Eur. Radiol. 7, 10–16 (1997)

European Radiology

Original article

Effects of lipiodol retention on MRI signal intensity from hepatocellular carcinoma and surrounding liver treated by chemoembolization

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Received 21 June 1995; Revision received 22 January 1996; Accepted 24 January 1996

Abstract. Opinion is divided regarding the influence of iodized oil on MRI signal intensity of hepatic tumours treated with transcatheter arterial chemoembolization (TACE), in which lipiodol deposits. The aim of our study was to ascertain whether or not lipiodol directly influences the MRI signal intensity of hepatocellular carcinoma (HCC) treated by TACE and that of the surrounding liver. Thirteen patients with HCC were studied retrospectively. CT and MRI scans were performed both before and 3 months after TACE. The CT scan was performed to check whether embolized nodules contained lipiodol and how lipiodol was distributed within them. In addition, eight patients were examined prospectively within 7 days after TACE. In these patients a CT scan was performed to see how lipiodol was distributed in the neoplastic nodules and in normal hepatic parenchyma. In the first group of patients the contrast-to-noise (C/N) ratio on T1-weighted (T1W) images and the T2 relaxation time on T2-weighted (T2W) images were calculated for both neoplasm and surrounding liver. In the second group of patients we also measured the signal intensity of non-neoplastic liver that was either permeated or not permeated by lipiodol. The data were analysed with Wilcoxon's test. On T1W images we observed that the retention of lipiodol increased the C/N ratio in all the tumours studied within 1 week after TACE. In the patients studied 3 months after TACE the C/N ratio was not significantly increased. On T2W images lipiodol retention did not change tumour signal intensity. The iodized oil did not change the signal intensity of the liver surrounding the tumour, in comparison with the liver not permeated by lipiodol, on either T1W or T2W images. The results indicate that lipiodol does not modify the signal intensity in non-neoplastic hepatic parenchyma in which it is deposited; after 3 months it does not significantly affect the signal of the tumours that accumulated it. Lipiodol produces a high signal on T1W images over the first few days following TACE in those tumours in which it is deposited.

Key words: Liver, neoplasm – Magnetic resonance imaging – Contrast media, fatty acid

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Introduction

Transcatheter arterial chemoembolization (TACE) is a widely used technique in the non-surgical treatment of hepatocellular carcinoma (HCC) [1, 2]. It requires the combined use of iodized oil (lipiodol; Guerbet, Aulnay-sous-Bois, France), anti-blastic drugs and gelatin sponge particles. Lipiodol deposits in the tumour and then is slowly released [3, 4]. MRI is increasingly being used for the diagnosis and follow-up of HCC treated by TACE [5, 6]. Opinion is divided regarding the influence of lipiodol on MRI signal intensity of hepatic tumours in which it is deposited [7, 8]. However, no study has been conducted that systematically evaluates the effects of lipiodol on signal intensity in both embolized HCC and surrounding hepatic parenchyma. The aim of our study was to ascertain whether or not lipiodol directly influences the MRI signal of HCC treated by chemoembolization and of the surrounding liver.

Methods

We examined retrospectively patients who had undergone TACE for HCC during the past 2 years. Among these patients we selected those who could be examined with MRI both prior to the 3 months after TACE.

The MR examinations used in the investigation had identical procedural characteristics such as repetition time (TR), echo time (TE), field of view (FOV), matrix, slice thickness and number of excitations (nex). Examinations were performed on a 1.5 T superconductive sys-

tem (Signa; General Electric, Milwaukee, Wis.), using SE T1-weighted (T1W) images (TR 400 ms, TE 20 ms, 128 × 256 matrix, 4 nex, 360 FOV) and T2-weighted (T2W) images (TR 2000 ms, TE 80 ms, 128 × 256 matrix, 2 nex, 360 FOV). The sections imaged were 8 mm thick, with an interslice gap of 1.5 mm. Respiratory compensation was used to reduce respiratory artefacts.

Thirteen patients (10 men and 3 women, 52–69 years old) fulfilled the entry criteria and were selected for the study. Eight other patients (7 men and 1 woman, 50–72 years old) with HCC were also selected; this group was studied prospectively with the use of MRI prior to their TACE treatment. MRI was repeated within 7 days following TACE, and 3 months after in four cases, always using the same scan parameters already described (TR, TE, FOV, matrix, nex, slice thickness).

