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REVIEW ARTICLE

Cirrhosis-related musculoskeletal disease: radiological review

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ABSTRACT

Musculoskeletal problems in patients with liver disease are common; however, they are not so well described in the literature. Therefore, there is a need to collate information on these disorders, as their incidence is on a constant rise and some of these pathologies can severely debilitate the patient's quality of life. These disorders are parietal wall varices with or without bleeding, spontaneous intramuscular haematoma (e.g. rectus sheath), abdominal wall hernia, anasarca, hepatic osteodystrophy, septic arthritis, osteomyelitis, necrotizing fasciitis, osseous metastases from hepatocellular carcinoma etc. While portal hypertension plays a key role in disorders, in others, dysregulation of the coagulation system or a compromised immune system are responsible. Imaging plays an essential role in the assessment of these complications and awareness of these musculoskeletal manifestations is vital for establishing a timely diagnosis and planning of appropriate therapy, as these disorders can significantly impact the morbidity and mortality and also influence candidacy for liver transplantation. We herein comprehensively appraise various musculoskeletal complications associated with chronic liver disease/liver cirrhosis especially from an imaging perspective which, to the best of our knowledge, have not been collectively described in English literature.

INTRODUCTION

Liver cirrhosis is the common end stage of all chronic liver diseases characterized by scarring, architectural distortion and progressive depletion of functional reserve, constituting a leading cause of morbidity and mortality worldwide.¹ Viral hepatitis and heavy alcohol consumption are by far the commonest causes, while non-alcoholic fatty liver disease is rapidly emerging as an important precipitating factor.² Clinically, cirrhosis leads to multisystem complications, reducing life expectancy markedly. Virtually, any system can be involved and in the past few decades, there has been major progress in the knowledge of multisystem effects of liver disease especially on the nervous and cardiopulmonary systems.^{1,2} However, very few reviews have studied the effects of chronic liver disease on the musculoskeletal system.

Cirrhosis-related musculoskeletal diseases range from: abdominal wall varices, spontaneous intramuscular bleeding, post-paracentesis parietal wall bleeding with or without pseudoaneurysm formation, abdominal wall hernia, anasarca, hepatic osteodystrophy (HO), septic arthritis, osteomyelitis, necrotizing fasciitis (NF), osseous metastases from hepatocellular carcinoma (HCC) etc. (Table 1). While portal hypertension plays a key role in

some, in others, fluid retention, hypoproteinaemia, dysregulation of the coagulation system or a compromised immune system are responsible.

To the best of our knowledge, musculoskeletal disorders associated with cirrhosis have not been discussed under one heading in the English literature. Some of these disorders can lead to long-term incapacitating sequels such as avascular osteonecrosis of the femoral heads (OFHs) or collapsed vertebral bodies, leading to a chronic state of pain and incapacitation. Moreover, active infective complications (for e.g. spondylodiscitis, septic arthritis, osteomyelitis etc.) as well as extrahepatic musculoskeletal dissemination of HCC can influence candidacy for liver transplantation. Imaging plays an important role in the assessment of these pathologies and we herein review the clinico-radiological perspectives of diverse liver diseases and liver cirrhosis-related disorders affecting the joints, bones, skin and soft tissues.

ABDOMINAL WALL VARICES

Portal hypertension is defined as portal pressure >5–10 mmHg. In an attempt to decompress the portal system, blood is shunted away from the liver into the low-pressure

Table 1. Cirrhosis-related musculoskeletal disease

Stigmata of portal hypertension
Abdominal wall varices
Haemorrhagic complications
Spontaneous rectus haematoma
Infective complications
Cellulitis
NF
Septic arthritis
Spondylodiscitis
Osteomyelitis
Osseous (non-infective) complications
HO
Fractures
Non-traumatic osteonecrosis
HCC metastases
Osseous/soft-tissue metastases
Iatrogenic
Post-paracentesis parietal wall bleeding
Post-FNA needle track seeding
Miscellaneous
Sarcopenia
Abdominal wall hernia
Body fat oedema/anasarca

