

UC San Diego

UC San Diego Previously Published Works

Title

Computed tomography of hyper-attenuated liver: Pictorial essay

Permalink

<https://escholarship.org/uc/item/6g07847v>

Journal

Clinical Imaging, 97

ISSN

0899-7071

Authors

Ghahremani, Gary G

Hahn, Michael E

Fishman, Elliot K

Publication Date

2023-05-01

DOI

10.1016/j.clinimag.2023.02.012

Peer reviewed



Body Imaging

Computed tomography of hyper-attenuated liver: Pictorial essay

Gary G. Ghahremani^{a,*}, Michael E. Hahn^a, Elliot K. Fishman^b^a Department of Radiology, University of California-San Diego Medical Center, 200 West Arbor Drive, San Diego, CA 92103, USA^b Department of Radiology, Johns Hopkins University Hospital, 733 North Broadway, Baltimore, MD 21205, USA

ARTICLE INFO

Keywords:

High-density liver
Computed tomography
Drug-induced hepatic changes
Amiodarone therapy
Hemolytic anemias
Wilson's disease

ABSTRACT

Demonstration of a very dense or hyper-attenuated liver on the pre-contrast CT images of the abdomen can be an unexpected finding. It may present as a diagnostic challenge if the underlying cause of it is not apparent from the provided clinical history. There are about 12 different pathologic conditions that are associated with deposition of radiopaque elements within the hepatic parenchyma, resulting in diffuse or multi-lobar hyperdense appearance of the liver on abdominal radiographs and CT. Most of them are drug-induced or iatrogenic in nature, while others are the sequelae of genetic disorders like thalassemia, Wilson's disease, and primary hemochromatosis. This pictorial essay will present the CT appearance and etiology of hyper-attenuated liver in various clinical entities.

1. Introduction

Computed tomography (CT) of the abdomen is a sensitive method for the evaluation of liver abnormalities, particularly when a dedicated contrast-enhanced examination is performed.^{1,2} However, the non-contrast CT images can also provide relevant information if the liver attenuation number deviates from the physiologic range. This measures normally between 50 and 60 Hounsfield units (HU) on multidetector CT examinations and about 10 HU higher than the spleen.² A lower attenuation is typically associated with hepatic steatosis due to non-alcoholic fatty liver.³ In contrast, an increased attenuation exceeding 70 HU is caused by a wide spectrum of pathological conditions that lead to the deposition of high-density or radiopaque elements within the liver.

There has been an increasing trend in the clinical practice to perform abdominal CT examinations after contrast enhancement only and eliminate the pre-contrast phase to reduce the radiation dose.^{1,4} Therefore, the presence of a hyper-attenuated liver may be overlooked or difficult to recognize after its enhancement by intravenous contrast material. The modern multi-detector and dual-energy CT scanners are capable of reconstructing virtual unenhanced images that resemble the appearance of true pre-contrast scans. This is achieved by a post-processing technique, using dual-energy CT to remove iodine from the acquired post-contrast images, and thereby demonstrate conditions that cause either hypo- or hyper-attenuation of the liver.^{1,5}

The purpose of this article is to illustrate the CT features of hyper-attenuated liver caused by various etiologies, and to review the

pertinent literature about the prevalence and pathogenesis of these conditions.

2. Amiodarone therapy

The most common cause of a hyper-attenuated liver is the long-term treatment with amiodarone. This drug has been used very extensively for the management of ventricular arrhythmia and atrial fibrillation.⁶ It accounted for 2.4 million prescriptions dispensed to 677,240 patients in the United States during 2020.⁷ Amiodarone has a molecular weight of 681 and contains 37.3% iodine. In comparison, the water-soluble contrast agent iothalamate meglumine (Conray 60, Guerbet Company, Paris, France) has 28.2% organically bound iodine.

Amiodarone is administered orally or intravenously, and most patients receive it for as long as 5 to 10 years. It is highly lipophilic and metabolized in the liver to its active derivative. This drug has an elimination half-life of 35 to 100 days, thus remains in the body for a prolonged time after its intake has been discontinued (Fig. 1, A and B).

Adverse effects of amiodarone include thyroid dysfunction in 14%–18% of patients caused by iodine content of the medication, and pulmonary changes due to interstitial fibrosis and pneumonia in about 3% of cases. Approximately 25% of patients receiving amiodarone will have a transient asymptomatic rise of their liver enzyme levels. Amiodarone-induced hepatotoxicity can develop after long-term therapy, causing abnormal liver function tests in 15% to 50% of patients, and hepatitis or cirrhosis in about 3% of cases.⁸ LV and Zhao recently performed dual-

* Corresponding author.