All patients suffered from post-hepatitic cirrhosis as determined at liver biopsy. According to Child's classification is patients were class A and six were class B at the initial TACE. HCC was diagnosed by percutaneous tissue core biopsy under ultrasonographic control in each patient.

A requirement for this study was that all lesions were detectable by MRI before TACE. MRI detected 30 tumours in 21 patients. The tumours ranged in size from 2 to 8 cm. We do not have histological proof of each nodule detected by MRI, because in the patients with multiple lesions only one tumour underwent biopsy. To exclude lesions other than HCC that are common in cirrhotic liver, such as regenerative nodules or adenomatoid hyperplasia, we only evaluated those lesions which had a tumoral stain at angiography.

All patients in both the first and second groups underwent a CT scan on the same day as the post-TACE MRI examination.

CT scans were performed within 7 days of TACE in the eight prospectively studied patients, in order to see how lipiodol was distributed in the neoplastic nodules as well as in normal hepatic parenchyma and to determine which areas of the hepatic parenchyma had been permeated with lipiodol and which had not. CT scans performed 3 months after TACE were performed to check whether embolized nodules contained lipiodol and how lipiodol was distributed within them. Tumours which did not contain lipiodol or in which lipiodol fixation was not homogeneous were excluded from the study.

TACE was performed by selectively introducing a catheter into the right or left hepatic artery and injecting a mixture of 8–15 ml of lipiodol and doxorubicin hydrochloride (Adriblastina; Farmitalia; Carlo Erba; Italy) 0.6–1 mg/kg body weight and gelatin sponge particles (Spongostan, Ferrosan, Denmark). TACE was never performed on the entire liver.

The aim of MRI was twofold: (1) to observe variations in signal intensity of the embolized neoplastic nodules proved to contain lipiodol by the CT scans in the first week and after 3 months; (2) to observe the variations in signal intensity of non-neoplastic hepatic parenchyma permeated with lipiodol, within 7 days after TACE.

T1W and T2W images were analysed separately. For pre-TACE T1W images we calculated signal intensity by using a region of interest (ROI) in the tumour and non-tumoral liver which exhibited lipiodol permeation during the CT scan after TACE. ROI dimensions, as defined by the operator, were identical for each patient. When the tumour was visible in at least three contiguous slices, the ROI was calculated on the middle slice so as to avoid partial volume effects. When the tumour was visible in only one or two slices, the ROI was calculated on the middle portion of the tumour or in two contiguous areas, and the results averaged.

The ROI for the hepatic parenchyma was calculated in a central hepatic zone not intersected by large vessels. Furthermore, noise standard deviation was measured anteriorly to the patient, by using the largest possible ROI.

For the MR images obtained within the first week and 3 months after TACE, we proceeded in the same manner, taking care to measure tumour signal intensity after examining lipiodol CT images, performed on the same day, in order to place the ROI in tumoural areas definitely permeated with lipiodol. Non-neoplastic hepatic parenchyma ROI were located in identical areas of parenchyma on pre- and post-TACE MR images.

For T1W images we calculated the following:

1) Contrast-to-noise (C/N) ratio of lesions prior to and 3 months after TACE, according to the formula [(SA–SB)/noise SD], where SA is the measured signal intensity of the lesions, SB is the measured signal intensity of the liver and noise SD is the standard deviation of the intensity of background noise [9].
2) Lesion C/N ratio prior to and within the first week of embolization according to the aforementioned formula, taking care to measure liver signal intensity in parenchymal areas proven free of lipiodol during the CT scan.
3) Signal-to-noise (S/N) ratio of the non-neoplastic liver, proven to be permeated with lipiodol by CT performed after TACE, prior to and within 7 days after TACE, and the S/N ratio of non-neoplastic liver not permeated with lipiodol within the first week after TACE, for comparison, according to the formula [S/N = SI/noise SD], where SI is the measured signal intensity of the liver and noise SD is the standard deviation of the intensity of background noise. For each patient this was calculated on two distinct areas of parenchyma: one corresponding to zones subsequently permeated with lipiodol after TACE and the other, for comparison, non-embolized parenchyma of the right or left hepatic lobe that was therefore not permeated with lipiodol after TACE.

