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Multiple liver abscesses secondary to *Listeria monocytogenes* complicated by hepatic artery mycotic aneurysm

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SUMMARY

A woman in her 50s presented to the emergency department with 5 days of abdominal pain, nausea and vomiting. CT imaging of her liver demonstrated three enhancing, cystic lesions in her hepatic parenchyma and a large enhancing lesion in her porta hepatis concerning for a hepatic artery aneurysm. Radiographic-guided drainage was performed on two accessible liver abscesses, and cultures from this drainage grew *Listeria monocytogenes*. Serial imaging of the aneurysm demonstrated that the aneurysm spontaneously thrombosed and did not require further intervention. She was subsequently discharged on intravenous ampicillin with a plan to continue until radiographic resolution of the abscesses.

BACKGROUND

Listeria monocytogenes is a gram-positive, motile, facultative intracellular bacillus found worldwide in soil, vegetation and animals. *L. monocytogenes* is notorious for causing human infection (listeriosis) via ingestion of contaminated food, due to its ability to multiply at low temperatures and survive acidic or salty conditions.[1] Healthy individuals exposed to *L. monocytogenes* may develop a self-limited, febrile, gastrointestinal illness. In contrast, immunocompromised individuals are at risk of severe, invasive listeriosis which includes manifestations such as rhombencephalitis, myocarditis and bacteraemia.[1]

Following ingestion, *L. monocytogenes* invades the intestinal epithelium and Peyer's patches, then is transported to mesenteric lymph nodes, spleen and liver.[2] Animal models of listeriosis suggest *L. monocytogenes* may then further escape host defences and infect hepatocytes through cell-to-cell spread.[3] Although *L. monocytogenes* replication is suspected to occur primarily within hepatocytes,[3] listeriosis rarely presents with prominent hepatic involvement.[2] Of available reports on hepatic listeriosis, solitary or multiple liver abscesses, diffuse hepatitis and granulomatous hepatitis have been recorded.[2] Mycotic arterial aneurysms secondary to *L. monocytogenes* are likewise rare, and the described reports frequently involve the abdominal aorta, iliac artery,[4] or include the presence of prosthetic material.[5]

CASE PRESENTATION

A woman in her 50s with no known prior medical history presented to the emergency department for 5 days of right upper quadrant abdominal pain, nausea and vomiting. She reported that the pain developed gradually and was accompanied by multiple episodes of yellow emesis with one episode of haematemesis. At the time of presentation to the emergency department, she was having minimal abdominal pain. She denied fever, chills, night sweats, diarrhoea or constipation associated with this pain. The patient reported no medical problems and was taking no medications. She denied tobacco, drug or alcohol use.

On presentation, the patient was afebrile with a temperature of 36.7°C, her heart rate was 87 beats per minute, respiratory rate was 16 breaths per minute, blood pressure was 139/77 mm Hg and oxygen saturation was 99% on room air. The abdominal exam was soft, non-tender, non-distended and with no guarding.

Initial laboratory studies demonstrated a leucocytosis to 15.0×10^9 cells/L, with a neutrophilic predominance of 78%. Initial liver enzymes are in table 1 and did not demonstrate significant transaminitis. Other significant laboratory findings included a haemoglobin A1C of 14.2%.

INVESTIGATIONS

CT of the chest, abdomen and pelvis, and CT liver multiphase protocol were obtained and revealed
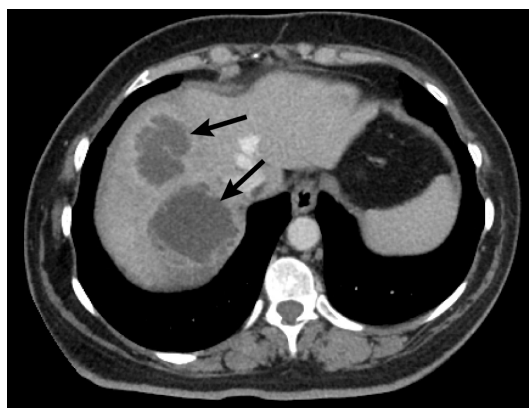

Figure 1 Multiphase liver CT in the portal venous phase demonstrates three low density lesions in the liver parenchyma with irregular peripheral enhancement. This includes a lesion in segment VII measuring 5.1×5.8×4.2 cm (lower black arrow), in segment VIII measuring 4.3×3.8×4.0 cm (upper black arrow) and in segment VI measuring 5.4×7.0×4.4 cm (not shown).