E-mail address: gghahremani@ucsd.edu (G.G. Ghahremani).<https://doi.org/10.1016/j.clinimag.2023.02.012>

Received 26 September 2022; Received in revised form 7 February 2023; Accepted 20 February 2023

Available online 23 February 2023

0899-7071/© 2023 Elsevier Inc. All rights reserved.

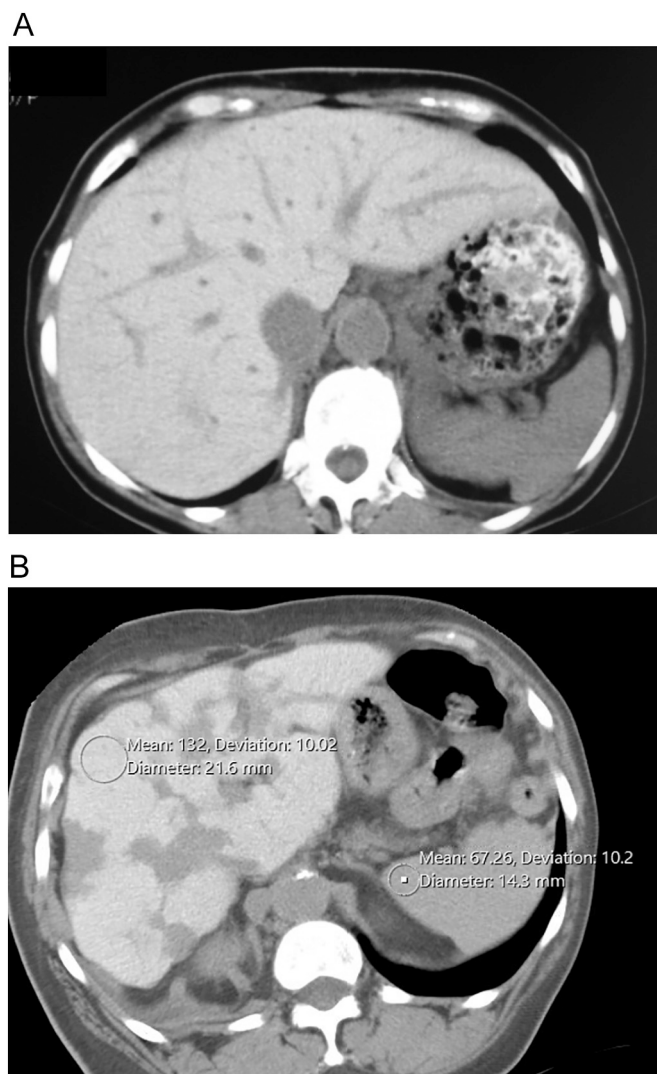


Fig. 1. CT images of hyper-attenuated liver due to amiodarone in 2 patients. A: This 57-year-old woman had a 5-year history of amiodarone therapy for cardiac arrhythmia. Note the appearance of low-density hepatic vasculature within the liver. The radiopaque gastric content is from amiodarone and calcium tablets. B: Distorted configuration of the dense liver (132 HU) in this 62-year-old woman is due to amiodarone-induced cirrhosis. Attenuation numbers for the spleen (67 HU) and muscles (53 HU) are within the normal range.

energy CT of the abdomen in 3 such patients.⁹ The liver attenuation ranged from 110 to 144 at 100 kVp and from 86 to 104 at 140 kVp, almost double the normal values.

3. Thalassemias

These genetic blood disorders affect about 280 million people, who are predominantly of Middle Eastern, South Asian, and African descent. An estimated 1.5% of the global population or about 80–90 million patients have beta-thalassemia major, also known as Cooley's anemia.¹⁰ The prevalence of this disease in the United States has increased during the past 5 decades due to the rising immigration of individuals from the affected regions.¹¹

Thalassemias are characterized by severe anemia due to decreased or absent hemoglobin production, thus the patients require regular blood transfusions. The resulting iron overload leads to its deposition in the liver parenchyma and accounts for its high attenuation on CT (Fig. 2, A and B). Other abdominal CT features of thalassemia include para-vertebral masses of extramedullary hematopoiesis, splenomegaly,