The following formula was used in the pre-TACE comparison: A/B, where A is the S/N ratio of pre-TACE hepatic parenchyma which was permeated with lipiodol after TACE, and B is the S/N ratio of the non-permeated hepatic parenchyma. For the post-TACE comparison the formula is C/D, where C is the S/N ratio of post-TACE hepatic parenchyma permeated with Lipiodol and corresponding to A, and D is the S/N ratio of

non-lipiodol-permeated hepatic parenchyma corresponding to B.

For T2W images we calculated T2 relaxation time (in ms) both of the tumour and of the non-tumoral, non-lipiodol-permeated and lipiodol-permeated liver, prior to, within the first week, and 3 months after TACE by setting a ROI with the same methods described for T1W images, using long TR sequences (2000 ms) and at least two echo images of 20–80 ms or 30–80 ms, by means of a specially designed software program.

Finally, we measured the signal intensity and C/N ratio of pure lipiodol compared with the liver and its T2 relaxation time, with the same methods described for HCC, by using a phial of lipiodol placed over the skin of two patients, in the right hypochondrium.

All data were analysed with Wilcoxon's test and a p value < 0.05 was considered significant.

Results

We evaluated with MRI 30 neoplastic lesions in 17 patients prior to and 3 months after TACE. In 16 patients lipiodol CT detected 39 lesions; 9 lesions smaller than 1 cm were not detected by prior MRI examination and, as stated in Methods, these nodules were excluded from the study. In one patient (Fig. 2) lipiodol CT showed dozens of very small lesions missed at MRI before TACE.

On SE T1W images before TACE 16 tumours (53 %) were hyperintense, 8 (27 %) were isointense and 6 (20 %) were hypointense. Pseudocapsules were seen on T1W images in 8 tumours. On T1W images 3 months after TACE 8 tumours (26 %) were hyperintense, 17 (58 %) were isointense and 5 (16 %) were hypointense. On SE T1W images, 13 lesions showed an increase in the C/N ratio (Fig. 1), 14 lesions showed a reduction in the C/N ratio (Fig. 2) and 3 lesions showed an identical C/N ratio before and 3 months after TACE (Fig. 1). There was no statistically significant difference between the three groups (p = 0.8759) (Table 1).

On SE T2W images before TACE all the tumours were hyperintense; 25 nodules showed a reduction in T2 relaxation time in examinations performed 3 months after TACE (Fig. 3), 4 nodules showed an increased T2, and in 1 lesion T2 remained the same before and after TACE. Wilcoxon's test demonstrated a statistical significance for these data of p = 0.0003 (Table 2).

Eight HCCs in the same number of patients were examined prior to and within 1 week after TACE. On CT scan lipiodol fixation was complete and homogeneous in each tumour. T1W images in all cases after TACE showed an increased C/N ratio (Fig. 3). Wilcoxon's test showed a statistical significance of p = 0.0117 (Table 3). Four of these 8 patients were examined again by MRI 3 months after TACE; in all 4 cases the C/N ratio was not increased for embolized nodules compared with the pre-TACE test (Fig. 3).

On T2W images in 5 cases we observed a reduction of the T2 relaxation time and in 3 cases an increase of T2 with, however, no statistical significance (p = 0.1235) (Table 4).

Ten patients died within 12–24 months from the growth of HCC. Two of these showed neoplastic portal thrombosis during the follow-up. Two patients died within 1 year from progressive liver cirrhosis. Nine pa-

Table 1. Variations in contrast-to-noise ratio (C/N) on SE T1-weighted images in hepatocellular carcinoma 3 months after TACE

No. of patients	No. of lesions	Change in C/N		
		Increase	Decrease	No change
17	30	13	14	3

p = 0.8759 (NS)

Table 2. Variations in T2 relaxation time in hepatocellular carcinoma 3 months after TACE

No. of patients	No. of lesions	Change in T2		
		Increase	Decrease	No change
17	30	4	25	1

p = 0.0003

Table 3. Variations in the contrast-to-noise ratio (C/N) on SE T1-weighted images in embolized hepatocellular carcinomas by the first week after TACE