FNA, fine-needle aspiration; HCC, hepatocellular carcinoma; HO, hepatic osteodystrophy; NF, necrotizing fasciitis.

systemic circulation *via* portosystemic shunts. Consequently, many different venous collateral pathways can be encountered including gastro-oesophageal varices, lienorenal and gastrosplenic shunts and paraumbilical venous collaterals.³⁻⁷ On imaging, the recanalized paraumbilical vein is a relatively common venous collateral pathway in patients with cirrhosis (Figure 1).^{3,5,7} The

paraumbilical vein arises from the umbilical portion of the left portal vein and traverses along the falciform ligament, frequently extending towards the umbilicus. It anastomoses with either the superior epigastric/internal thoracic veins and drains into the superior vena cava or the inferior epigastric vein to drain into the inferior vena cava *via* the common femoral or external iliac vein.³⁻⁷ “Caput medusae” refers to distended abdominal wall collateral vessels that radiate from the umbilicus (Figure 1) and are supplied by the paraumbilical and/or omental veins and drain into the systemic circulation *via* one of the two inferior epigastric veins.⁵ Multidetector CT, owing to its volumetric data acquisition and multiplanar reformatting capabilities, allows excellent visualization of these portosystemic collaterals including detailed information about their extent and physiopathology (afferent and efferent drainage pathways). Knowledge of the presence and extent of these parietal wall collaterals is crucial, as an inadvertent collateral vessel injury during paracentesis or abdominal surgery can cause catastrophic blood loss.^{3,6} This is especially important while contemplating a hernia repair (umbilical hernia is a common occurrence in patients with liver cirrhosis), as the true extent and complexity of the paraumbilical vein can be explicitly delineated only with contrast-enhanced cross-sectional imaging such as CT or MRI.⁶

SPONTANEOUS RECTUS HAEMATOMA

Development of spontaneous and potentially fatal retroperitoneal and rectus muscle haematomas is an uncommon complication of patients with liver cirrhosis. Studies have found that these haematomas are often caused by minor abdominal trauma, abdominal wall straining (such as violent coughing) and iatrogenic causes (such as intra-abdominal injections, paracentesis or laparoscopic surgery). Anticoagulation therapy has also been implicated as a risk factor.⁸ Spontaneous rectus and retroperitoneal haematomas usually respond well to conservative treatment. However, overall fatality rates of up to 4% have been reported in literature, with increased mortality seen in patients having a deranged coagulation profile and in those requiring surgical intervention.^{8,9} It is speculated that the source of

Figure 1. (a) The coronal maximum intensity projection image and (b) axial contrast-enhanced CT image showing a recanalized paraumbilical vein (arrowheads) serving as an afferent for multiple periumbilical and abdominal wall collaterals (dashed arrows), forming a “caput medusae” and draining *via* the inferior epigastric veins.

Figure 2. (a,b) Greyscale ultrasound images showing a well-defined heteroechoic collection in the rectus muscle (arrows).

bleeding in these patients is either from the venous collaterals associated with portal hypertension or, when abdominal trauma is the cause, from the epigastric arteries. Although the diagnosis can often be made clinically, bedside ultrasonography can be helpful in confirming the clinical suspicion (Figure 2), delineating the extent of haematoma and in regular follow-up.¹⁰ For retroperitoneal haematomas, CT is more useful. Moreover, CT angiography can be used to rule out any active source of bleeding and to aid in therapeutic (endovascular or surgical) planning (Figure 3).¹¹

