominant hypoenhancing mass demonstrating mild extralesional rim enhancement, however, a mild peripheral intralesional hyperenhancement on delayed phase is less common in bacterial abscess. MRI shows similar enhancement pattern to CT with additional information of no restricted diffusion, and no Gd-EOB-DTPA contrast uptake on hepatobiliary phase [7].

In our case, ALA was not considered as differential diagnosis since symptoms were of extreme late onset 4-years post travel to India, and travel history not obtained until after final diagnosis was made. Initial CT report deemed the liver and L4 vertebral body lesion as suspicious. Multidisciplinary management including radiology sub-specialist reporting at our cancer centre lead to the diagnosis of amoebic colitis with ALA. The patient underwent successful antimicrobial therapy (metronidazole, paromomycin) with resolution of colonic findings [8]. Follow-up 6-week post-treatment US was unable to visualise the high subcapsular ALA. Therefore, serial MRIs were performed that showed minimal residual scarring at 18 and 24 months (Figure 6) after which the patient was discharged from further surveillance.

Any newly detected liver lesion requires careful evaluation of prior medical, clinical and travel history as well as laboratory and imaging findings to guide further management [9]. Complex cases should be reported by a sub-specialised radiologist and undergo multidisciplinary discussion to ensure optimal outcomes [10]. Liver tissue sampling may be required during diagnostic workup [11], however, a percutaneous biopsy of uncomplicated ALA (lesion <10cm) should be avoided due to higher risk of liver rupture, seeding, and peritonitis [12].

Differential Diagnosis List: Amoebic liver abscess and amoebic colitis, Bacterial Liver Abscess , Liver Metastasis , Hepatocellular Carcinoma , Cholangiocarcinoma , Focal Hepatitis , Liver Haemangioma

Final Diagnosis: Amoebic liver abscess and amoebic colitis

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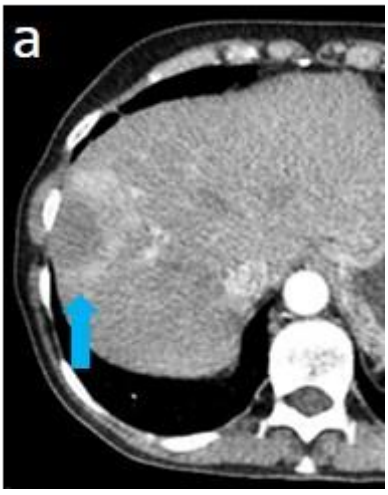
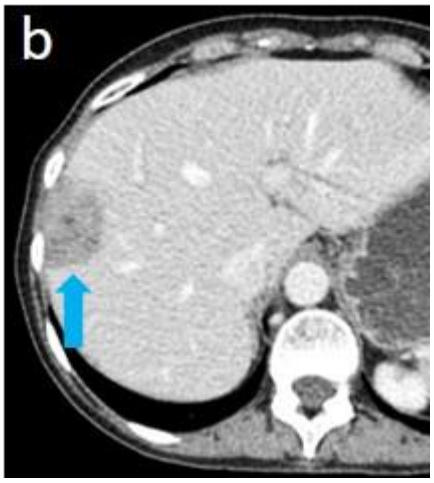
Figure 1

a



Description: Multi-phasic CT in arterial, portal venous and delayed phase shows a 28mm lesion within subcapsular liver segment 8 in axial and coronal reformats. The lesion demonstrates peripheral extralesional arterial hyperenhancement (a, e), heterogenous hypoenhancement in portal-venous phase (b, f) and a heterogenous appearance with peripheral hyperenhancement in delayed phase (cg). Bone axial and coronal reformats demonstrate a well defined lytic lesion without cortical breach within the right L4 vertebral body (d, h) **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021

b



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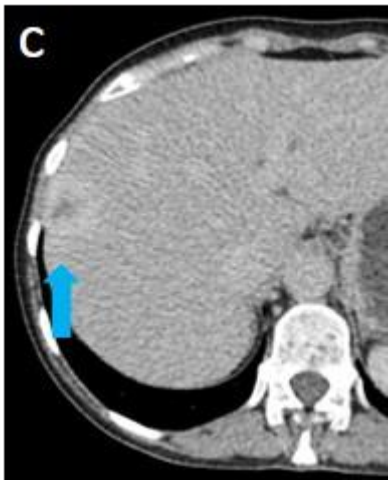

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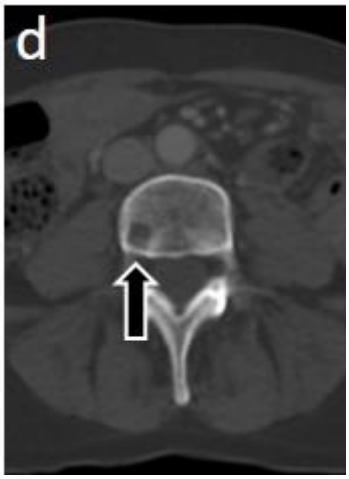

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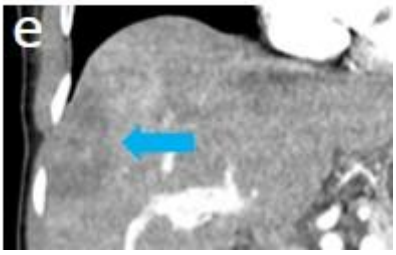
e



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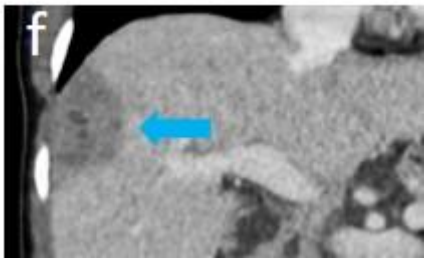

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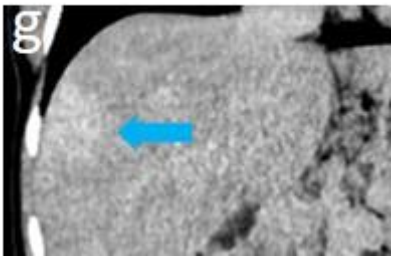
g



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h

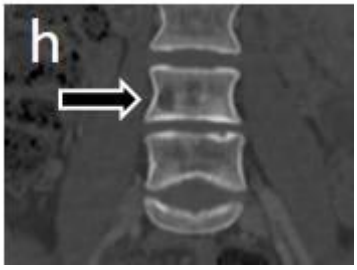

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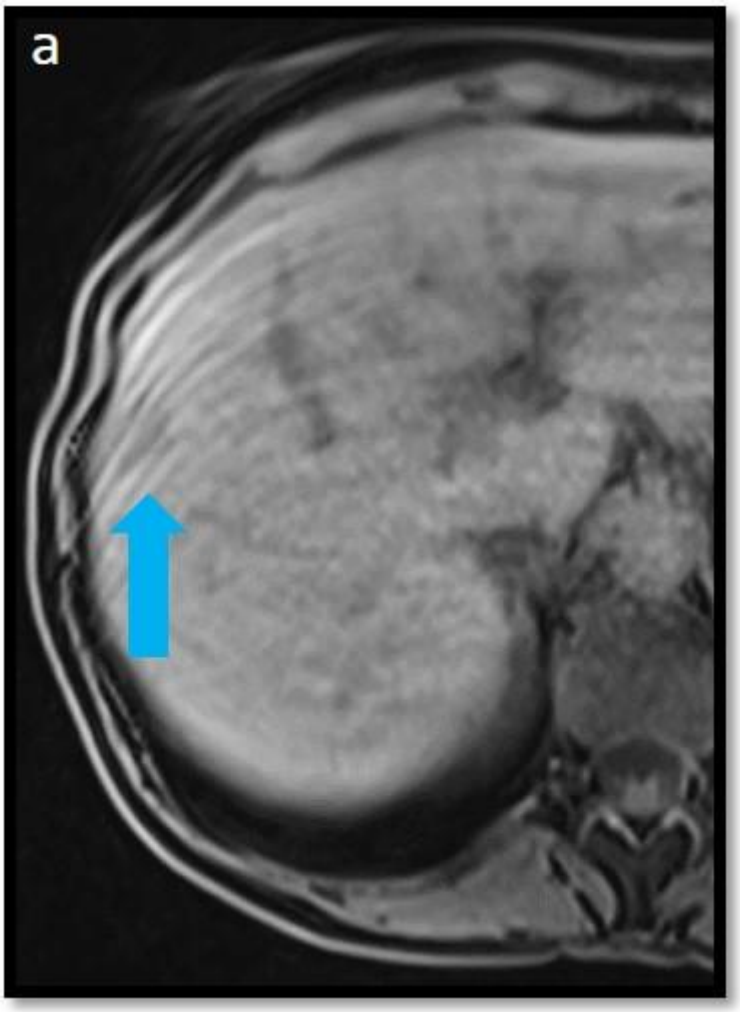
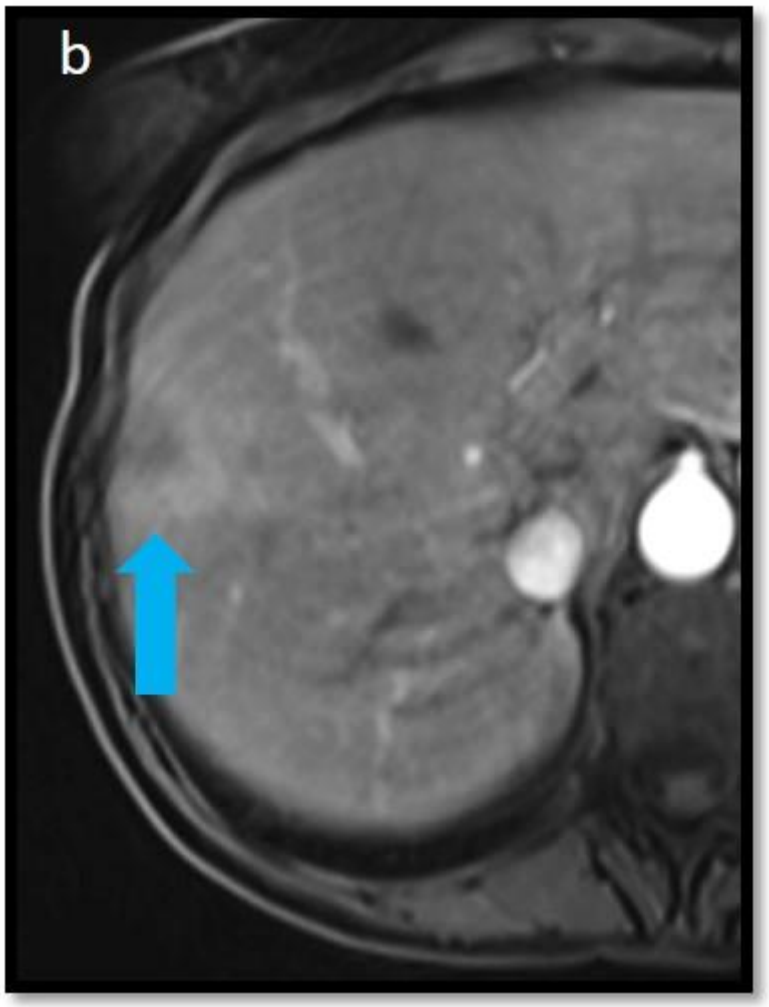
Figure 2