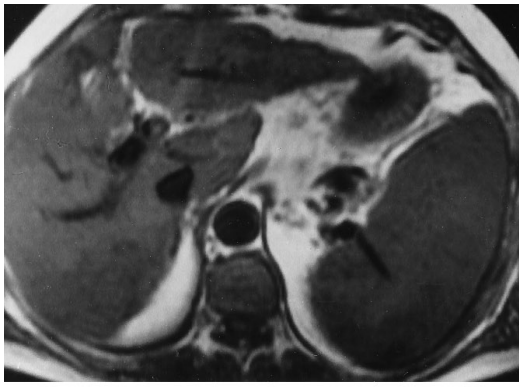
No. of patients	No. of lesions	Change in C/N		
		Increase	Decrease	No change
8	8	8	0	0

p = 0.017

Fig. 1. **a** The T1-weighted (T1W) image before transcatheter arterial chemoembolization (TACE) does not show any lesion. **b** CT scan 3 months after TACE shows dense accumulation of lipiodol in two tumours *(arrows)*. **c** The T1W image 3 months after TACE show that one lesion is hyperintense *(arrow)* with an increase in the contrast-to-noise (C/N) ratio relative to MR images before TACE and the second one is isointense, with the same C/N ratio relative to MR examination before TACE

Fig. 2. **a** The T1W image before (TACE) shows many hyperintense encapsulated hepatocellular carcinomas. **b** CT scan 3 months after TACE shows lipiodol retention in many lesions. **c** The T1W image 3 months after TACE shows that one lesion is decreased in size and isointense relative to surrounding liver *(arrows)*, with a reduction in the C/N ratio compared with the MR image before TACE; the other tumours are decreased in size but still hyperintense

Fig. 3. **a** The T1W image before TACE shows a large hypointense tumour in segment 4 *(arrows)*. **b** The tumour is hyperintense on the T2W image *(arrows)*. **c** On the T1W image 4 days after TACE the tumour is iso-hyperintense relative to surrounding liver, with an increase in the C/N ratio compared with the MR image before TACE. **d** On the T2W image the tumour is still hyperintense but shows hypointense areas in the centre. Three months after TACE the tumour is decreased in size and is hypointense on both the T1W image (**e**) *(arrow)* and T2W image (**f**) *(arrow)*. **g** CT scan 3 months after TACE shows complete retention of lipiodol in the tumour *(arrow)*



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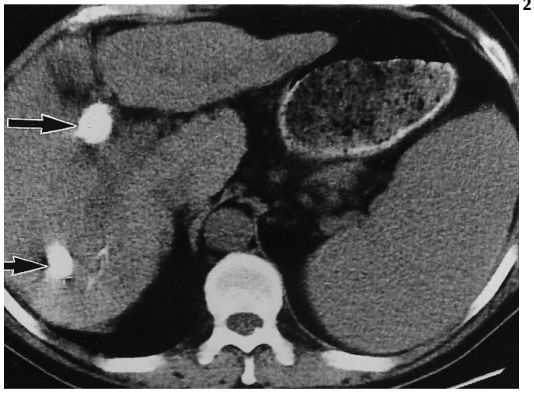

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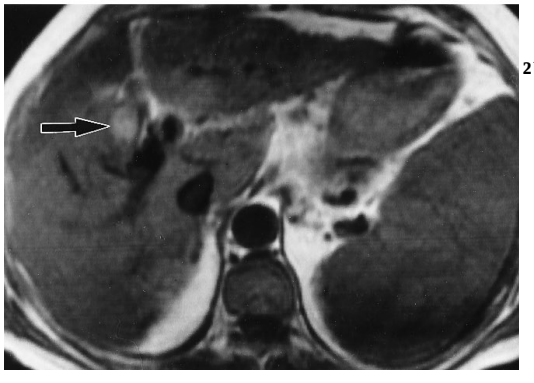