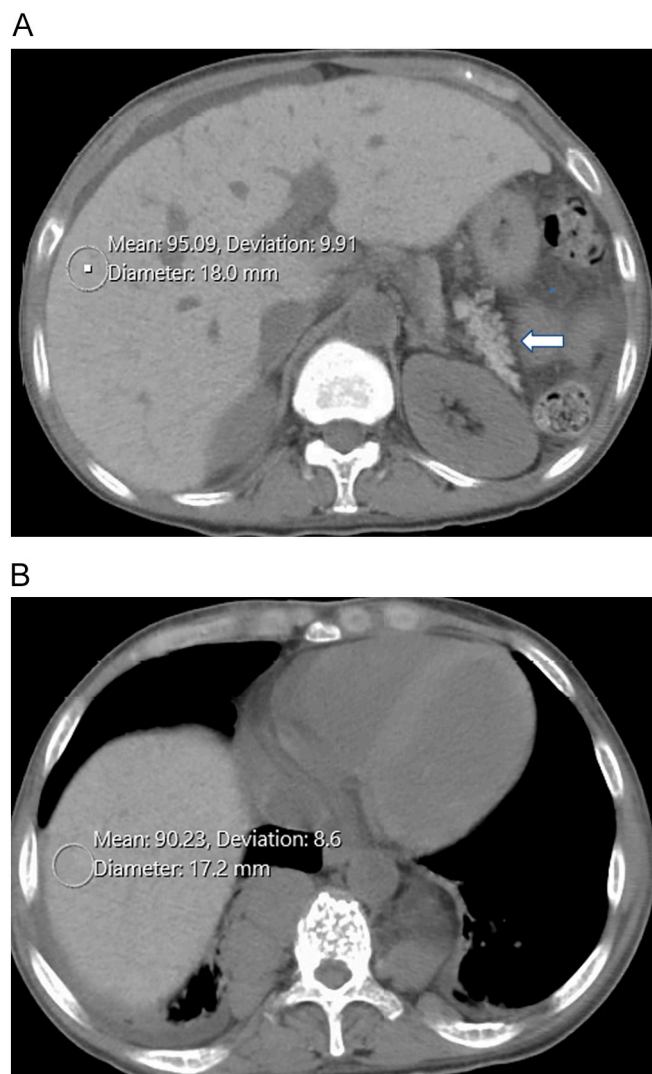


Fig. 2. A: CT images of the upper abdomen in a 31-year-old man with thalassemia reveal a hyper-dense liver (95 HU) and pancreas (arrow) due to iron deposition. B: A higher section shows the para-spinal masses caused by extramedullary hematopoiesis, low-density of ventricular blood content due to severe anemia, and the abnormal appearance of visualized bones.

gallstones, and osteopenia of vertebrae and ribs with striated or coarse trabecular appearance.¹²

4. Wilson's disease

Wilson's disease or hepatolenticular degeneration is a genetic disorder of copper metabolism, occurring with a prevalence of 1 in 30,000 individuals.^{13,14} This congenital disease is autosomal recessive and manifests only when the mutated gene is inherited from both parents. This gene prevents the incorporation of intestinally absorbed copper in to ceruloplasmin for subsequent urinary excretion, thereby resulting in progressive accumulation of copper in the liver, kidneys, and brain (Fig. 3).

The main feature of Wilson's disease on abdominal CT is a diffusely hyper-attenuated liver (Fig. 3). Concomitant findings include liver cirrhosis, renal calculi, premature osteoarthritis, osteopenia, and chondrocalcinosis of intervertebral discs and hip joints.^{15,16} Note that copper is non-ferromagnetic and invisible on MRI, but contour abnormalities and parenchymal nodules of the liver are frequently seen in patients with Wilson's disease.



Fig. 3. Abdominal CT of this 10-year-old girl with Wilson's disease demonstrates a markedly dense liver (138 HU) due to copper deposition. She had a history of seizures, and intellectual disability that may account for the radiopaque gastric content of ingested eggshells and foreign objects.

The clinical symptoms of Wilson's disease usually manifest between the ages of 5–35 years. The early stage of it causes deposition of copper in the liver and hepatitis, with gradual development of cirrhosis.^{15,16} Neurological symptoms related to the brain involvement will occur as well. A characteristic feature of Wilson's disease is the appearance of a brown circle around the iris, called Kayser-Fleischer ring.¹³

Early diagnosis of this genetic disorder is important because several effective therapies are available, such as chelating compounds to remove the excess copper, dietary measures to reduce copper absorption, and liver transplant in advanced cases.^{13,14}

5. Hemochromatosis

The primary type of hemochromatosis is a hereditary disease due to abnormal genes that regulate iron absorption from the gastrointestinal tract. It results in iron overload and its deposition in the liver and other organs. About 2–5% of the Caucasian population is carrier of the mutated genes, and hemochromatosis is a common genetic disorder among the Caucasians of Northern European ancestry.^{17–19}

The secondary form of hemochromatosis is an acquired condition associated with hemolytic anemias and diseases treated with iron supplements that cause iron deposition in organs of reticuloendothelial system (liver, spleen, lymph nodes) and other tissues like the pancreas and kidneys.¹⁸

Iron deposition in the liver of patients with hemochromatosis manifests a homogeneously increased liver attenuation to about 75–130 HU on non-contrast CT images (Fig. 4). The portal and hepatic veins will appear conspicuous because of their relatively low density within the hyper-attenuated liver.¹⁹ This condition is usually diagnosed through blood tests, liver biopsy, and abdominal CT or MRI. The latter modality is particularly useful for quantification of the liver iron content because its superparamagnetic effect leads to a markedly reduced signal intensity on T2-weighted images.²⁰

It should be noted that hemochromatosis refers to the systemic iron deposition with tissue damage, whereas hemosiderosis represents focal deposition of hemosiderin due to bleeding and breakdown of red cells without causing organ damage or require treatment.