a



Description: The lesion in liver segment 8 (blue arrows) is midly hypointense on T1 fatsat non contrast (a), showing perilesional hyperenhancement and minimal intralesional enhancement on arterial phase (b) which increases in portal venous phase (c) and no definite retention of contrast with mild perilesional hypoenhancement on delayed hepato-biliary phase (d). On T2 and DWI the lesion is heterogenous mildly hyperintense with an iso to hypointense centre (e, f) and shows a minimal peripheral hypointense rim on ADC suggestive for mild restricted diffusion with no restricted diffusion (“shine-through”) within its centre (g) **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021

b



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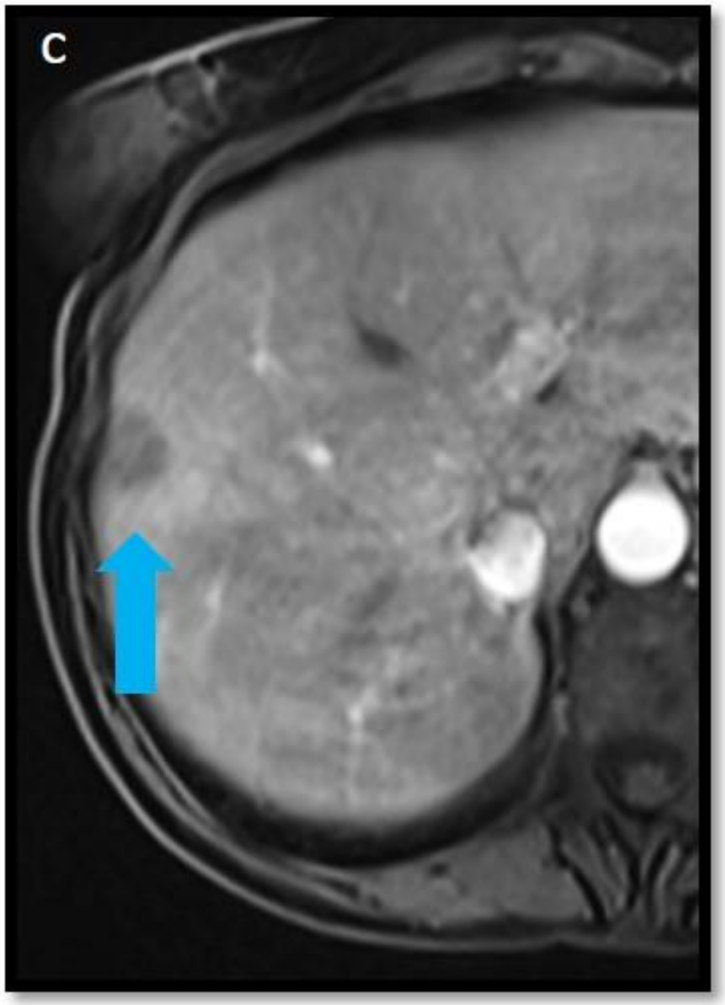

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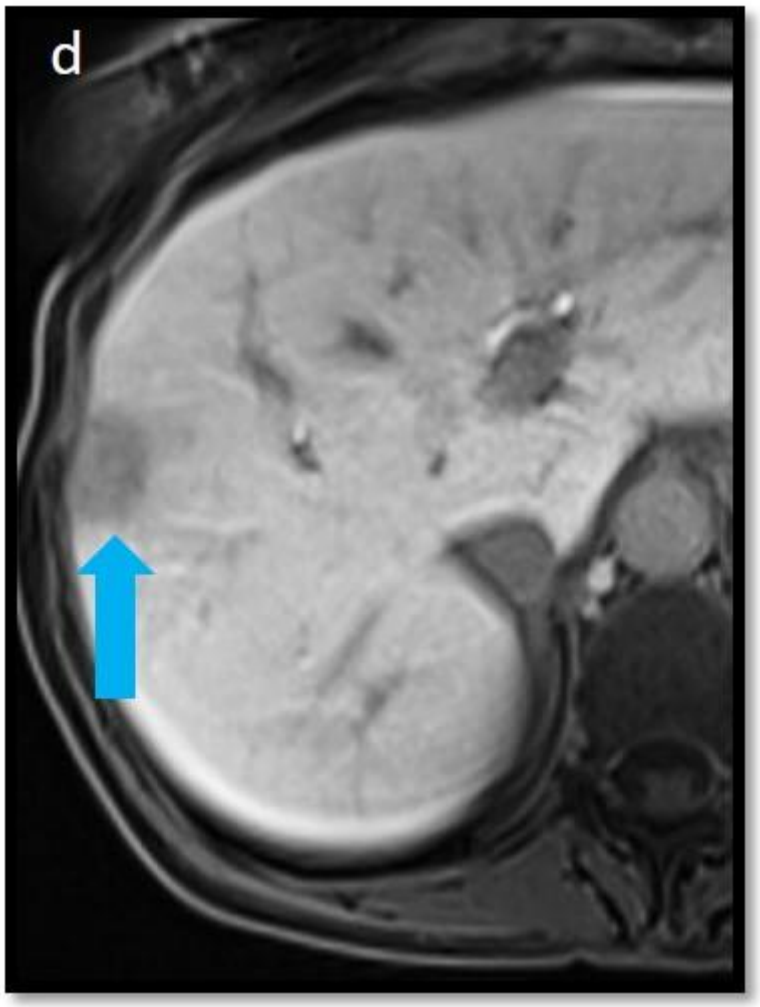

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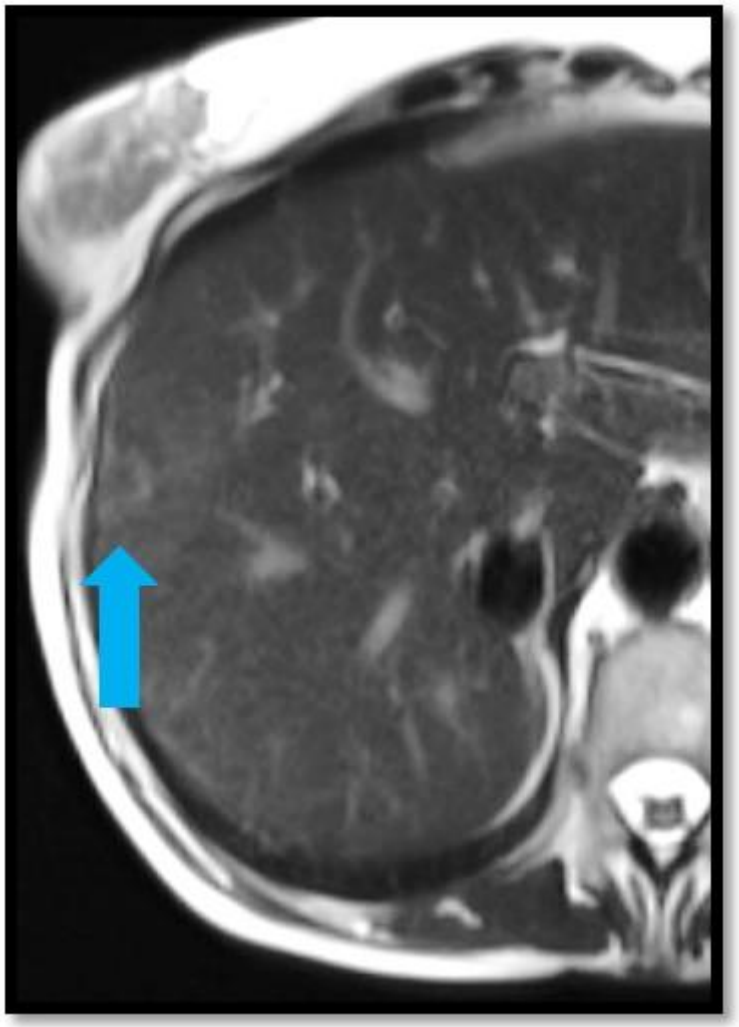