1

three peripherally enhancing, cystic lesions in the hepatic parenchyma in segments VI (5.4×7.0×4.4 cm), VII (5.1×5.8× 4.2 cm) and VIII (4.3×3.8×4.0 cm) (figure 1). The hepatic vein was nearly completely effaced due to mass effect. CT also demonstrated a large, arterially enhancing, vascular lesion in the porta hepatis measuring up to 3.2 cm which followed blood pool on
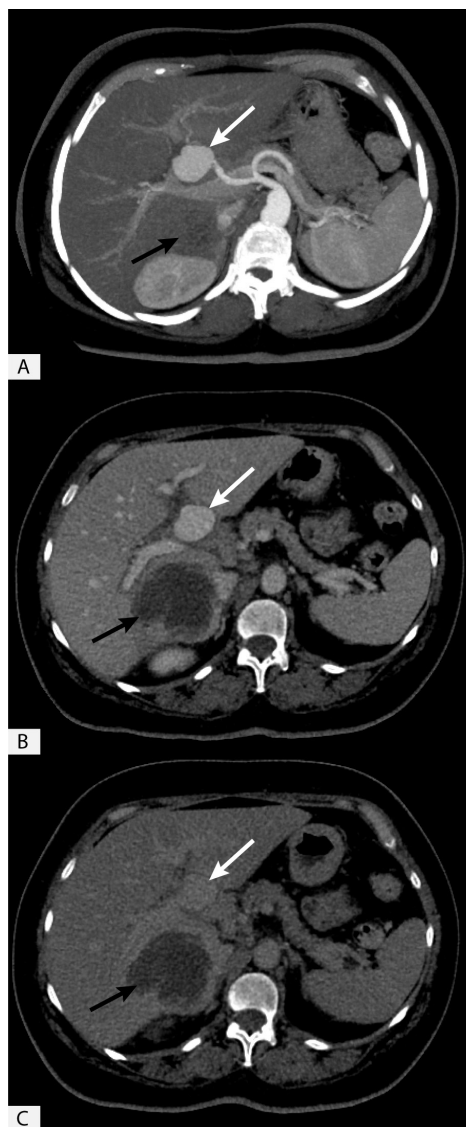

Figure 2 Multiphase liver CT shows a lesion with irregular peripheral enhancement in segment VI measuring 5.4×7.0×4.4 cm (black arrow) and a vascular lesion in the porta hepatis measuring 2.8×3.1×3.2 cm (white arrow) originating from the proper hepatic artery. This lesion demonstrates arterial enhancement on the arterial phase (A) and follows blood pool on portal venous (B) and delayed phase (C) consistent with an aneurysm.
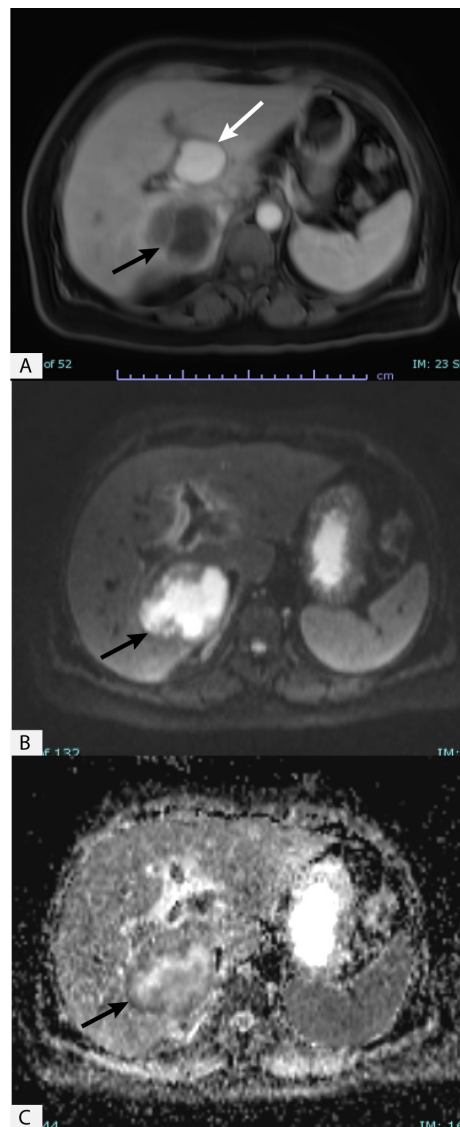