6. Transfusional iron overload

The body of an average adult has 4–5 g of iron, and each unit of transfused blood contains 200–250 mg of iron. Therefore, patients who

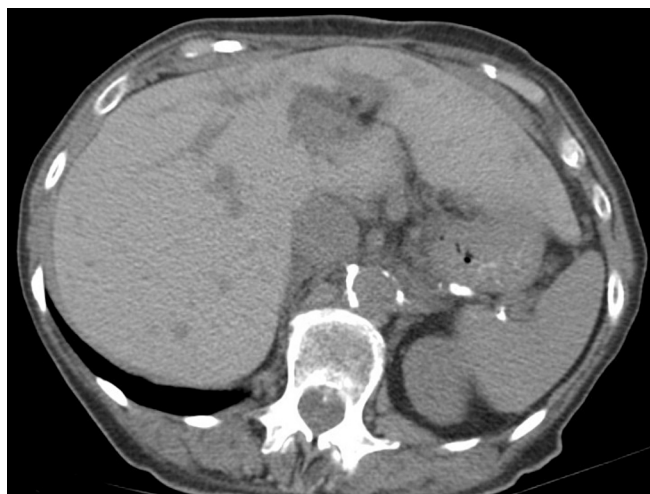


Fig. 4. CT appearance of the liver due to hemochromatosis in a 79-year-old woman. The hepatic attenuation measured 88 HU.

receive more than 10 units of blood are at risk of developing iron overload and its accumulation mainly in the liver parenchyma. It has been calculated that a patient with severe anemia requiring 2 units of blood transfusion per month over 4 years would accumulate 20 g of iron, which is sevenfold greater than the normal amount of body iron.²¹

The intestinal resorption of iron is well regulated and based on 10–20 mg of it in a typical daily diet. There is no physiological mechanism to remove iron from the body, and any excess amount of it is deposited in the tissues. The liver is the principal site of iron storage and regulation of its homeostasis. Therefore, its attenuation on CT examination is increased in patients with sickle cell disease or other types of anemia (Fig. 5, A and B), particularly after receiving frequent blood transfusions and iron supplements.^{22,23} However, MR imaging is currently the optimal technique for quantification of hepatic iron.^{20,24} It is of interest to note that chronic anemia and iron overload are two of the most prevalent disorders worldwide and affect over a billion people.²¹

7. Thorotrast

Thorotrast had been widely used as an angiographic contrast material for 3 decades until 1950s, when it was finally disapproved for clinical usage in the United States. It was produced as a colloidal suspension of thorium dioxide, a radioactive substance with biological half-life of 22 years and emitting alpha radiation. Almost 10 million patients worldwide had been examined with thorotrast, particularly while undergoing cerebral angiography.²⁵

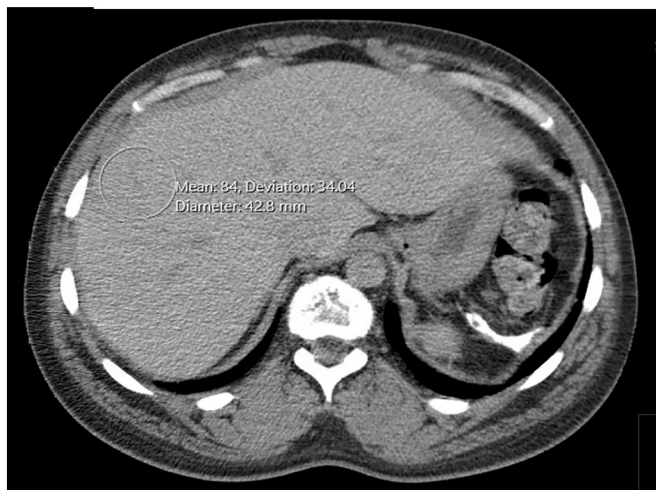
Thorotrast is retained in the liver, spleen, lymph nodes and bone marrow for prolonged time, and its radioactivity can cause various hepatosplenic malignancies such as angiosarcoma.²⁶ In the surviving patients, however, the increased density of the liver and spleen is clearly recognized on abdominal radiographs and CT examinations many decades after it had been injected (Fig. 6).