CELLULITIS

Cellulitis is a relatively common soft-tissue infection which results in dermal thickening, swelling, erythema and pain. If left untreated, it may progress to involve the deep fascia, synovium or bone, depending on its location. In patients with liver cirrhosis, the impaired intestinal portal barrier and intestinal bacterial overgrowth contribute to bacterial translocation from the intestines more easily than in normal hosts. In addition, the

low complement levels, poor opsonization capacity, portosystemic shunting and impaired reticuloendothelial system in patients with cirrhosis makes them more prone to bacteraemia.^{12–15} Hence, these patients are more prone to developing cellulitis. Also, cellulitis in patients with cirrhosis may be underestimated owing to the frequent swelling of skin and soft tissues from hypoalbuminemia.^{15,16} Thus, healthcare providers should actively look for inflammatory changes in the skin and signs and symptoms of infection in patients with liver cirrhosis, especially in those with a complicated status, to facilitate early detection of these cases and prompt commencement of therapy. The diagnosis of cellulitis is essentially clinical and imaging is primarily used to assess the extent of the disease and rule out any complications. CT shows dermal and soft-tissue thickening and fat stranding. Hyperaemia can be seen on contrast-enhanced images. The presence of air in the soft tissue in the absence of penetrating trauma suggests progression to NF, while rim-enhancing fluid collections in the setting of cellulitis suggest formation of abscess. MRI reveals heterogeneously T_2

Figure 3. (a–d) Axial contrast-enhanced CT images at different levels from the same patient demonstrating mixed attenuation hyperdense haematoma in the left rectus (dashed arrow) with active extravasation of the contrast (solid arrows). An attendant large extraperitoneal haemorrhagic collection contiguously extending into the left hemipelvis (asterisk) can be seen, indenting the bladder. Minimal bleed is also seen in the right rectus (arrowhead).

Figure 4. Coronal oblique T_2 weighted images from a patient with cellulitis showing extensive bilateral linear hyperintensities (arrowheads) in the subcutaneous plane of both legs.

hyperintense thickening (Figure 4) and variable contrast enhancement of the involved soft tissues. MRI is also more helpful than CT in ruling out progression to osteomyelitis and NF.

NECROTIZING FASCIITIS

NF is a rapidly progressive and often fatal soft-tissue infection that spreads along the deep fascia. It is most commonly the result of polymicrobial infection. Several reports have shown that liver cirrhosis is a common underlying disease in patients with NF.^{17–19} Despite improved medical care, the mortality rates in patients with NF remain high, especially among patients with cirrhosis, in whom rates have been reported to be nearly nine-fold when compared with that among patients with NF without cirrhosis.¹⁷ Although the diagnosis is essentially clinical, imaging is useful to map the extent of disease to aid in surgical planning and to exclude other disease processes. CT usually reveals dermal thickening, increased soft-tissue attenuation and inflammatory

fat stranding with or without crescent-shaped fluid collection or air in the subfascial planes. CT is the most sensitive modality for the detection of soft-tissue gas and compared with radiography, it is superior in evaluating the extent of bony involvement. MRI is the modality of choice for the detailed evaluation of soft-tissue infection. MRI of NF shows circumferential dermal and soft-tissue thickening having variable signal intensity on T_1 weighted images and increased signal intensity on T_2 weighted images (Figure 5).

SEPTIC ARTHRITIS

Septic or infectious arthritis is inflammation of a joint caused by a bacterial, viral or fungal infection. The pathogens usually enter the joint capsule *via* the haematogenous route, although direct penetration during surgical procedures or trauma is also speculated. The joints affected by septic arthritis often have pre-existing damage, including traumatic injuries or degeneration. The incidence of septic arthritis is high especially among those who have underlying comorbidities such as diabetes mellitus, malignancy and chronic renal failure and among patients who are immunocompromised. Metabolic derangements and impaired immunity make patients with liver cirrhosis more prone to infections, osteoporosis and osteonecrosis and hence to septic arthritis.^{20–23} Large joints with abundant blood supply to the metaphyses are most prone to bacterial infection, with the most commonly affected joints being the hip, knee and shoulder. Patients usually present with pain and swelling over the affected joint with fever. Imaging generally plays a complementary role to arthrocentesis in the diagnosis of joint sepsis. Also, imaging can be used to guide joint aspirations in uncommon or difficult sites such as the sacroiliac or sternoclavicular joints. If the synovial fluid cannot be retrieved, however, radiologic studies become of utmost importance. A plain X-ray film usually reveals the destruction of the subchondral bone on both sides of the joint with juxta-articular osteoporosis. CT features of septic arthritis include joint effusion, joint-space narrowing, bone and cartilage erosions, gas within the joint and soft-tissue swelling (Figure 6). MRI is highly sensitive and specific in the early diagnosis of septic arthritis. It is especially useful in detecting early cartilaginous damage. Synovial enhancement and the presence of a joint effusion have been reported to have the highest