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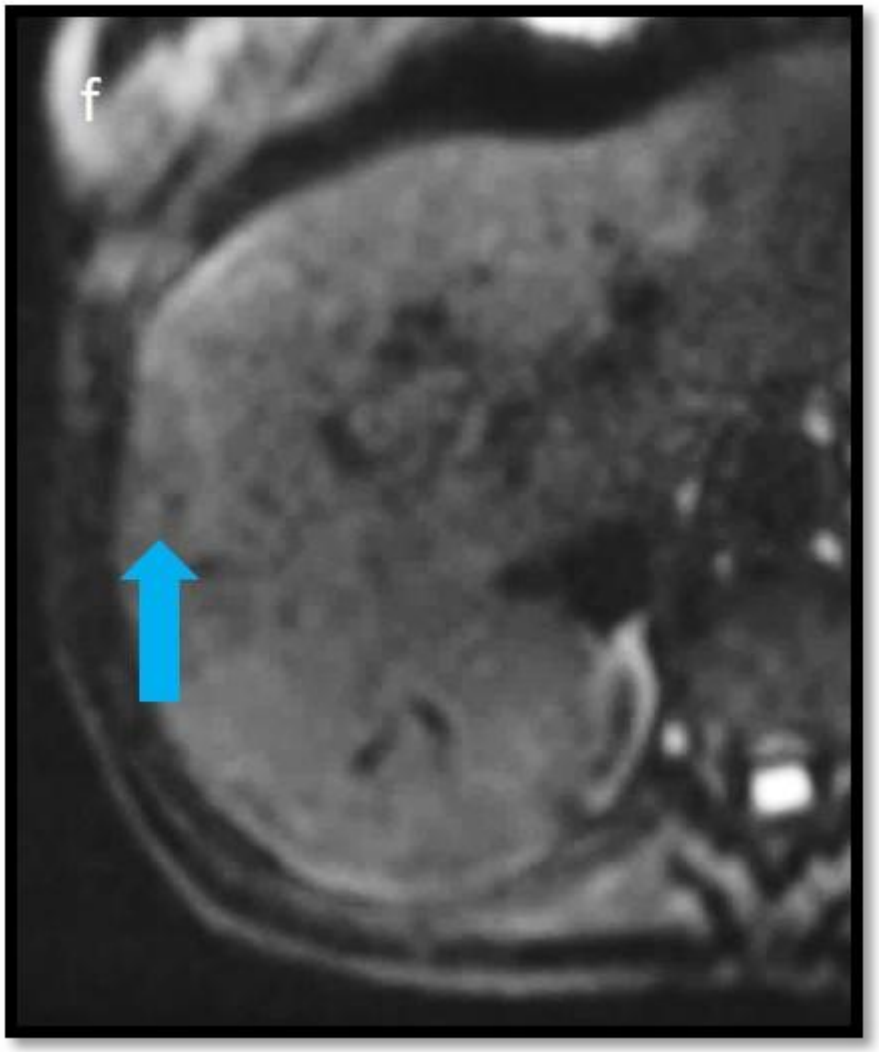

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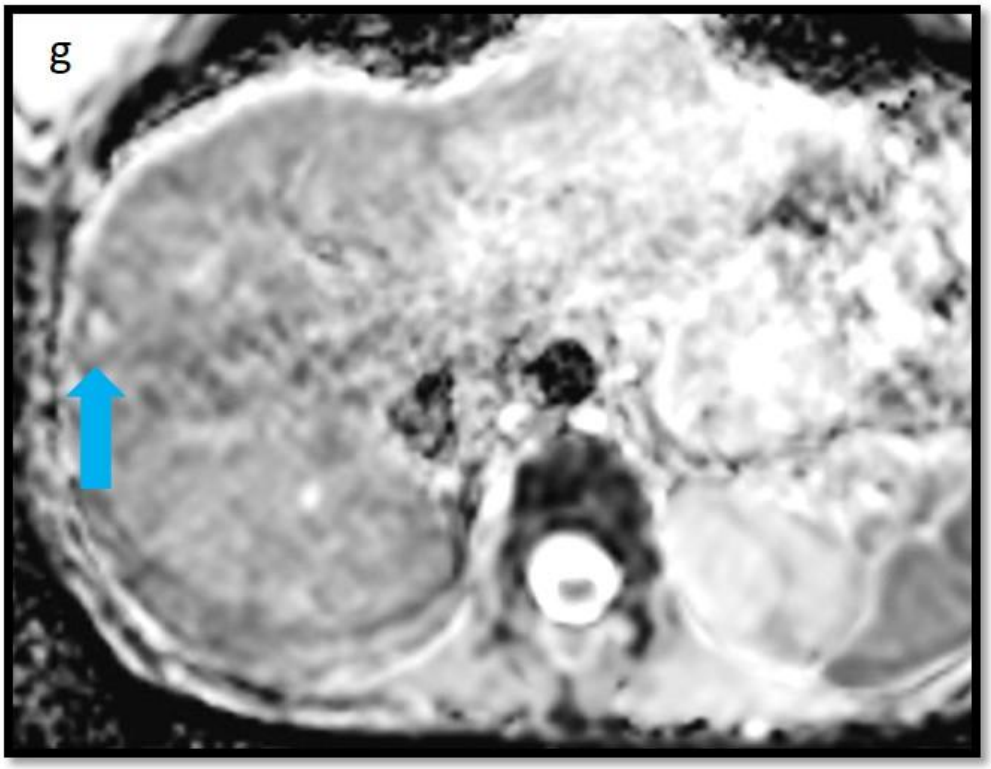