Figure 3 Contrast enhanced MRI of liver. Post contrast VIBE (volumetric interpolated breath-hold examination) sequence (A) shows interval enlargement of the proper hepatic artery aneurysm (white arrow), now measuring 3.9×3.1 cm. The fluid collections in the liver demonstrate peripheral enhancement (black arrow). Diffusion-weighted imaging (B) and corresponding apparent diffusion coefficient (C) show restriction of diffusion centrally within the cystic liver lesions, typical for pyogenic liver abscesses.

arterial, portal venous and delayed phases, concerning for a hepatic artery aneurysm (figure 2).

DIFFERENTIAL DIAGNOSIS
Given leucocytosis and peripheral enhancement of lesions on CT, the patient was presumed to have multiple liver abscesses. Differential diagnosis also included metastatic disease, primary liver malignancy and atypical haemangioma.

TREATMENT
After two sets of blood cultures were drawn, the patient was started on intravenous antibiotics with ceftriaxone 2 g every 24 hours and metronidazole 500 mg every 8 hours to target typical organisms for pyogenic liver abscesses.
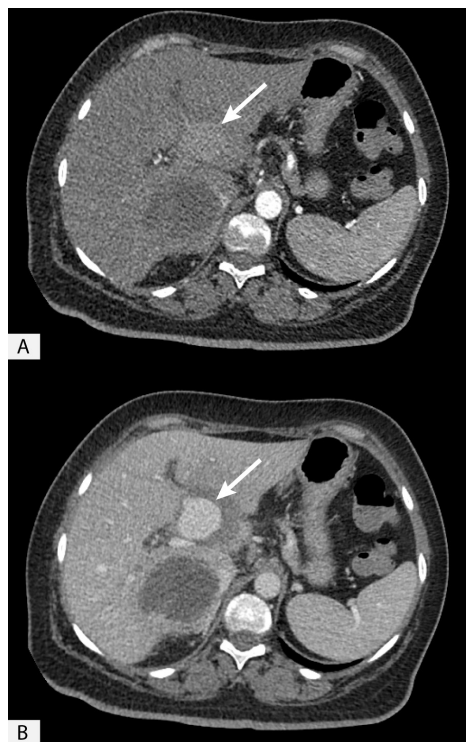
Figure 4 CT angiography demonstrates worsening inflammatory changes, including wall thickening of the aneurysm, periportal oedema and segmental occlusion of the proper hepatic artery due to extrinsic compression or vasospasm. The aneurysm (white arrow) shows an interval change in contrast enhancement, with minimal arterial phase enhancement (A) and avid, uniform contrast enhancement on the portal venous phase (B).