Figure 5. (a) Coronal oblique and (b) axial T_2 weighted images from a patient with necrotizing fasciitis showing extensive subcutaneous and myofascial plane hyperintensities involving the right thigh (arrowheads) with attendant dermal thickening and myositis. A disproportionate enlargement of the girth of the right lower limb (arrows) as compared with the contralateral side can be noted.

Figure 6. (a, b) Axial contrast-enhanced CT images demonstrating a large juxta-articular abscess (arrows) in a patient with septic arthritis secondary to *Staphylococcus aureus*.

correlation with the clinical diagnosis of a septic joint. Perisynovial soft-tissue oedema and collections are also commonly seen (Figure 7). Patients with tuberculous arthritis may have more bone erosions and less marrow signal abnormality on MRI than patients with pyogenic arthritis. If unrecognized and left untreated, septic arthritis can result in irreversible joint damage. Thus, in patients with cirrhosis and fever, it is imperative to have a high index of suspicion to promptly diagnose and treat septic arthritis.

SPONDYLODISCITIS

Spondylodiscitis, a term encompassing vertebral osteomyelitis, spondylitis and discitis, is the most common manifestation of haematogenous osteomyelitis in patients aged over 50 years.^{24,25}

Similar to septic arthritis, spondylodiscitis has an increased incidence in patients with liver cirrhosis.²⁶ Patients usually present with non-specific back or neck pain. Diagnosis is difficult and often delayed or missed owing to the rarity of the disease and the high frequency of low back pain in the general population. Thus, imaging plays a crucial role in the accurate diagnosis of this entity and to rule out complications. Plain radiographs demonstrate poor definition of the vertebral end plates, subchondral lucency and decrease in the height of the disc space. As the disease progresses, end-plate destruction may ensue, resulting in loss of vertebral height with formation of soft-tissue masses in the paravertebral region. CT is the imaging modality of choice in accurately assessing these bony abnormalities, including early end-plate changes, before they can be picked up on radiographs. However, it is inferior to MRI in imaging neural tissue and abscesses. On MRI, the characteristic changes consist of decreased signal intensity from the disc and adjacent vertebral bodies and loss of end-plate definition on T_1 weighted images while on T_2 weighted sequences, the disc and adjoining vertebral bodies show increased signal intensity (Figure 8). Enhancement of the vertebral bodies, discs and the surrounding soft tissue can be used to differentiate early infective lesions from degenerative changes on MRI. The prevalence of skeletal tuberculosis is also high in patients with liver cirrhosis.^{27–29} Tuberculous spinal infection is suggested by a lack of disc involvement, the presence of paraspinal abscesses (Figure 9), posterior vertebral changes and involvement of multiple non-contiguous levels.³⁰

HEPATIC OSTEODYSTROPHY

The term HO is used to define the group of alterations in the bone mineral metabolism found in patients with chronic liver disease. The exact prevalence of HO is unknown, ranging between 11 and 56% depending on the series.³¹ The aetiology is complex and multifactorial and manifests clinically as osteopenia and osteoporosis. The causes of decreased bone mass in these patients include poor cholecalciferol absorption through the intestine, poor activation of vitamin D, impaired osteoblastic function due to hyperbilirubinaemia, chronic hypogonadism, chronic steroid therapy, low body mass index and concomitant intake of alcohol by many of these patients.^{32,33} The decreased bone mineral density can be assessed at an early stage by dual-energy X-ray bone absorptiometry performed on the lumbar

Figure 7. (a, b) Axial short tau inversion-recovery images in a patient with cirrhosis with septic arthritis showing marrow oedema (solid arrows) along the femoral and acetabular articular surfaces with attendant myositis (dashed arrows) and juxta-articular fluid collection (arrowheads).