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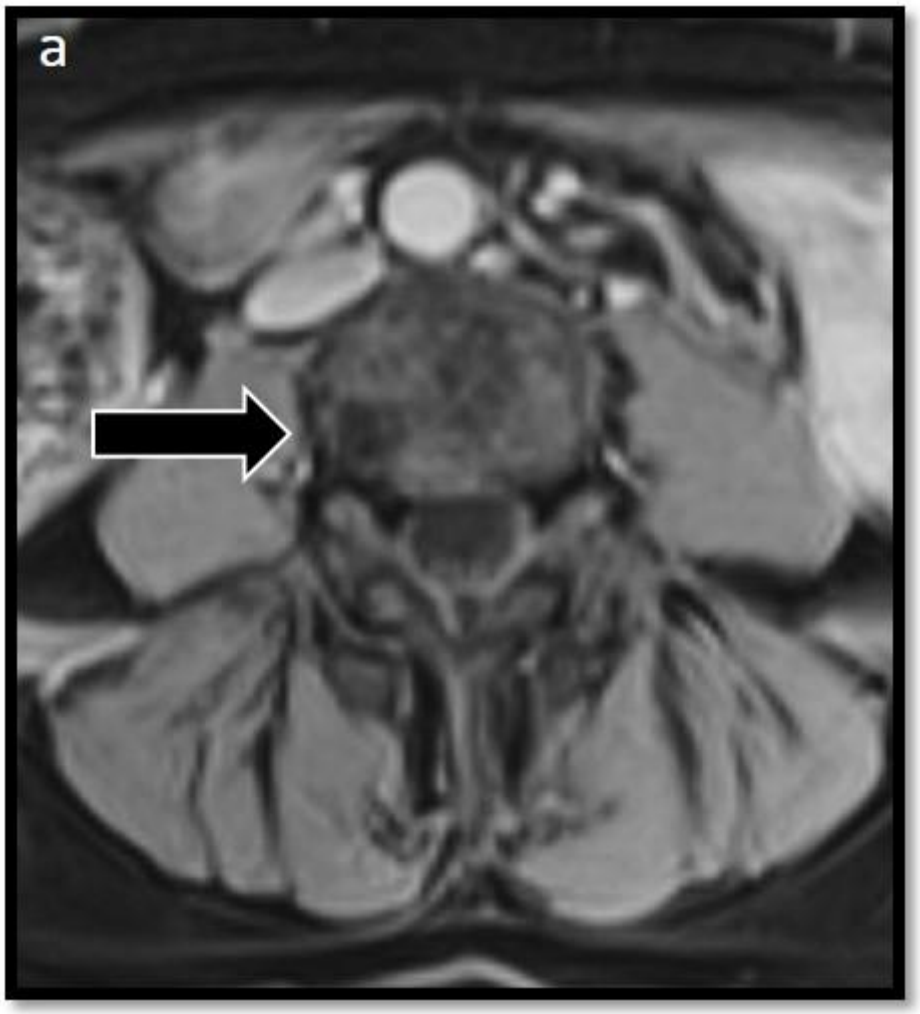
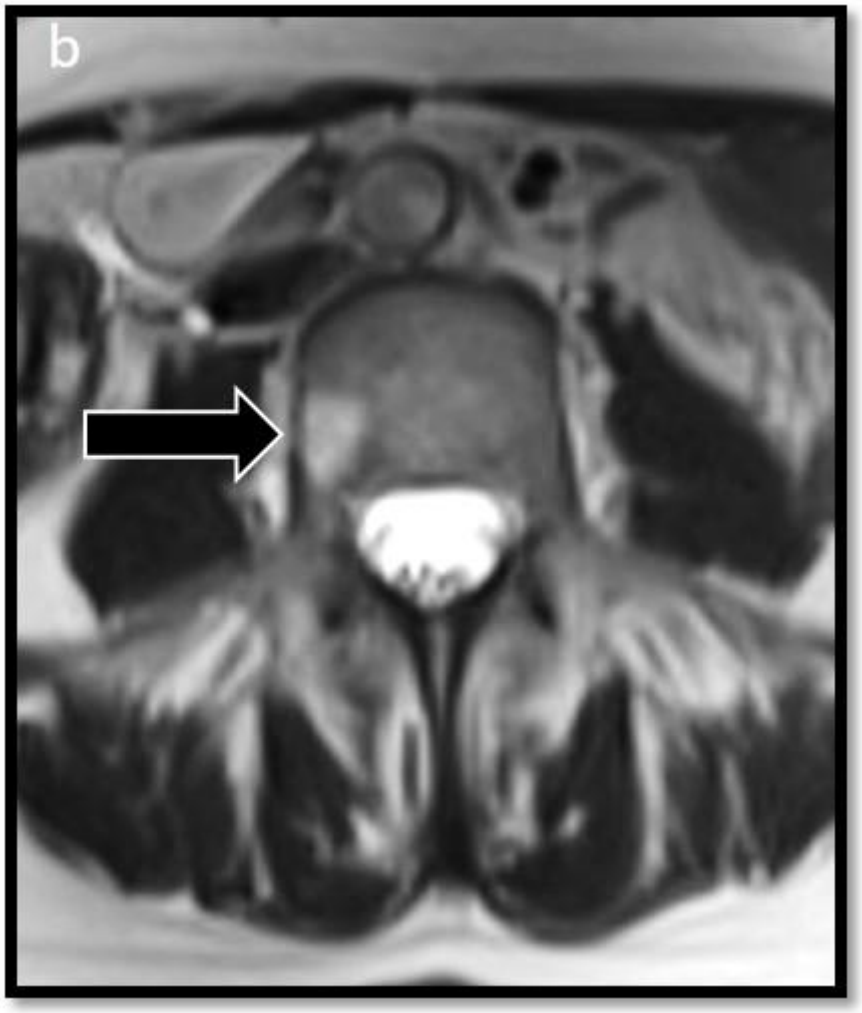
Figure 3

a



Description: The L4 right vertebral body lesion shows typical features of a benign vetrebral haemangioma with T1 hypointensity (a), T2 and DWI hyperintensity (b,c) and shine-through on ADC map (d) **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021

b



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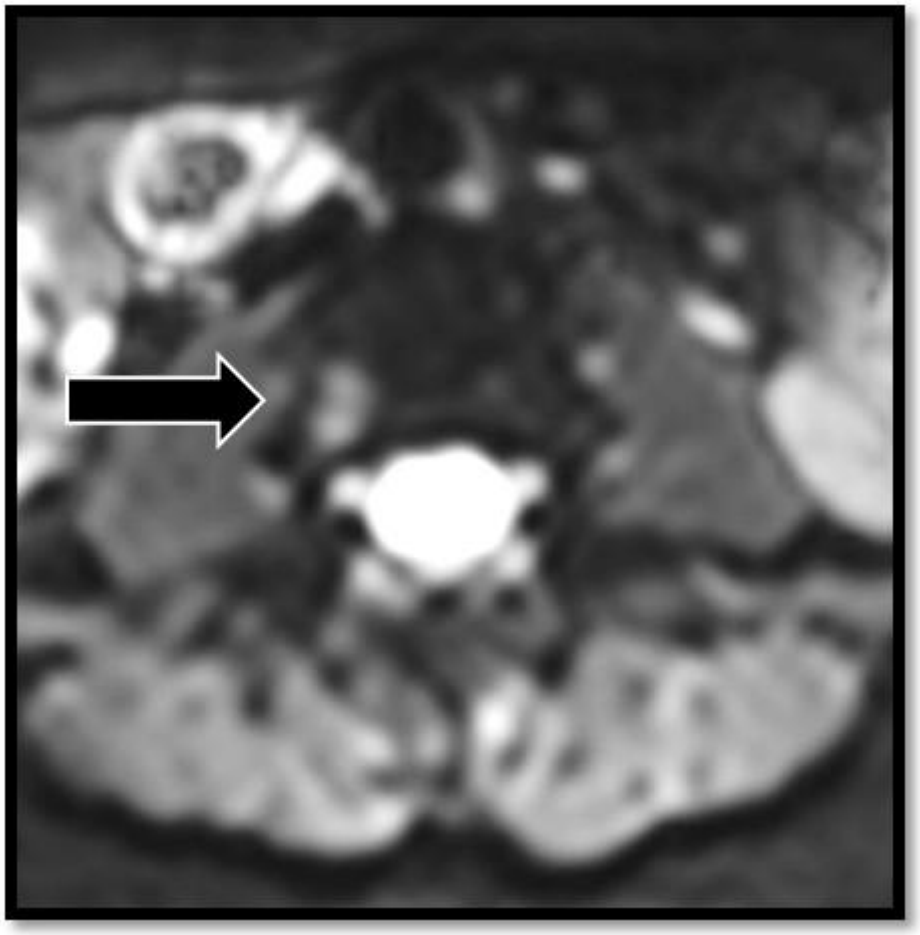
c



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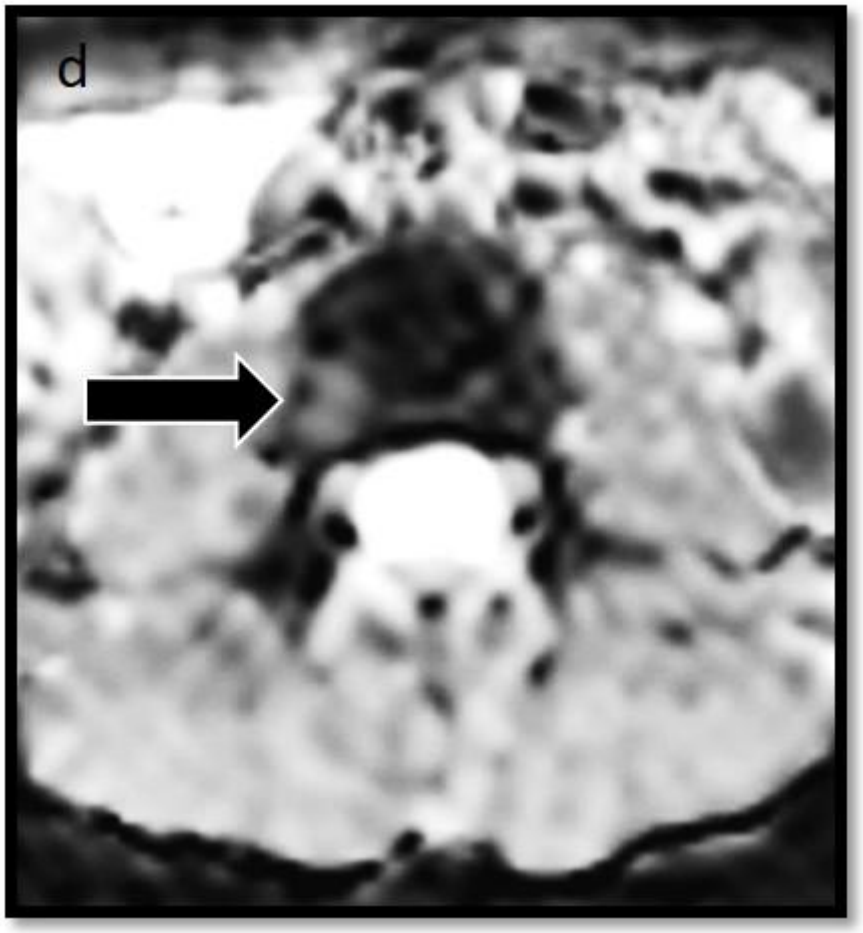