8. Gold therapy

The treatment of rheumatoid arthritis with water-soluble gold salts has been widely used over the last seven decades. The two compounds called sodium aurothiomalate and aurothioglucose are administered orally or by intramuscular injection because of their profound anti-inflammatory effects.^{27,28} The gold particles are then retained in the liver, spleen, and lymph nodes, thereby causing their markedly increased density on abdominal CT examination. These findings have been illustrated in several publications.^{29,30}

It should be noted that the CT images of this entity are similar to

A



B



Fig. 5. Transfusional iron overload in 2 cases. A: Axial CT section of the upper abdomen of this 37-year-old man with sickle cell anemia shows a calcified spleen and increased hepatic attenuation (84 HU) due to iron deposits from repeated blood transfusions. Typical changes of the spine and pelvis were seen on bone window. B: CT image of the hyper-attenuated liver (143 HU) and spleen (116HU) caused by transfusion-induced iron overload in a patient with multiple myeloma.

those seen with thorotrast deposition (Fig. 6). However, the clinical history of rheumatoid arthritis and coexisting arthritic changes of the spine or extremities can provide clues to suspect gold therapy as the potential cause of dense liver and spleen.

The preferential retention of gold in the hepatic parenchyma and spleen has led to recent usage of gold nanoparticles as a liver-specific contrast agent for CT imaging and for targeted therapy of cancer in these 2 organs.^{31–33}

9. Mineral supplements

Mineral supplements and vitamins are commonly used in the United States by about 70% of adults aged over 65 years and by a third of children and adolescents.³⁴ The over-the-counter products typically contain multiple high-density minerals in the form of calcium phosphate or carbonate, potassium iodide, copper sulfate, as well as water-soluble salts of iron, zinc, chromium, selenium, molybdenum, and several trace elements.

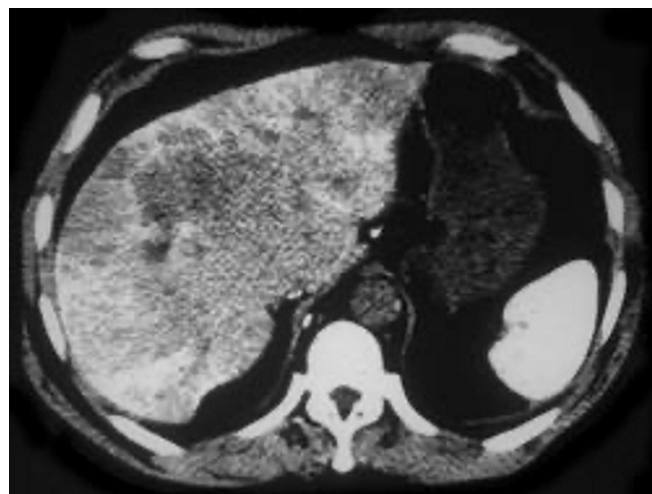


Fig. 6. CT image demonstrates the hyper-dense liver and spleen caused by retained thorotrast that had been used for cerebral arteriography several decades earlier.

In patients who have been taking excessive amounts of these dietary supplements for prolonged time, the intestinal resorption of these minerals can lead to the accumulation of radiopaque elements within the liver. Therefore, an increased hepatic attenuation may be demonstrated on non-contrast CT images (Fig. 7).

There have been some reports of liver injury following long-term ingestion of these dietary supplements. However, their beneficial effects on health status appear to be minimal or negligible according to some investigations.³⁵

10. Iatrogenic causes

Intravasation of barium during radiological studies of the gastrointestinal tract is very unusual but may occur due to inadvertent mishaps.³⁶ A case of diffuse opacification of the liver and spleen due to massive intravascular embolization of barium was recently published.³⁷ This complication had occurred when the balloon-tipped enema catheter was misplaced into the vagina and caused its perforation (Fig. 8). A similar case of this complication had been reported previously by Steinman and colleagues.³⁸

A hyper-attenuated liver may be seen following intravasation of the

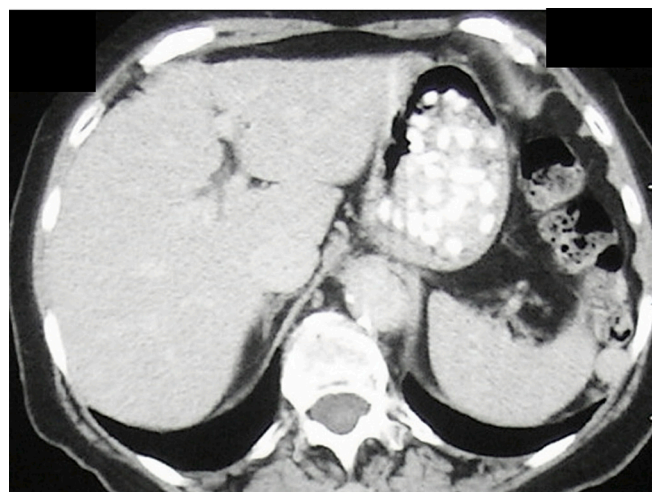


Fig. 7. CT appearance of the hyper-attenuated liver and spleen in a 77-year-old woman with prolonged history of ingesting large doses of mineral supplements. Note that many of such radiopaque tablets are visible within her stomach.