OUTCOME AND FOLLOW-UP

The patient was admitted to the hospital with plans to obtain MRI of the liver to further evaluate the suspected aneurysm and cystic parenchymal lesions. Multiphase abdominal MRI on hospital day (HOD) 3 showed interval enlargement of aneurysm, measuring 3.9×3.1 cm, and again noted three stable lesions consistent with abscesses (figure 3). Given the interval enlargement of aneurysm, the concern for a mycotic aneurysm was raised, and infectious disease and vascular surgery were consulted. CT angiography of the abdomen obtained on HOD 5 demonstrated worsening inflammatory changes in the porta hepatis, including new wall thickening of the aneurysm and diffuse periportal oedema. A significant regional mass effect had developed, causing near complete luminal narrowing of the proper hepatic artery. There was an interval change in the enhancement pattern of the vascular lesion (figure 4), which showed minimal filling on the arterial phase, presumably due to compromised arterial supply and uniform opacification on the portal venous phase.

Following a multidisciplinary team meeting with vascular surgery, infectious disease and internal medicine, a decision was made to proceed with Interventional radiologyembolisation of hepatic artery given concern for aneurysmal growth and rupture. The patient was not considered a candidate for surgical intervention due to the risk of blood loss and the benefits of coiling or intervening on an enlarging aneurysm were thought to outweigh the risks of endovascular management in a potentially infected space. In preparation for planned endovascular coiling, catheter angiography of the proper hepatic artery was conducted
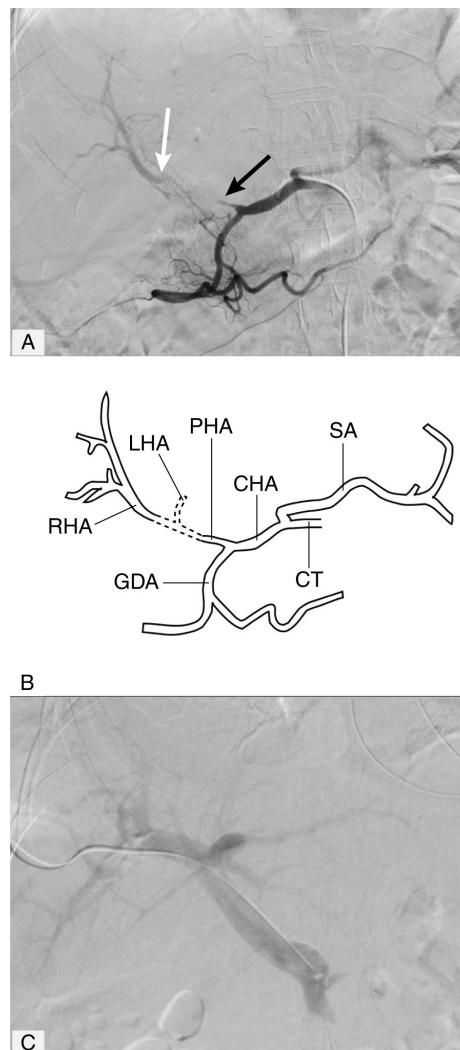

Figure 5 Digital subtraction angiogram (DSA) of the common hepatic artery shows no opacification of the aneurysm (A). There is significant mass effect in the porta hepatis, causing segmental narrowing of the proper hepatic artery (black arrow) with reconstitution distally (white arrow). These findings suggest either thrombosis of the aneurysm or a lack of arterial communication. Hepatic arterial anatomy is illustrated in diagram (B, created by author XQ), with non-visualised segment marked in dashed lines. CT, coeliac trunk; SA, splenic artery; CHA, common hepatic artery; PHA, proper hepatic artery; GDA, gastroduodenal artery; LHA, left hepatic artery; RHA, right hepatic artery. The DSA of the portal system shows normal enhancement of the main portal vein with no communication with the aneurysm (C).

on HOD 6. This demonstrated a lack of opacification of the aneurysm sac (figure 5A,B) suggestive of thrombosis. There was significant narrowing of mid and proximal portion of the proper hepatic artery, possibly due to compression from the adjacent thrombosed aneurysm, similar to that seen on the previous CT. To further evaluate the vascular supply to this lesion, portal venous angiography was performed on HOD 7 (figure 5C). This showed no appreciable communication between the vascular lesion and the portal vein. On HOD 7, drainage catheters were placed under ultrasound guidance in the two right hepatic liver abscesses. The third abscess was in an anatomical position unamenable to drainage.