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Figure 4

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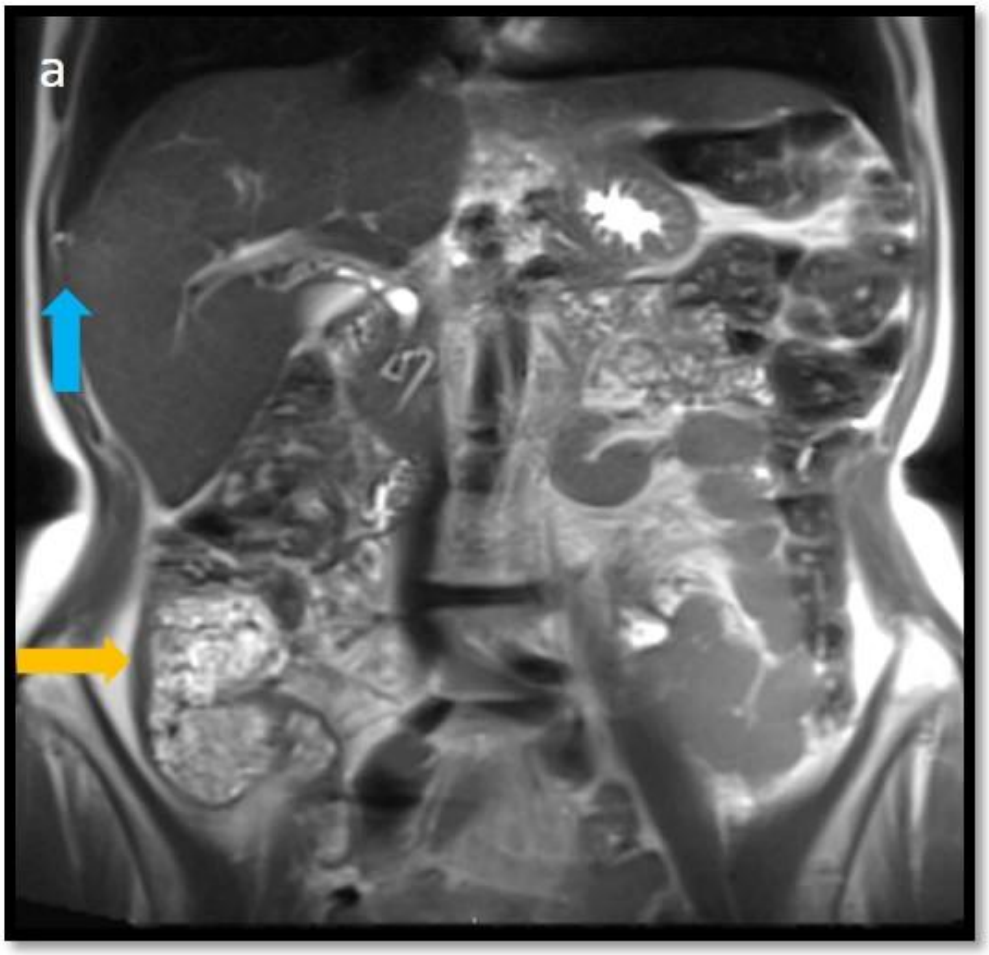
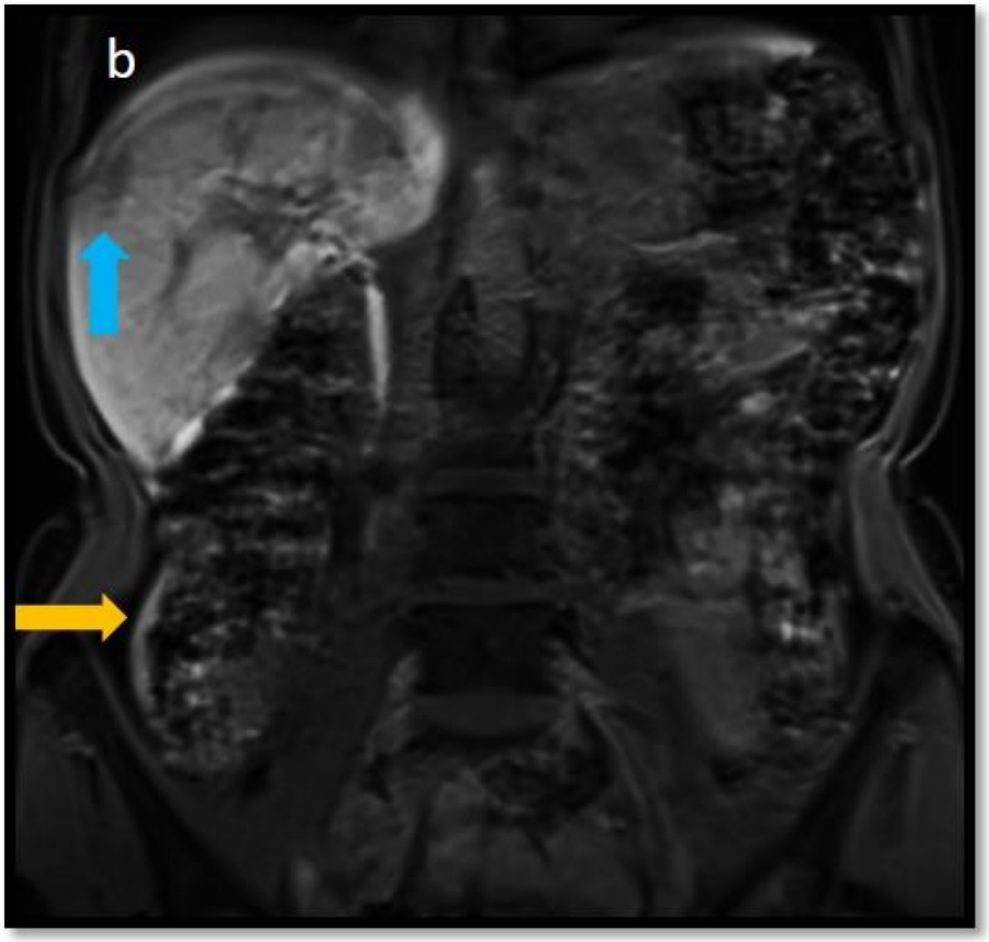


Description: Coronal T2 and T1 post contrast reformats (a, b) show minor changes within the periphery of the liver lesion (blue arrows) and mildy abnormal focal thickening of the caecum (orange arrows) **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021

b



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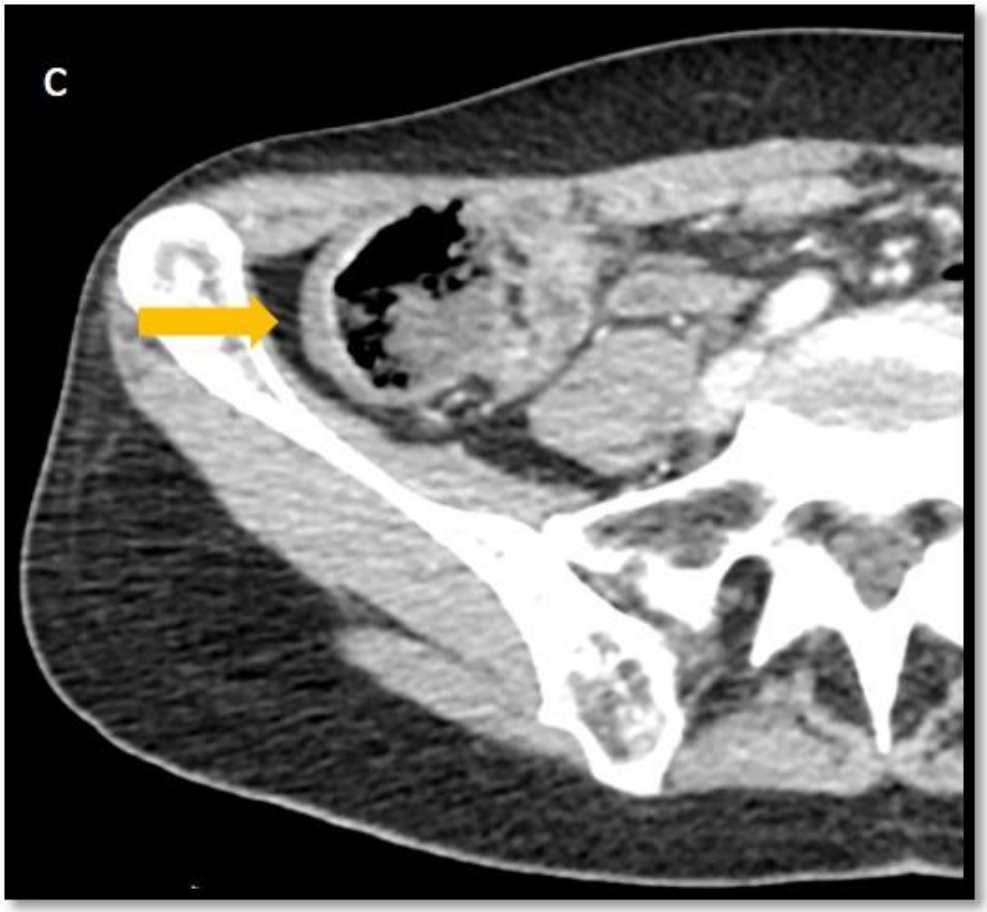