Figure 8. Sagittal (a) T_2 weighted and (b) short tau inversion-recovery images showing discal hyperintensity at the L4-L5 level (arrows) with attendant marked vertebral enhancement (dashed arrows) and minimal discal enhancement in keeping with acute spondylodiscitis.

vertebrae and femoral neck. The rate of decrease in bone mass increases as the disease progresses, with the patient becoming more prone to bone fractures and the morbidity associated with it. The fracture rate ranges from 5 to 20%.^{34,35} Vertebral fractures are most frequent in these patients, manifesting in 3–18% of cases (Figures 10 and 11). Therefore, an assessment of risk factors for bone loss and bone mineral density measurement should be a part of routine protocol in patients with chronic liver disease. An early diagnosis of HO is imperative to correct reversible risk factors which predispose to bone mass loss. Treatment and prevention strategies include dietary and lifestyle changes, calcium and vitamin D supplementation and bisphosphonates.

AVASCULAR NECROSIS

OFH is a devastating disease that can lead to collapse of the femoral head and secondary degenerative osteoarthritis, hampering the patient quality of life.^{36,37} Alcohol and steroid use are the most important factors, contributing to more than two-third of cases of non-traumatic OFH. The association of non-traumatic osteonecrosis in cirrhotics is not easy to verify, as this could be attributed, at least partly if not fully, to excessive alcohol consumption (in patients with alcoholic cirrhosis) or steroid usage (for *e.g.* in patients with primary biliary cirrhosis).^{38–41} Nevertheless, recent studies have shown liver cirrhosis, irrespective of underlying aetiology, to be an independent risk factor for the

Figure 9. Sagittal (a) and axial (b) fat-saturated post-contrast T_1 weighted images in a patient with Pott's spine showing changes of spondylodiscitis (solid arrow) with attendant peripherally enhancing left psoas abscess (dashed arrow).

Figure 10. Plain radiographs (frontal and lateral) of the lumbar spine in a patient with hepatic osteodystrophy showing gross osteopenia with flattening of L4 and L5 vertebral bodies. AP, anteroposterior; LAT, lateral; LT, left; RT, right.

development of non-traumatic osteonecrosis regardless of oral corticosteroid use, alcohol consumption and smoking.^{39–41} Hung et al⁴⁰ evaluated the nationwide population-based database to examine the occurrence of OFH in patients with cirrhosis and in those without cirrhosis during a 3-year follow-up. Their study cohort comprised 40,769 adult patients with liver cirrhosis compared with a comparison cohort of 40,769 randomly selected age- and sex-matched healthy subjects. 321 (0.8%) patients with cirrhosis vs 126 (0.3%) patients without cirrhosis were found to have OFH ($p < 0.001$). During the 3-year period, Cox's regression analysis, adjusted by the

patient age, sex and other confounding factors, revealed that patients with cirrhosis had a higher risk for OFH than patients without cirrhosis (hazard ratio = 2.38; $p < 0.001$). It is important to note that only 19.4% cirrhosis cases were alcohol related, while the remaining 80.6% were attributed to viral hepatitis. As in the general population, prior joint trauma, male gender, younger age and gouty arthritis were found to have increased risk of developing OFH.⁴⁰ MRI is certainly the imaging modality of choice, offering the highest sensitivity and specificity both for early diagnosis and in looking for complications (Figure 12).⁴²

Figure 11. Sagittal (a) T_1 weighted, (b) T_2 weighted and (c) short tau inversion-recovery sequence showing acute collapse of the L1 vertebral body (arrows) with attendant marrow oedema. Prior collapse of the T11 vertebral body can also be noted (arrowheads).

Figure 12. Coronal (a) T_2 weighted image and (B) short tau inversion-recovery sequence showing subchondral collapse of left femoral head collapse (arrows) with reduced cranial joint space and minimal joint effusion in a patient with non-alcoholic steatohepatitis-related cirrhosis.