Fig. 8. CT of the upper abdomen in this 72-year-old woman shows a markedly dense opacification of the liver and spleen caused by massive barium intravasation during colon examination through a misplaced enema tip. (Reproduced with permission, Reference³⁵).

oil-based ethiodized iodine known as Lipiodol (Guebert Company, France). This contrast material has been used for several radiological procedures that include hysterosalpingography and lymphography.^{39,40} A recent comprehensive review of 19,339 women, who had undergone Lipiodol hysterosalpingography revealed a 2.7% incidence of intravasation and potential complications despite a favorable effect on their fertility outcomes.⁴¹ The current application of Lipiodol is mainly for transarterial chemoembolization of the hepatic tumors.^{42,43} Therefore, CT examination performed after this procedure will demonstrate multiple densely opacified liver lesions due to retained Lipiodol (Fig. 9).

In patients with renal failure the clearance of administered intravenous contrast material through the glomerular filtration is markedly delayed.⁴⁴ It leads to a rather prolonged increase in the hepatic and renal attenuation on CT examination. This finding may persist for 1–3 days in patients with severe uremia unless they are dialyzed promptly.⁴⁵

11. Diffuse or multi-lobular liver calcification

Hepatic calcifications represent a common source of increased liver density on abdominal CT images, and they are associated with a wide spectrum of pathological processes.⁴⁶ Most of them appear as focal or clusters of radiopacity caused by granulomatous diseases (tuberculosis, histoplasmosis), parasitic infections (hydatid disease), liver abscesses, and post-traumatic hematomas. However, large multilobular and scattered punctate calcifications may occur in hepatocellular carcinoma, metastatic mucinous adenocarcinoma of the gastrointestinal tract and ovaries, or metastases from osteogenic malignancies (Fig. 10). Shibuya et al. reported a patient with diffuse liver calcification, which had developed following a severe episode of hypotensive shock from massive gastrointestinal bleeding.⁴⁷ Other authors have also described large areas of liver calcification due to infarct or other pathologic conditions.⁴⁸

12. Conclusions

The detection of a hyper-dense liver on non-contrast CT examination is not unusual, and it obviously indicates the deposition of one or several radiopaque elements in the hepatic parenchyma. However, this finding may be overlooked or difficult to appreciate if the abdominal CT is performed only after contrast enhancement, and the pre-contrast phase of the study is not obtained.⁴ This may not be a problem with modern multi-detector and dual-energy CT scanners that allow a post-processing

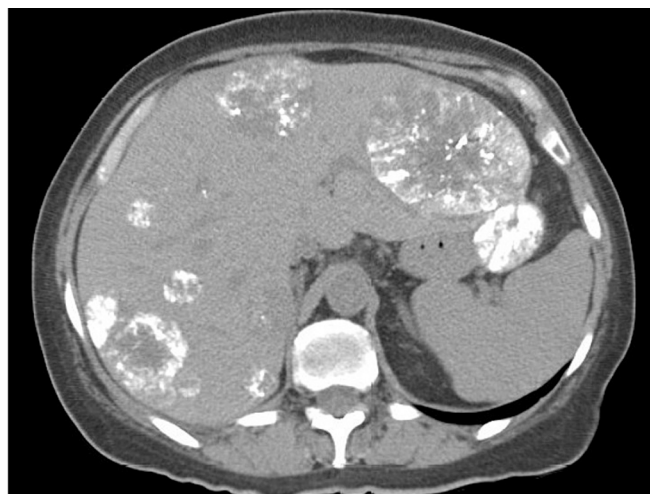


Fig. 9. CT image of the liver after trans-arterial chemoembolization of metastases from colon cancer in this 59-year-old woman demonstrates multiple hyperdense areas of Lipiodol accumulation.

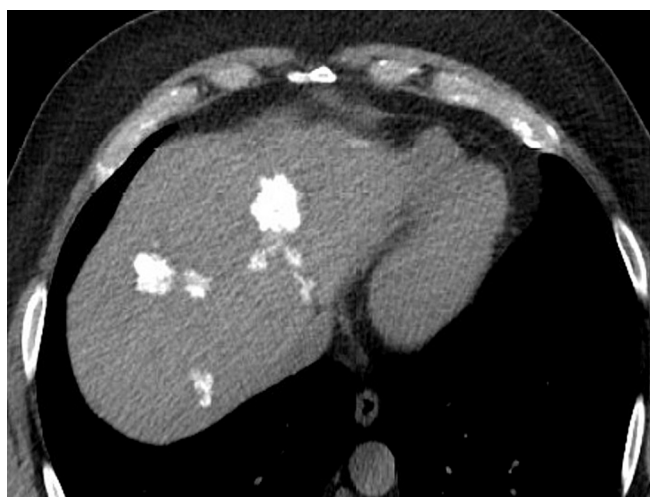


Fig. 10. CT section of the liver shows several densely calcified metastases from a mucinous colorectal carcinoma in this 49-year-old man.

