# MR imaging findings in a patient with hepatic veno-occlusive disease

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## Abstract

**We report the MRI findings in a 31-year-old woman with veno-occlusive disease. MRI demonstrated patent hepatic veins and patchy signal enhancement of the liver after gadolinium chelate injection. This enhancement was compatible with sinusoidal congestion. The diagnosis of veno-occlusive disease was confirmed by histological examination of liver biopsy. The diagnosis of veno-occlusive disease should be evoked when patchy liver enhancement suggestive of sinusoidal congestion is observed in the absence of hepatic vein thrombosis and congestive heart failure.** (Acta gastroenterol. belg., **2004**, 67, **236-238**).

**Key words** : veno-occlusive disease, MR imaging.

## Introduction

Impaired blood outflow from the liver usually results from disorders affecting the hepatic venous tree such as the Budd-Chiari syndrome and veno-occlusive disease (VOD). Hepatic venous outflow block is the best term for these disorders. VOD was originally described as a consequence of toxic injury to the minute hepatic veins by pyrrolizidine alkaloids taken as senecio in medicinal bush tea and has also been associated with various medications (oral contraceptives, cytotoxic agents) and with bone marrow transplantation (1, 2).

The definitive diagnosis of this disease is made by liver biopsy that reveals non-thrombotic, concentric luminal narrowing of intercalated (sub-lobular) veins by loose connective tissue, without obstruction of large-sized hepatic veins.

Imaging methods such as Doppler sonography and MRI usually show non-specific features (3-5). Here, we describe a more specific MRI pattern of hepatic venous outflow block in a patient with histologically proven VOD probably caused by oral contraceptives.

## Case report

A 31-year-old woman was admitted to our hospital because of pain in the right upper quadrant and marked abdominal swelling of recent onset. Her previous medical history was unremarkable except for a long lasting pulmonary infection treated with several antibiotics three months previously. Mycoplasma pneumoniae eventually proved to be the causative agent on the basis of a highly positive serology. There was no history of smoking or alcoholic abuse. Noteworthy, the patient was taking an oral contraceptive (Mercilon®) for several years. Physical examination on admission revealed subicteric mucosa, marked hepatomegaly (20 cm), and abundant ascites. Relevant biological data were : total bilirubin 2.4 mg/dL with 1.8 mg/dL direct bilirubin, alanine aminotransferase 26 U/L, aspartate aminotransferase 26 U/L, alkaline phosphatase 217 U/L (normal value < 94 U/L), and serum albumin 2.4 g/dL. Serology for viral hepatitis A, B, and C was negative as was autoimmune serology. Analysis of the ascites revealed a transsudate. Aminopyrine breath test was markedly decreased at 0.4% (normal > or = 2.8%).

Chest X-ray demonstrated a moderate right-sided pleural effusion. Abdominal sonography revealed a markedly enlarged hyperechoic liver associated with ascites. Doppler sonography showed patent hepatic veins and normal portal flow. MRI showed liver enlargement, ascites, and right pleural effusion. The hepatic veins appeared permeable on the gadolinium-enhanced and the MR angiographic images (true FISP images). Dynamic gradient-echo images after gadolinium chelate injection showed patchy enhancement of the liver, suggestive of sinusoidal congestion (Fig. 1).

Transjugular catheterization revealed a free hepatic pressure of 8 mmHg (normal 2-10 mmHg) and a blocked hepatic pressure pressure of 20 mmHg (normal 4-11 mmHg).

Histological examination of a liver biopsy demonstrated typical lesions of veno-occlusive disease (Fig. 2). Treatment with diuretics (furosemide and spironolactone) was initiated. The subsequent clinical and biological evolution was favourable. Aminopyrine breath test was nearly normal six months after admission (2.5%). At that time, the clinical examination and the liver function tests were unremarkable. A control MR examination showed a liver of normal size with nearly normal enhancement. Only slight heterogeneities persisted during the portal phase in the right posterior segment (segment 7). The ascites and pleural effusion had totally disappeared.

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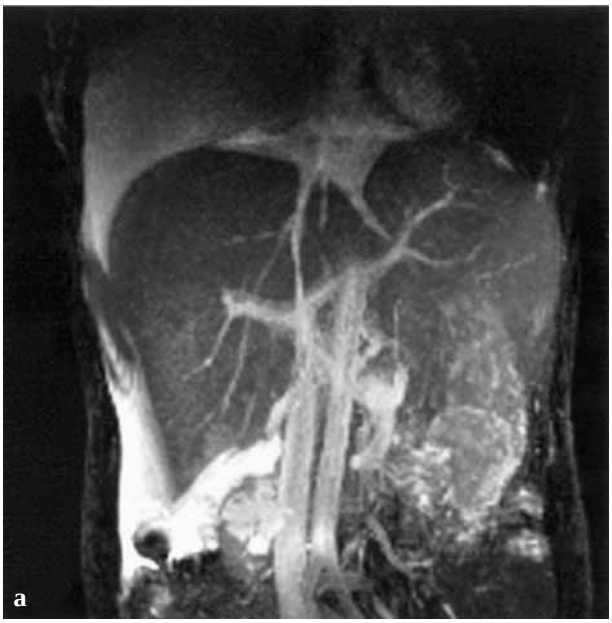