EXTRAHEPATIC OSSEOUS/SOFT-TISSUE METASTASES OF HEPATOCELLULAR CARCINOMA

The incidence of HCC is on an increase world over and it now accounts for the second leading cause of cancer deaths and fifth most common cancer globally.^{43–45} The incidence is especially high in Asia and Africa, owing to the high endemic prevalence of viral hepatitis-related chronic liver disease. Unfortunately, the worldwide incidence is expected to escalate further because of increasing alcohol abuse, hepatitis C infections and obesity-related comorbidities (non-alcoholic fatty liver disease). Despite the evolution of newer therapeutic modalities, patients with HCC still have a dismal long-term survival and prognosis. The tumour is known to invade the portal and hepatic veins, and venous infiltration predisposes to pulmonary metastasis, which may be followed by haematogenous or lymphatic dissemination to sites such as bones and lymph nodes. Lungs are by far the commonest metastatic site followed by lymph nodes and bones.^{43–46} Katyal et al⁴⁵ examined CT findings in 403 consecutive patients with HCC and reported extrahepatic metastases in 148 cases. The most common sites included the lung in 81 (55%) patients, the abdominal lymph nodes in 60 (41%) patients and the bone in 41 (28%) patients. 128 (86%) of 148 patients had advanced tumour (either intrahepatic Stage IVA or Stage III) at the occurrence of metastases. The authors concluded that incidental extrahepatic lesions at CT in patients with Stage I or II intrahepatic

HCC are unlikely to represent metastatic HCC. Various other studies have reported extrahepatic metastasis in up to 30–50% of cases of HCC, with osseous metastases constituting 6–39% of all extrahepatic metastases. Osseous metastases in HCC tend to be multiple, lytic, expansile and hypervascular and are most commonly seen in the vertebrae.^{43,47} The lumbar and thoracic vertebrae are the most common sites to get involved. Other unusual extrahepatic metastatic sites reported include the adrenals, peritoneum, diaphragm, soft tissues (Figure 13), brain, skin and oral cavity.^{43–47}

POST-PARACENTESIS ABDOMINAL WALL BLEEDING

Refractory ascites is one of the most frequent causes of morbidity in patients with end-stage liver disease. Considerable evidence favours the use of large-volume paracentesis (LVP) with albumin replacement as the preferred treatment of tense ascites in these patients.⁴⁸ Patients treated with LVP supported by plasma volume expansion have shortened hospitalization, better preserved systemic haemodynamics and renal function. Studies have shown that LVP is a safe procedure, with a low risk of complications. These complications include leakage of the ascitic fluid, local infection, abdominal wall haematomas, intraperitoneal haemorrhage and intestinal perforation.⁴⁹

Figure 13. (a, b) Axial contrast-enhanced CT images with extensive multifocal hepatocellular carcinoma showing parietal wall metastatic deposit (dashed arrows). Also, the large mesenteric deposits can be noted (arrowheads).

Figure 14. (a) Coronal contrast-enhanced CT and (b) maximum intensity projection images showing post-paracentesis parietal wall bleed (arrow) and an enhancing pseudoaneurysm (interrupted arrows), which was subsequently closed through percutaneous glue injection.

Abdominal wall bleeding is a rare but lethal complication of LVP.^{49–51} It has often been anecdotally attributed to underlying coagulopathy and thrombocytopenia in patients with cirrhosis. However, literature is divided over this and there are reports both supporting and refuting this hypothesis.^{48–51} Although the exact incidence of haemorrhagic complication in patients with cirrhosis and portal hypertension is unknown, studies have reported incidence rates of between 0.8 and 3.0%.⁵¹ Signs and symptoms of haemorrhage can manifest from minutes to days after the procedure. Bleeding at the time of or immediately after the procedure is usually due to direct injury to the superficial epigastric vessels or abdominal wall collaterals resulting in abdominal wall haematomas. Delayed bleeding is rare and has been reported to occur up to 4 days after the initial procedure.⁵² A postulated mechanism for delayed haemorrhage is rapid decompression of splanchnic circulation owing to release of intra-abdominal pressure, leading to a marked increase in portosystemic blood flow through collaterals with subsequent haemorrhage from collaterals. Imaging usually reveals a mixed attenuation haemorrhagic collection in the abdominal wall. Active leak of the intravenously administered contrast can often be detected with or without a pseudoaneurysm (Figure 14). Associated intraperitoneal haemorrhage is a usual accompaniment.