Description: When compared to initial CT (performed 3 weeks prior to MRI), the caecal findings are subtle but persistent (c, d) **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021

d

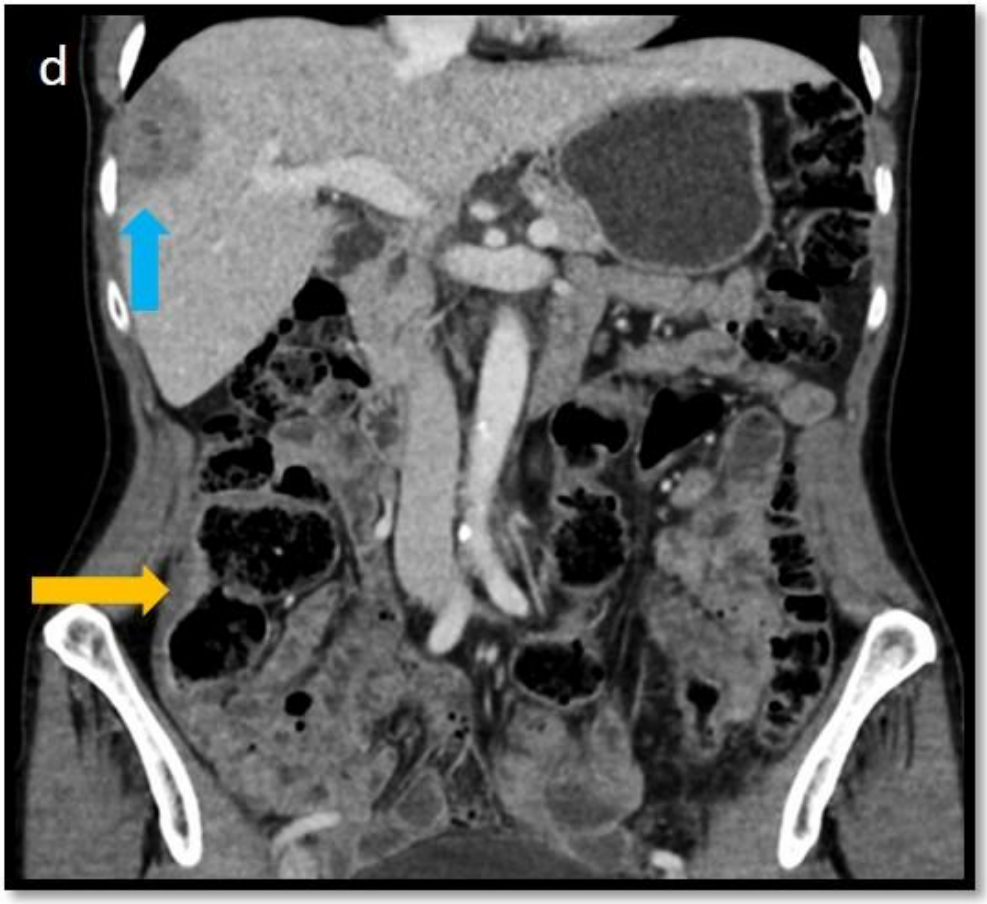

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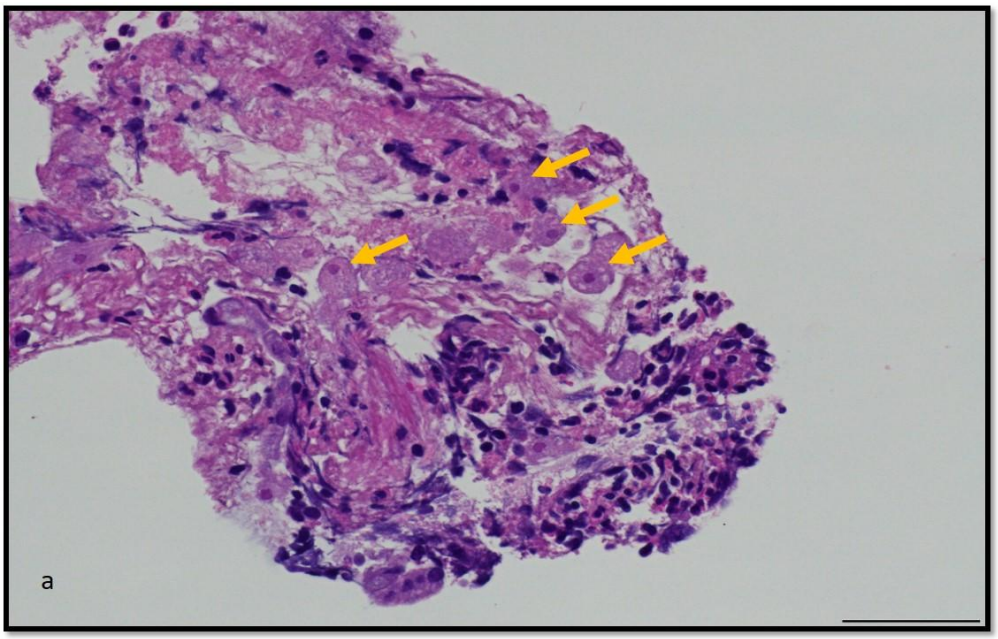
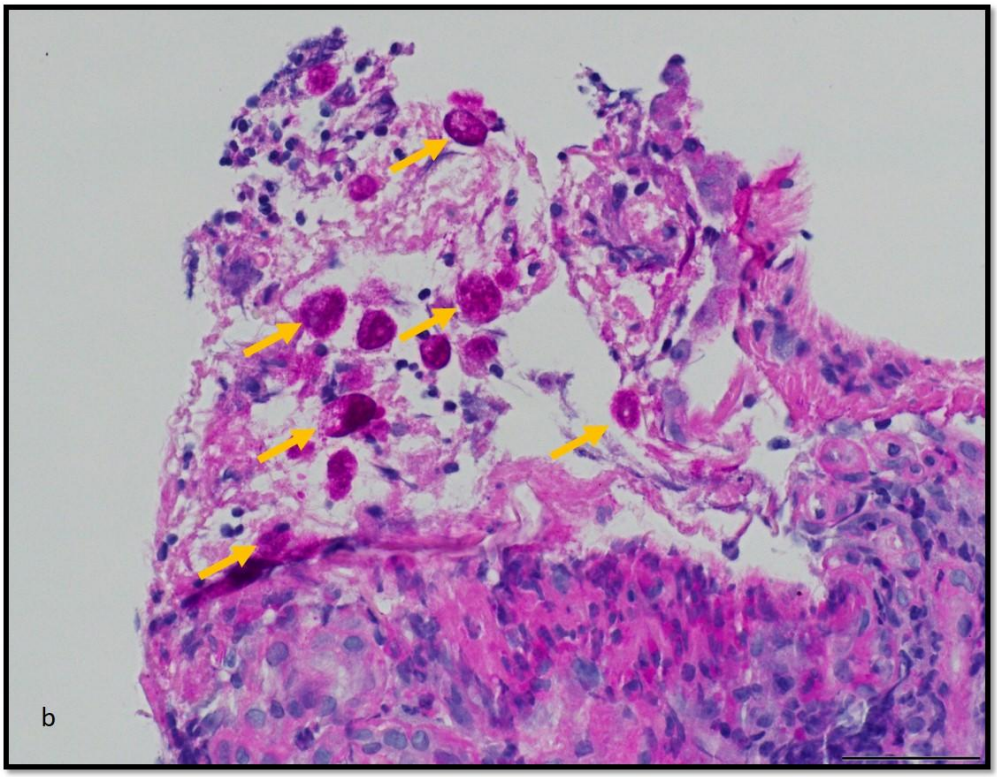
Figure 5

a



Description: Haematoxylin and eosin stain of caecal biopsy showed focally ulceration and round organisms with prominent round eosinophilic nuclei within superficial fibrin debris **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021

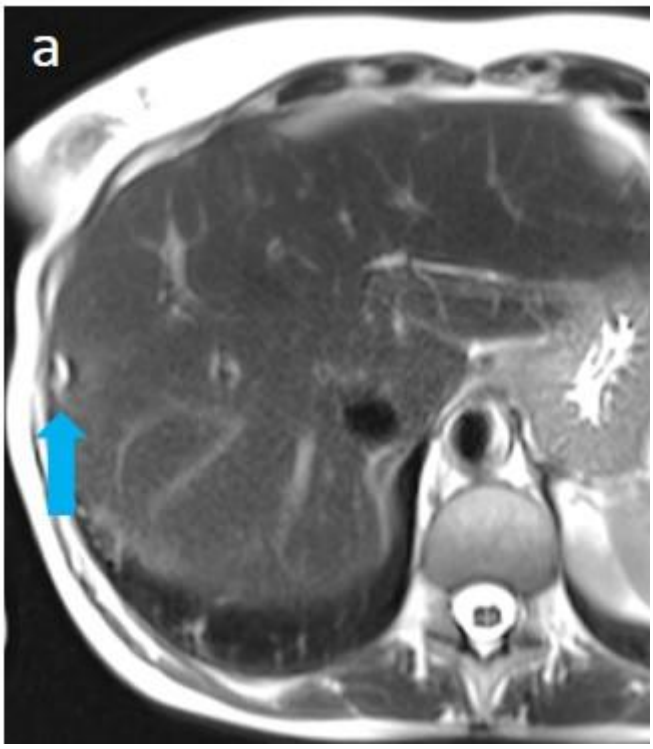
b



Description: The organisms stain strongly with periodic acid Schiff (PAS) stain. An immunostain for a macrophage marker (CD163, not shown) was negative. Scale bar (bottom right) = 50 microns**Origin:**