On HOD 12, *L. monocytogenes* grew from two hepatic abscess cultures. Blood cultures remained without growth. The patient

Table 2 Brief literature review of non-aortic aneurysms associated with *Listeria monocytogenes* infection

Ref	Age/sex	Location of aneurysm	Surgical intervention	Antibiotics and duration	Survival
(4)	76/M	Left common iliac artery	Debridement, in situ Y graft	Ampicillin for 22 days, then amoxicillin for 62 days	Survived
(10)	66/M	Bilateral common femoral arteries	Resection, repaired with Dacron rube graft, replaced by venous bypass after listeria was found	Ampicillin and gentamicin for 6 weeks	Survived
(11)	85/M	Right superficial femoral artery	Resection, bypass with vein graft	Amoxicillin-clavulanic acid for 3 days, then amoxicillin for unknown duration, then meropenem for unknown duration; after discharge: intravenous ertapenem for 2 weeks, then oral linezolid indefinitely	Survived
(12)	72/M	Left popliteal artery	Resection	Unknown	Survived
(13)	73/M	Left internal carotid	Resection, bypass with vein graft	Amoxicillin for unknown duration	Survived
(14)	83/M	Left popliteal artery	Ligation of aneurysm, arterial bypass, redo aortic valve replacement, aortic graft replacement	Ampicillin and gentamicin for 6 weeks, then amoxicillin indefinitely	Survived
(15)	80/M	Right common iliac artery	Resection, right axillo-bifem bypass	Ampicillin and gentamicin for 6 weeks	Survived
(16)	52/M	Inferior mesenteric artery	Resection, left colectomy	Amoxicillin and rifampin for unknown duration	Survived
(17)	57/M	Superior mesenteric artery	None	Penicillin for 6 days	Death due to aneurysmal rupture

Extended review with additional clinical details in Supplementary Table 1.

Afib, atrial fibrillation; CABG, coronary artery bypass graft; COPD, chronic obstructive pulmonary disease; CVA, cerebral vascular accident; DM, diabetes; HLD, hyperlipidaemia; HTN, hypertension; MI, myocardial infarction; TIA, transient ischaemic attack.

was switched from ceftriaxone and metronidazole to ampicillin given intravenously, 2 g every 4 hours. On directed questioning, the patient recalled a severe episode of gastroenteritis about 6–8 weeks prior after eating food from a street vendor. Susceptibility testing demonstrated that the isolate was sensitive to ampicillin (minimal inhibitory concentration (MIC) 0.25), meropenem (MIC 0.125), penicillin (MIC 0.25) and TMP/SMX (MIC 0.032). All abscesses decreased in size on repeat liver CTs on HOD 13 and HOD 20, and the hepatic artery aneurysm remained thrombosed. Percutaneous drains were removed due to minimal drainage, and the patient was discharged in good condition on HOD 23 with outpatient intravenous ampicillin until the resolution of undrained liver abscess on imaging. A repeat CT of her liver done at 5 weeks of treatment demonstrated ongoing improvement in abscess size. She was switched to oral amoxicillin with a plan for repeat imaging. The patient remained in good condition without abdominal pain, nausea or vomiting.