Transcatheter coiling or percutaneous glue embolization appears to be superior to both open and laparoscopic surgery in the management of these pseudoaneurysms.

POST-FINE-NEEDLE ASPIRATION SOFT-TISSUE METASTASES

Needle biopsies, especially fine-needle biopsies, are considered to be relatively safe and accurate in obtaining a diagnosis of malignancy. However, depending on the retrieval rate, the number of passes needed to obtain sufficient material for histological examination varies. This can result in the possibility of cancer cells being disseminated along the needle track. The incidence of seeding has been the subject of a number of studies, with reported incidence varying widely from 0 to 11%.^{53–58} A literature review performed by Stigliano et al⁵⁵ concluded that the overall mean risk of seeding is 1.73%, based on results from 2242 patients. The exact incidence of needle tract seeding is difficult to accurately determine, as there have been only case reports and small retrospective studies. Proposed mechanisms for needle tract seeding include adhesion of tumour cells to biopsy needle and relocation of tumour cells *via* post-biopsy bleeding. Possible risk factors for needle tract seeding include larger needle diameter, more needle passes, patient immunosuppression and tumour aggressiveness.⁵⁶

Figure 15. (a, b) Axial contrast-enhanced CT in a patient with prior radiofrequency ablation for left lobe hepatocellular carcinoma showing extensive disease recurrence in the left lobe with extension of tumour up to the skin surface along the needle tract (arrowheads).

Figure 16. (a) Sagittal scannogram and (b) axial contrast-enhanced CT in a patient with liver cirrhosis and gross tense ascites (asterisk) showing a large umbilical hernia (solid arrows). The recanalized paraumbilical vein (dashed arrow) is seen at the neck of the umbilical hernia.

The incidence of needle track seeding may rise as a result of the puncture of tumours detected at an early stage in patients with a longer life expectancy. Needle tract seeding invariably presents with one or multiple rounded enhancing nodules along the needle biopsy tract. Nodules can be located within the peritoneum, along the abdominal muscles which were penetrated by the needle, or in the subcutaneous and cutaneous tissues (Figure 15).⁵³ Seeding metastases demonstrate similar enhancement characteristics, appearance and doubling time as an intrahepatic tumour, which typically demonstrates a strong arterial phase enhancement with washout of contrast on venous and equilibrium phases. Appearance is generally non-specific, with heterogeneous echogenicity, variable T_1 signal intensity, usually high T_2 signal intensity and hypermetabolic activity on the positron emission tomography scan. Radiographs are most likely to appear normal, although a soft-tissue mass or rib erosion could potentially be seen.

MUSCLE WASTING OR SARCOPENIA

Severe muscle wasting or sarcopenia is one of the most common but frequently overlooked complications in patients with cirrhosis, which negatively impacts survival, quality of life and response to stresses, including infections and surgeries.^{59–61} In general, sarcopenia is defined as a muscle mass two standard deviations below that of the mean muscle mass of healthy young adult reference groups. Malnutrition can affect up to 90% of patients with cirrhosis.⁶⁰ Impaired capacity of the liver to

metabolize nutrients, inadequate oral intake mainly due to early satiety in ascites, malabsorption and metabolic abnormalities are few of the important factors causing malnutrition and resultant sarcopenia in patients with cirrhosis.⁶⁰ A number of indirect methods have been employed to assess the body composition in cirrhotics. However, most of these are either not readily available or lack reproducibility. Also, their accuracy may be affected by the presence of fluid retention. Cross-sectional imaging modalities have emerged as the gold standard to quantify skeletal muscle mass and hence constitute a good objective method for the nutritional assessment of patients with cirrhosis and identification of sarcopenia.^{60,61} On cross-sectional imaging, the L3 skeletal muscle index (L3MI) expressed as cross-sectional muscle area height⁻² is used to assess the muscle mass. For calculating the L3MI, CT scan at the level of the third lumbar vertebra is analyzed with the help of commercially available software, which enables specific tissue demarcation using previously reported Hounsfield unit thresholds. Skeletal muscle is identified and quantified by Hounsfield unit thresholds, and cross-sectional area of the muscle is calculated. Specific cut-off values for L3MI to diagnose sarcopenia have been established for patients with malignancies and cirrhosis to readily identify this abnormality.⁶⁰