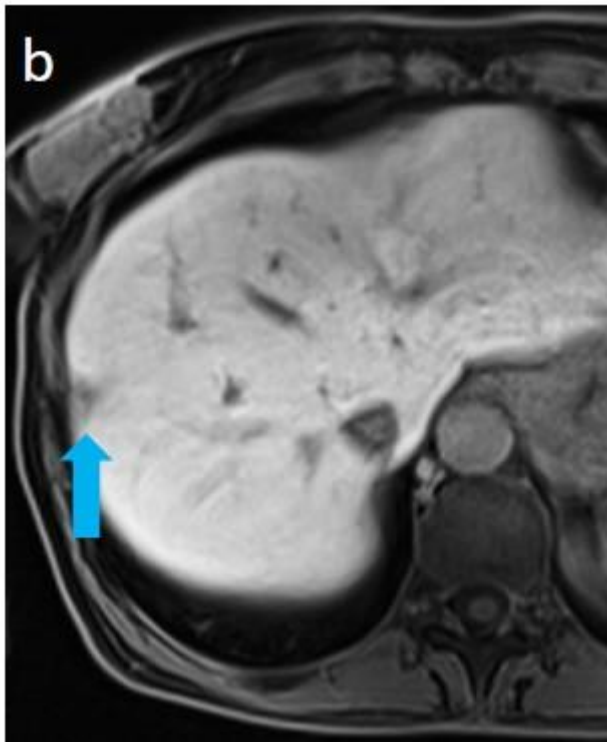
Figure 6

a



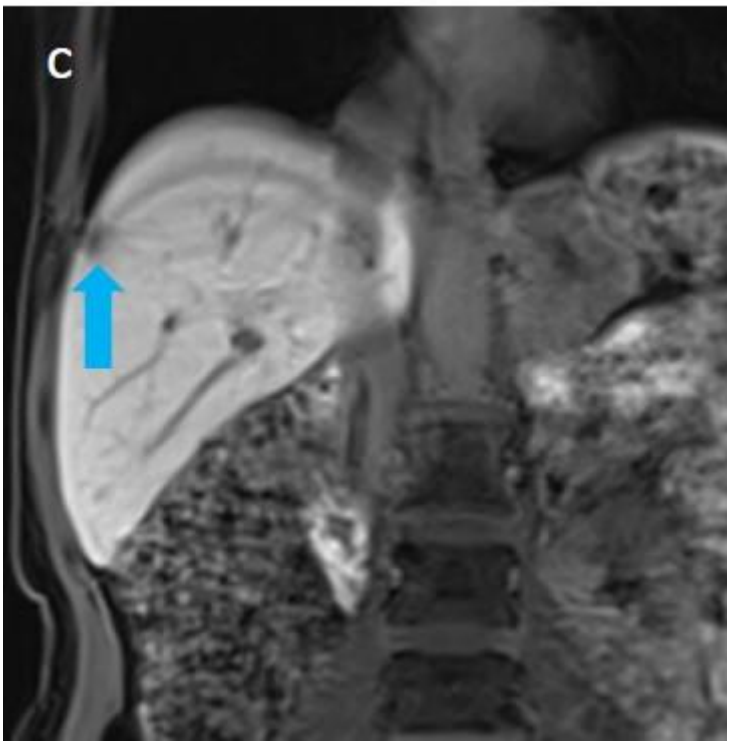
Description: MRI liver with hepatospecific contrast (Gad EOB DTPA) 6 months (a-c), 12 months (d-f), 18 months (g-i) and 24 months (j-l) after the intial diagnosis. The lesion in liver segment 8 continuously decreases in size until a minimal scar is formed by 18months. It is midly hyperintense on axial T2 HASTE (a, d, g, j), and shows no contrast uptake on delayed hepato-biliary phase on axial (b, e, h, k) and coronal (c, f, I, l) reformats. **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021

b



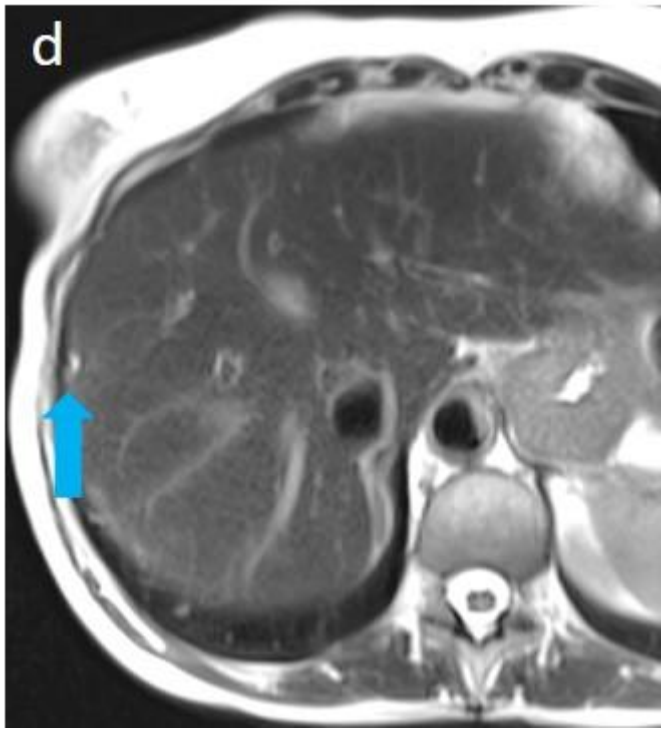
Description: MRI liver with hepatospecific contrast (Gad EOB DTPA) 6 months (a-c), 12 months (d-f), 18 months (g-i) and 24 months (j-l) after the intial diagnosis. The lesion in liver segment 8 continuously decreases in size until a minimal scar is formed by 18months. It is midly hyperintense on axial T2 HASTE (a, d, g, j), and shows no contrast uptake on delayed hepato-biliary phase on axial (b, e, h, k) and coronal (c, f, I, l) reformats. **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021

c



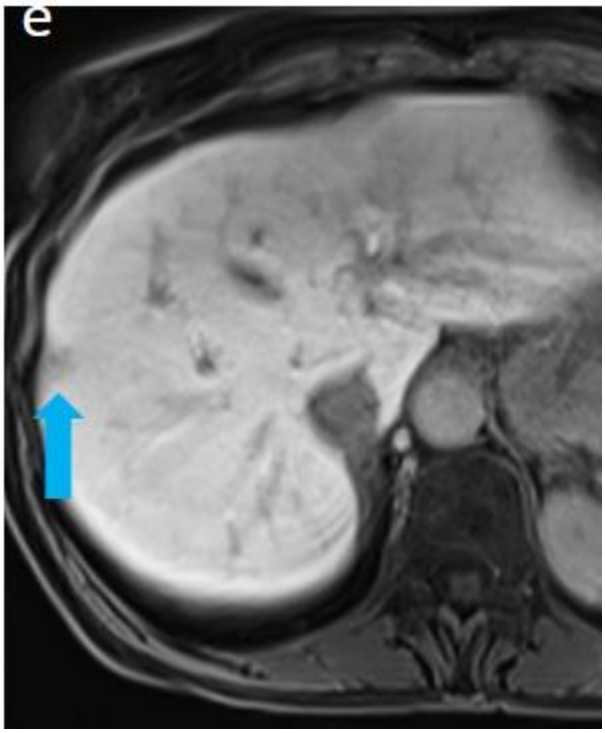
Description: MRI liver with hepatospecific contrast (Gad EOB DTPA) 6 months (a-c), 12 months (d-f), 18 months (g-i) and 24 months (j-l) after the intial diagnosis. The lesion in liver segment 8 continuously decreases in size until a minimal scar is formed by 18months. It is midly hyperintense on axial T2 HASTE (a, d, g, j), and shows no contrast uptake on delayed hepato-biliary phase on axial (b, e, h, k) and coronal (c, f, I, l) reformats. **Origin:**