technique to generate a virtual unenhanced image of the liver.^{1,5}

As illustrated in this article, many genetic, acquired, or iatrogenic conditions can be the underlying cause of hyper-attenuated liver (Figs. 1–10). The CT appearance of such dense livers may be very similar despite their different etiologies. This may pose as a diagnostic dilemma if the examining radiologist is unaware of the patient's clinical history. However, the additional CT findings of abdominal organs or osseous structures can be helpful in differentiating the underlying cause of the hyper-attenuated liver.

It is hoped that the herein presented information would enhance the knowledge of practicing radiologists about the prevalence, etiology, and pathogenesis of various entities which can manifest with a hyper-attenuated liver.

Declaration of competing interest

The authors declare that they have no conflict of interest, The authors declare that they have no disclosure relevant to the subject matter of this article.

References

- Kulkarni N, Fung A, Kambadakone AR, Yeh BM. CT techniques, protocols, advancements, and future directions in liver diseases. *Magn Reson Imaging Clin N Am* 2021;29:305–20.
- Boll DT, Merkle EM. Diffuse liver disease: strategies for hepatic CT and MR imaging. *Radiographics* 2009;29:1591–614.
- Hahn L, Reeder SB, Munoz del Rio A, Pickhardt JP. Longitudinal changes in liver fat content in asymptomatic adults: hepatic attenuation on unenhanced CT as an imaging biomarker for steatosis. *AJR* 2015;205:1167–72.
- Costa DMC, Salvadori PS, Monjardim RDF, et al. When the non-contrast-enhanced phase is unnecessary in abdominal computed tomography scans? A retrospective analysis of 244 cases. *Radiol Bras* 2013;46(4). 10.1590.
- Virarkar MK, Vulasala SSR, Gupta AV, et al. Virtual non-contrast imaging in the abdomen and the pelvis: an overview. *Semin Ultrasound CT MRI* 2022;43:293–310.
- Vassalo P, Trohman RG. Prescribing amiodarone: an evidence-based review of clinical indications. *JAMA* 2007;298:1312–22.
- Amiodarone. Drug usage statistics, United States, 2013–2020. In: *ClinCalc DrugStats Database*; 2022.
- Biancatelli RMLC, Congedo V, Calvo L, et al. Adverse reactions of amiodarone. *J Geriatr Cardiol* 2019;16:552–66.
- Lv HJ, Zhao HW. Amiodarone-induced hepatotoxicity- quantitative measurement of iodine density in the liver using dual-energy computed tomography: three case reports. *World J Clin Cases* 2020;8:4958–65.
- Kattamis A, Forni GL, Aydinok Y, Viprasak V. Changing patterns in the epidemiology of beta-thalassemia. *Eur J Haematol* 2020;105:692–703.
- Sayani FA, Kwiatkowski JL. Increasing prevalence of thalassemia in America: implications for primary care. *Ann Med* 2015;47:592–604.
- Gosein M, Maharaj P, Bakaransingh P, et al. Imaging features of thalassemia. *Br J Radiol* 2019;92(1096):20180658.
- Merle U, Schaefer M, Ferenci P, Stremmel W. Clinical presentation, diagnosis, and long-term outcome of Wilson's disease: a cohort study. *Gut* 2007;56:115–20.
- Hedera P. Update on the clinical management of Wilson's disease. *Appl Clin Genet* 2017;10:9–19.
- Akhan O, Akpınar E, Karcaaltincaba M, et al. Imaging findings of liver involvement of Wilson's disease. *Eur J Radiol* 2009;69:147–55.
- Li W, Zhao X, Zhan Q, et al. Unique CT imaging findings of liver in Wilson's disease. *Abdom Imaging* 2011;36:69–73.
- Jafari SH, Haseli S, Kaffashan S, et al. Assessment of the hallmarks of Wilson disease in CT scan imaging. *J Med Imaging Rad Sci* 2020;51:145–53.
- Tavill AS, Adams PC. A diagnostic approach to hemochromatosis. *Can J Gastroenterol* 2006;20:535–40.
- Kowdley KV, Brown KE, Ahn J, Sundaram V. ACG clinical guideline: hereditary hemochromatosis. *Am J Gastroenterol* 2019;114:1202–18.
- Franca M, Carvalho JG. MR imaging assessment and quantification of liver iron. *Abdom Radiol* 2020;45:3400–12.