ABDOMINAL WALL HERNIA

Abdominal wall hernias, especially umbilical hernias, are common in patients with liver cirrhosis and ascites, with an overall

Figure 17. (a, b) Axial contrast-enhanced CT showing subcutaneous fluid retention along the parietal wall (arrowheads) in a patient with liver cirrhosis, ascites and anasarca.

Figure 18. (a, b) Axial contrast-enhanced CT showing diffusely hazy subcutaneous fat (arrowheads) in a patient with compensated hepatitis-B-related liver cirrhosis.

incidence of approximately 20% (up to 40% in cases of major ascites).^{62–65} While the most common predisposing factor is increased abdominal pressure secondary to the presence of tense ascites (Figure 16), other contributory factors include weakening of the abdominal wall fascia and muscles owing to malnutrition and progressive muscle wasting. Another factor implicated is the dilatation and recanalization of umbilical veins, which leads to abdominal wall weakening and herniation at umbilicus.^{62,63} While the majority of patients remain asymptomatic, nevertheless, these patients are at a higher risk of complications such as incarceration, bowel strangulation, spontaneous rupture, ulceration, fistulae etc.^{62,63} One has to be mindful that rapid removal of the ascitic fluid during paracentesis can predispose these patients to incarceration. On imaging, umbilical hernias are frequently seen as an incidental finding during routine follow-ups.⁶² However, special attention should be given to the contents of the hernia sac and the presence of periumbilical vein or omental varices, if any, should be explicitly highlighted, keeping in mind that these patients might require surgical intervention. The optimal therapeutic management, surgical technique as well as timing of surgery repair remain a matter of debate owing to the relatively high perioperative morbidity and mortality associated with hernia repair in cirrhotics. By far, refractory ascites remain the most important risk factor for complications and recurrence. Ideally, these patients should undergo hernia repair either during or after successful liver transplantation.^{63,65}

BODY FAT OEDEMA

Fat oedema in patients with liver cirrhosis can be a result of multiple factors. The most important pathogenic mechanism is

portal hypertension causing increased hydrostatic pressure within the mesenteric veins leading to egress of fluid into the mesentery. In addition, hypoalbuminaemia and decreased aldosterone catabolism due to the underlying liver dysfunction leads to a state of persistent fluid overload (Figure 17).^{66,67} Tyrrel et al⁶⁷ compared the CT densities of mesenteric, retroperitoneal and subcutaneous fat in patients with and without liver cirrhosis and found a visually evident and statistically significant increase in the density of mesenteric fat in patients with cirrhosis. The increase in density was also seen in the retroperitoneal and subcutaneous fat (Figure 18), although the difference was not statistically significant. With increasing severity, this oedema in the mesentery and omentum assumes a more diffuse, nodular and mass-like appearance. Occasionally, these findings can be florid enough to be mistaken for an inflammatory or neoplastic infiltration of omentum or mesenteric lymphadenopathy.⁶⁸ Since superadded infections are quite common in patients with end-stage liver disease, it is important for the radiologist to be aware of the spectrum of various appearances of oedema in cirrhosis.

CONCLUSION

Cirrhosis-related musculoskeletal disorders constitute a major cause of hospitalization for patients with chronic liver disease. While preventing these complications is challenging, their timely detection can help in the commencement of appropriate therapy, thus helping curb the high morbidity and mortality associated with them. As imaging plays an essential role in their assessment, radiologists must recognize the possibility of these complications and vigilantly evaluate the imaging examinations for their potential presence.

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