d



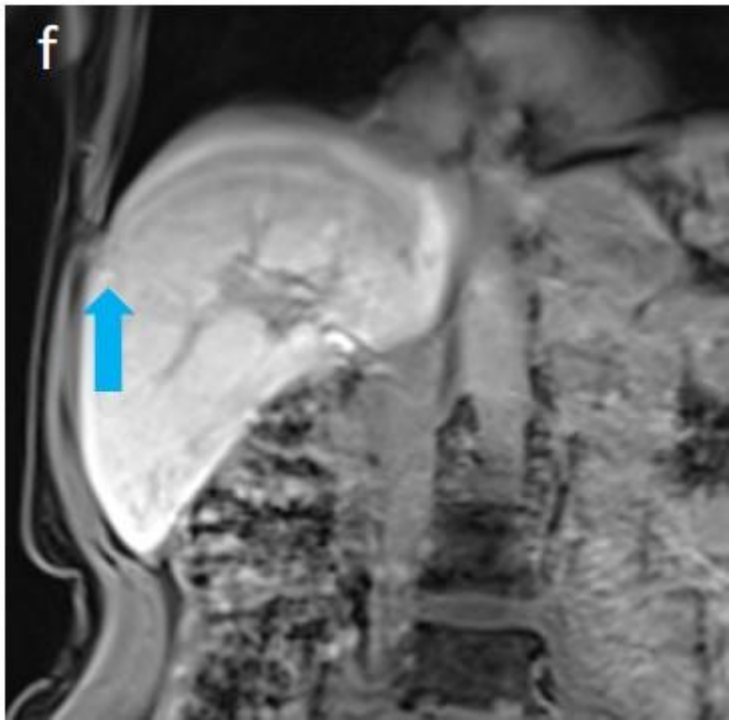
Description: MRI liver with hepatospecific contrast (Gad EOB DTPA) 6 months (a-c), 12 months (d-f), 18 months (g-i) and 24 months (j-l) after the intial diagnosis. The lesion in liver segment 8 continuously decreases in size until a minimal scar is formed by 18months. It is midly hyperintense on axial T2 HASTE (a, d, g, j), and shows no contrast uptake on delayed hepato-biliary phase on axial (b, e, h, k) and coronal (c, f, I, l) reformats. **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021

e



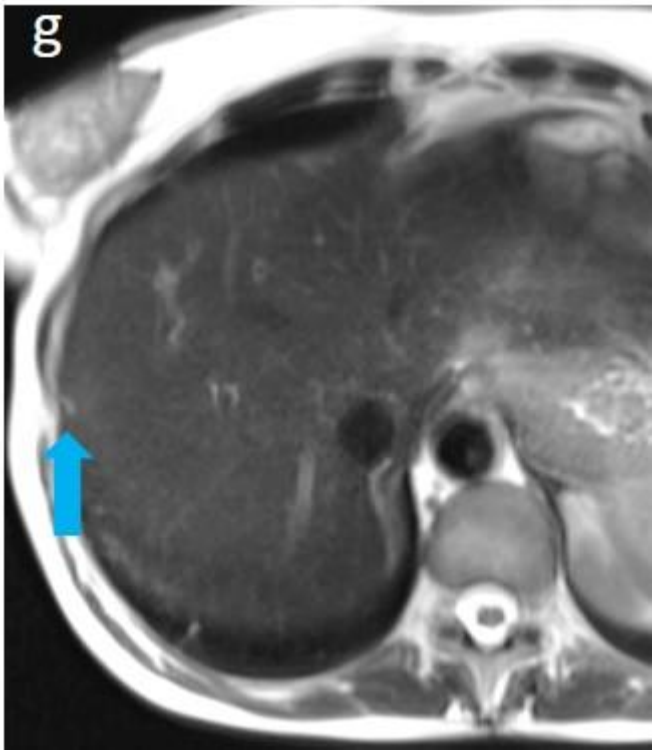
Description: MRI liver with hepatospecific contrast (Gad EOB DTPA) 6 months (a-c), 12 months (d-f), 18 months (g-i) and 24 months (j-l) after the intial diagnosis. The lesion in liver segment 8 continuously decreases in size until a minimal scar is formed by 18months. It is midly hyperintense on axial T2 HASTE (a, d, g, j), and shows no contrast uptake on delayed hepato-biliary phase on axial (b, e, h, k) and coronal (c, f, I, l) reformats. **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021

f



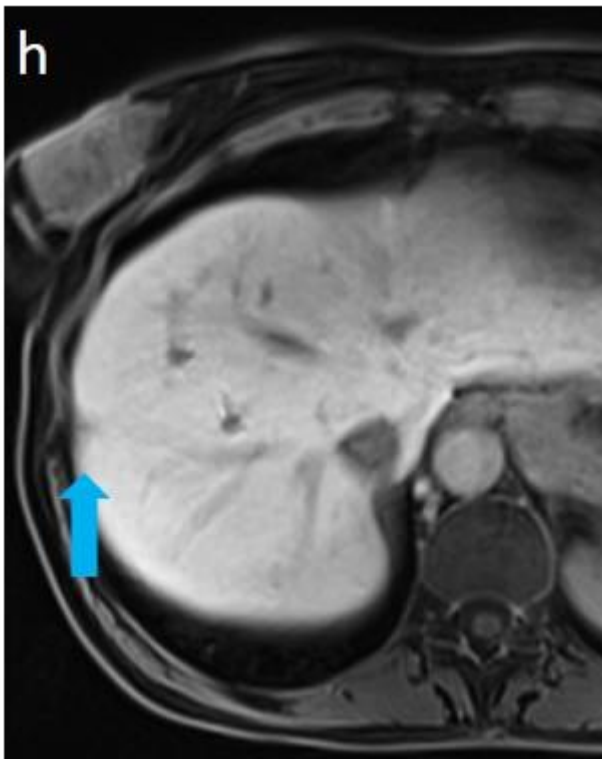
Description: MRI liver with hepatospecific contrast (Gad EOB DTPA) 6 months (a-c), 12 months (d-f), 18 months (g-i) and 24 months (j-l) after the intial diagnosis. The lesion in liver segment 8 continuously decreases in size until a minimal scar is formed by 18months. It is midly hyperintense on axial T2 HASTE (a, d, g, j), and shows no contrast uptake on delayed hepato-biliary phase on axial (b, e, h, k) and coronal (c, f, I, l) reformats. **Origin:**

g



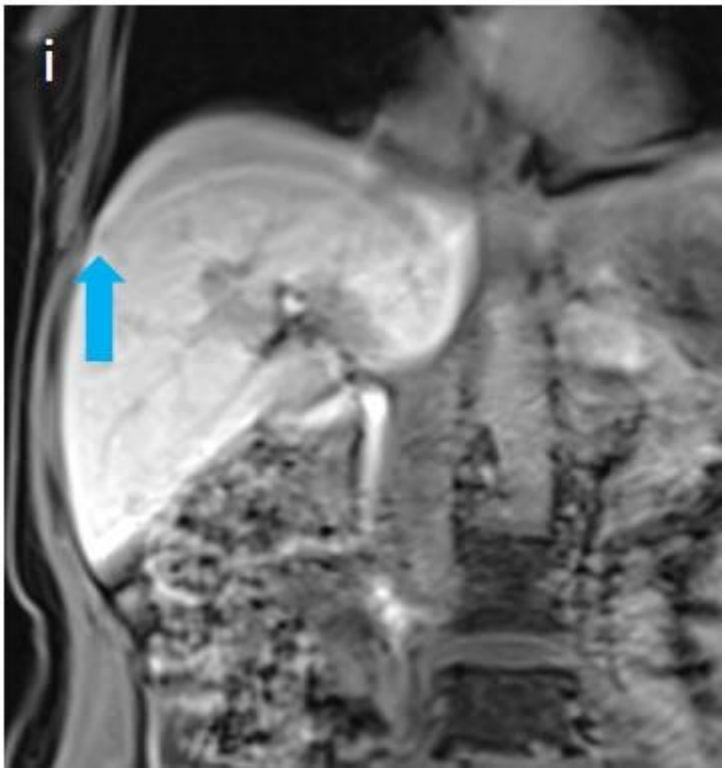
Description: MRI liver with hepatospecific contrast (Gad EOB DTPA) 6 months (a-c), 12 months (d-f), 18 months (g-i) and 24 months (j-l) after the intial diagnosis. The lesion in liver segment 8 continuously decreases in size until a minimal scar is formed by 18months. It is midly hyperintense on axial T2 HASTE (a, d, g, j), and shows no contrast uptake on delayed hepato-biliary phase on axial (b, e, h, k) and coronal (c, f, I, l) reformats. **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021

h



Description: MRI liver with hepatospecific contrast (Gad EOB DTPA) 6 months (a-c), 12 months (d-f), 18 months (g-i) and 24 months (j-l) after the intial diagnosis. The lesion in liver segment 8 continuously decreases in size until a minimal scar is formed by 18months. It is midly hyperintense on axial T2 HASTE (a, d, g, j), and shows no contrast uptake on delayed hepato-biliary phase on axial (b, e, h, k) and coronal (c, f, I, l) reformats. **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021

i



Description: MRI liver with hepatospecific contrast (Gad EOB DTPA) 6 months (a-c), 12 months (d-f), 18 months (g-i) and 24 months (j-l) after the intial diagnosis. The lesion in liver segment 8 continuously decreases in size until a minimal scar is formed by 18months. It is midly hyperintense on axial T2 HASTE (a, d, g, j), and shows no contrast uptake on delayed hepato-biliary phase on axial (b, e, h, k) and coronal (c, f, I, l) reformats. **Origin:** © Department of Radiology, Peter MacCallum Cancer Centre, VIC, Australia, 2021