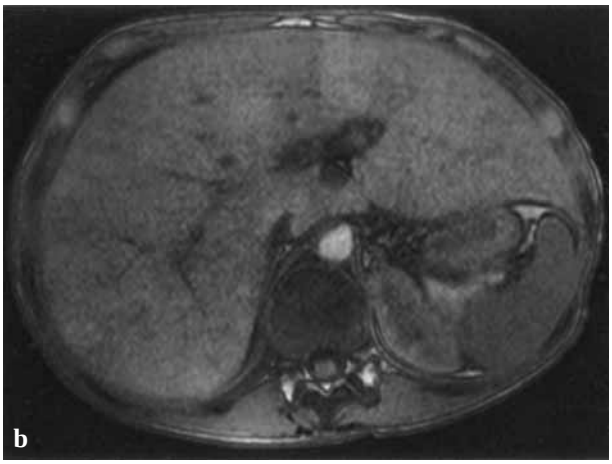


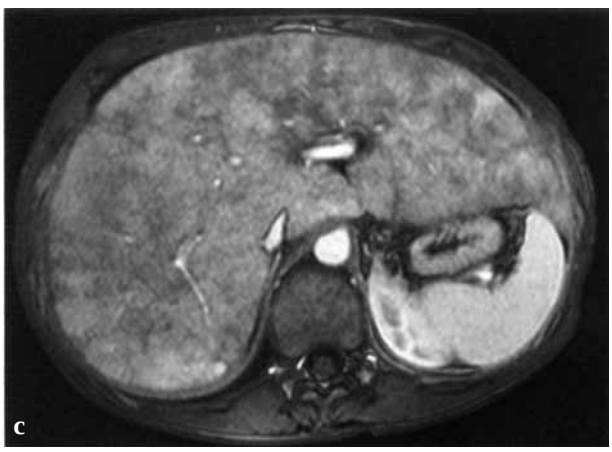


Fig 1. — (a) Coronal MR angiographic image shows hepatomegaly, ascites, right pleural effusion, and the confluence of patent hepatic veins with inferior vena cava. T1-weighted gradient-echo images before (b) and after (c) injection of a gadolinium chelate show patchy hepatic enhancement, suggestive of sinusoidal congestion, and central bright spots corresponding to patent branches of hepatic veins.

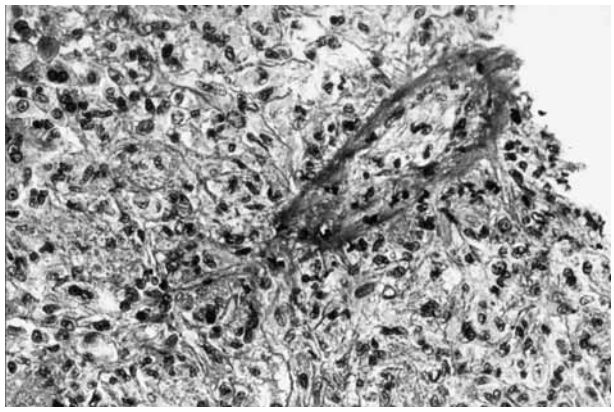

Fig. 2. — Liver biopsy (HE staining-obj 40×) showing non-thrombotic obliteration of a centrolobular vein by a fibro-oedematous material.

## Discussion

We report a case of VOD with spontaneous favourable evolution. In this patient, the etiology of VOD is uncertain. To the best of our knowledge, no relationship between VOD and Mycoplasma pneumoniae infection has been described. The most likely cause remains the oral contraceptive, but the potential role of the various antibiotics given previously cannot be totally excluded.

The diagnosis of VOD remains difficult. In our case, the diagnosis was confirmed by liver biopsy. However, MRI was contributory by showing patchy enhancement suggestive of sinusoidal congestion on gadolinium-enhanced images. Patchy liver enhancement has recently been reported in another patient with VOD (6). A similar enhancement pattern at MRI and CT has been reported in patients with Budd-Chiari syndrome and congestive heart failure (7-11). In our patient, MR angiography and contrast-enhanced MR images showed normal patency of the large hepatic veins arguing against the diagnosis of Budd-Chiari syndrome. In the appropriate clinical context, the patchy liver enhancement and the permeability of the hepatic veins observed in this patient may thus be specific features suggesting the diagnosis of VOD at MRI. Only few cases of VOD documented by MRI have been described (4, 6). The reported features are mainly hepatomegaly and compression of the hepatic veins. Gallbladder wall thickening, marked hyperintensity of the gallbladder wall on T2-weighted images, ascites, pleural effusion, and periportal cuffing have also been described. These signs are nonspecific and are also observed in patients with hypoalbuminemia of various causes. The features described at Doppler sonography in patients with VOD also lack specificity and mainly reflect portal hypertension. These signs include decrease in portal velocity, inversion of portal flow, increase in hepatic artery resistive index, and increase in thickness of the gallbladder wall (3, 5, 12).

In conclusion, patchy liver enhancement suggestive of sinusoidal congestion in the absence of hepatic vein thrombosis and congestive heart failure should suggest the diagnosis of VOD. The reason why this sign has not been described more often remains speculative. Patchy enhancement in VOD might be transitory as it has been described in Budd-Chiari syndrome.

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