DISCUSSION

L. monocytogenes is an important foodborne pathogen that leads to an estimated 1600 illnesses, 1500 hospitalisation and 260 deaths per year in the USA.[6] Risk factors for invasive disease include immunocompromising states such as pregnancy and diabetes mellitus, as well as increased age, male sex, and Asian, Black and Hispanic race/ethnicities.[6] In particular, Hispanic patients have been found to have higher rates of listeriosis,[6 7] potentially due to consumption of unpasteurised dairy products.[8]

Although pathogenesis of invasive listeriosis involves the portal system and the liver, clinical liver involvement of listeriosis is rare. In a 2007 literature review by Scholing et al,[2] they identified 34 case reports of listeriosis involving the liver including solitary liver abscess, multiple liver abscesses, diffuse hepatitis and granulomatous hepatitis. Only three out of 11 patients with multiple liver abscesses survived,

and all three received percutaneous abscess drainage.[2] In general, multiple liver abscesses from listeriosis have been described as carrying a poor prognosis, particularly if full percutaneous drainage of the abscesses cannot be performed.[2] However, more recently, a case report by van der Voort described a patient with fever and elevated transaminases who was found to have multiple small abscesses on CT unamenable to drainage and blood culture positive for *L. monocytogenes*.[9] The patient ultimately recovered and abscesses on repeat imaging resolved with intravenous amoxicillin therapy alone.[9] Similarly, our patient did not undergo percutaneous abscess drainage until 1 week into her hospitalisation, and did not receive Listeria-targeted antibiotics until HOD 12. Despite this, our patient remained stable throughout her hospitalisation and was discharged home in good condition.

In Scholing et al's review,[2] serum transaminase levels were elevated only in a few patients with liver abscesses. Blood cultures were positive rarely in solitary liver abscess but in most cases of multiple liver abscesses. Our patient with undiagnosed diabetes mellitus presented with multiple liver abscess without transaminase elevation or positive blood cultures, and the causative organism was only confirmed with percutaneous abscess drainage and culture. Thus percutaneous drainage and culture remain critical for microbiological diagnosis and antibiotic treatment.[2]

L. monocytogenes is a rare cause of mycotic aneurysms, with only 29 cases reported in the literature. Almost all cases reported have occurred in elderly patients with a male predominance, and most involved aortic and common iliac arteries. Among non-aortic aneurysms, none involved underlying hardware and most occurred in iliac, femoral and popliteal arteries, with only one case each involving the inferior mesenteric, superior mesenteric and internal carotid arteries was reported[4 10–17] (table 2). We thus report

the first known case of common hepatic artery aneurysm due to *L. monocytogenes*.

Also notable in our patient was the rapid evolution in size and ultimate self-thrombosis of the aneurysm. Mycotic hepatic aneurysms are rare, accounting for 0.1% or arterial aneurysms and 20% of visceral aneurysms.[18] Symptomatic hepatic aneurysms may present as right upper quadrant abdominal pain, haemobilia and obstructive jaundice, referred to as Quincke's triad, which is observed in less than one-third of patients.[18] The pathogenesis of hepatic artery aneurysm in this case is not well elucidated. It is unknown whether the mycotic aneurysm arose from Listeria infection of an existing aneurysm due to haematogenous spread, or whether it arose from contiguous spread of bacteria from adjacent abscess or biliary structures to the artery. In terms of treatment of mycotic aneurysms, surgical removal of infected tissue and culture directed antibiotic therapy are essential.[19] In our patient, the rapid increase in size of the aneurysm prompted concern for rupture, and endovascular coil embolisation was planned as a temporising measure. The aneurysm, however, self-thrombosed prior to the planned procedure without compromising end organ perfusion and remained stable on repeat imaging.

Learning points

- ► Invasive disease from *Listeria monocytogenes* should be considered in patients with appropriate risk factors including race, ethnicity, age, immunocompromise and recent history of potential foodborne illness.
- ► Hepatic involvement of listeriosis is rare but may present as liver abscess, diffuse hepatitis or granulomatous hepatitis.
- ► Transaminases may be normal and blood cultures negative in listeria liver abscesses.
- ► Drainage and directed antibiotic therapy are crucial in the diagnosis and treatment of listeria liver abscesses.
- ► Mycotic visceral artery aneurysms are rare and require prompt diagnosis and appropriate surgical and antibiotic therapy.

Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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