- Shander A, Cappellini MD, Goodnough LT. Iron overload and toxicity: the hidden risk of multiple blood transfusions. *Vox Sang* 2009;97:185–97.
- Wood JC. Diagnosis and management of transfusion iron overload: the role of imaging. *Am J Hematol* 2007;82:1132–5.
- Fischer MA, Reiner CS, Raptis D, et al. Quantification of liver iron content with CT-added value of dual-energy. *Eur Radiol* 2011;8:1727–32.
- Labranche R, Gilbert G, Cerny M, et al. Liver iron quantification with MR imaging: a primer for radiologists. *Radiographics* 2018;38:392–412.
- Takekawa S, Ueda Y, Hiramatsu Y, et al. History note: tragedy of thorotrast. *Jpn J Radiol* 2015;33:718–22.
- Levy DW, Rindsberg S, Friedman AC, et al. Thorotrast-induced hepatosplenic neoplasia: CT identification. *AJR* 1986;146:997–1004.
- Eisler R. Chrysotherapy: a synoptic review. *Inflamm Res* 2003;52:487–501.
- Bendix G, Bjelle A. A 10-year follow up of parenteral gold therapy in patients with rheumatoid arthritis. *Ann Rheum Dis* 1996;55:169–76.
- De Maria M, De Simone G, Laconi A, et al. Gold storage in the liver: appearance on CT scans. *Radiology* 1986;159:355–6.
- Bartalena T, Rinaldi MF. Hyperdense spleen after prolonged gold therapy. *JMAJCan Med Assoc J* 2010;182(18):E 858.
- Cole LE, Ross RD, Tilley JM, et al. Gold nanoparticles as contrast agents in X-ray imaging and computed tomography. *Nanomedicine* 2015;10:321–41.
- Sun IC, Eun DK, Na JH, et al. Heparin-coated gold nanoparticles for liver-specific CT imaging. *ChemEur J* 2009;15:13341–7.
- Sztandera K, Gorzkiewicz M, Klajnert-Maculewicz B. Gold nanoparticles in cancer treatment. *Mol Pharm* 2019;16:1–23.
- Kantor ED, Rehm CD, Du M, et al. Trends in dietary supplement use among US adults from 1999–2012. *JAMA* 2016;316:1464–74.
- Navarro V, Khan I, Bjornsson E, et al. Liver injury from herbal and dietary supplements. *Hepatology* 2017;65:363–73.
- White JS, Skelly RT, Gardiner KR, et al. Intravasation of barium sulphate at barium enema examination. *Br J Radiol* 2006;79:e32–5.
- Loo GH, Marzuki F, Henry F. Rare, and lethal complication of barium intravasation. *BJR Case Rep* 2018;4(E):20180017.
- Steinman RM, Torres G, Stoupis C. CT of abdominal barium intravasation. *J Comput Assist Tomogr* 1994;18:149–51.
- Peart JM, Sim R. Lipiodol hysterosalpingogram: a modified HSG technique to minimize risks associated with lipiodol use. *J Med Imaging Radiat Oncol* 2020;64: 516–21.
- Jardinet T, Van Veer H, Naftaux P, et al. Intranodal lymphangiography with high-dose ethiodized oil shows efficient results in patients with refractory, high-output postsurgical chylothorax: a retrospective study. *AJR* 2021;217:433–8.
- Roest I, Rosielle K, Van Welie N, et al. Safety of oil-based contrast medium for hysterosalpingography: a systematic review. *ReprodBiomed Online* 2021;42: 1119–29.
- Letzen BS, Malpani R, Miszczuk M, et al. Lipiodol as an intra-procedural imaging biomarker for liver tumor response to transarterial chemoembolization: post-hoc analysis of a prospective clinical trial. *Clin Imaging* 2021;78:194–200.
- De Baere T, Arai Y, Lencioni R, et al. Treatment of liver tumors with lipiodol TACE: technical recommendations from experts' opinion. *Cardiovasc Intervent Radiol* 2016;39:334–43.
- Erley CM, Bader BD, Berger ED, et al. Plasma clearance of iodine contrast media as a measure of glomerular filtration rate in critically ill patients. *Crit Care Med* 2001;29: 1544–50.
- Chou SH, Wang ZJ, Kuo J, et al. Persistent renal enhancement after intra-arterial versus intravenous iodixanol administration. *Eur J Radiol* 2011;80:378–86.
- Patnana M, Menias CO, PickhardtClean PJ, et al. Liver calcifications and calcified liver masses: pattern recognition approach on CT. *AJR* 2018;211:76–86.
- Shibuya A, Unuma T, Sugimoto T, et al. Diffuse hepatic calcification as a sequela to shock liver. *Gastroenterology* 1985;89:196–201.
- Kerlin P, Ash M, Strong R, Mitchell K. Diffuse hepatic calcification. *Br J Radiol* 1988; 61:1079–80.