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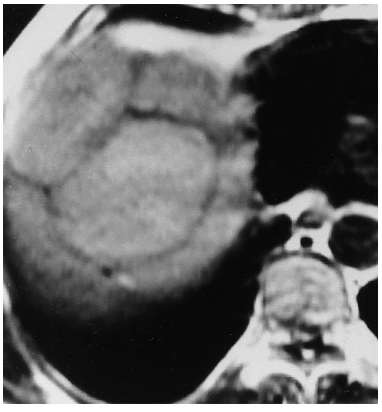

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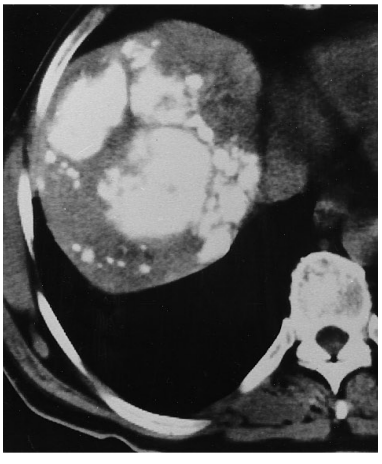

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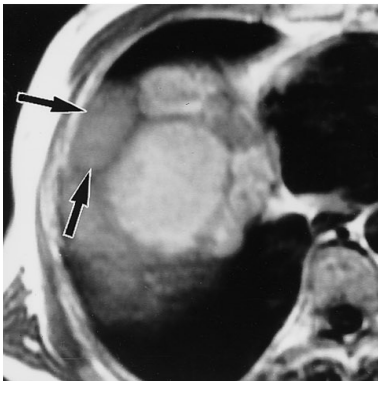

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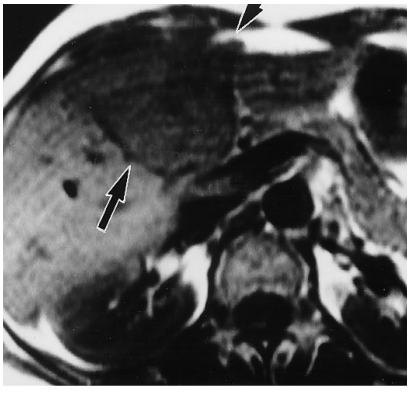

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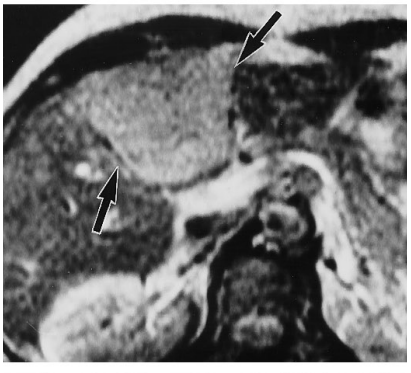

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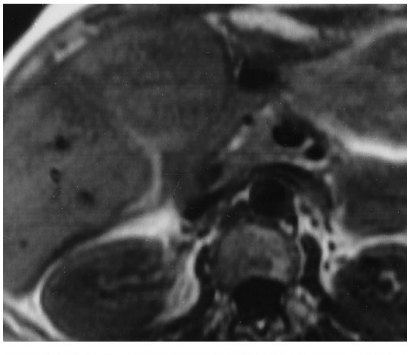

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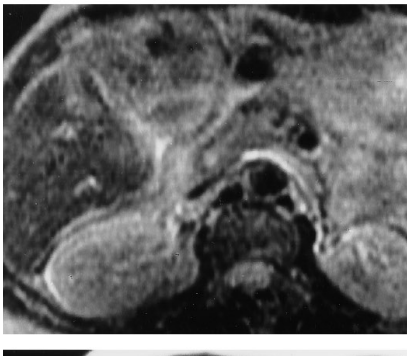

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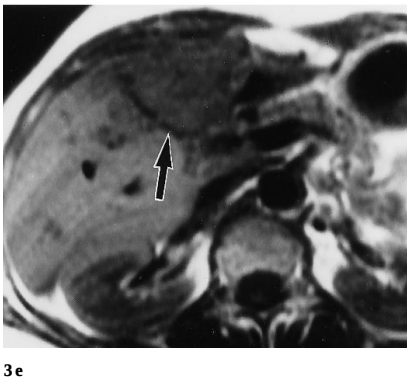

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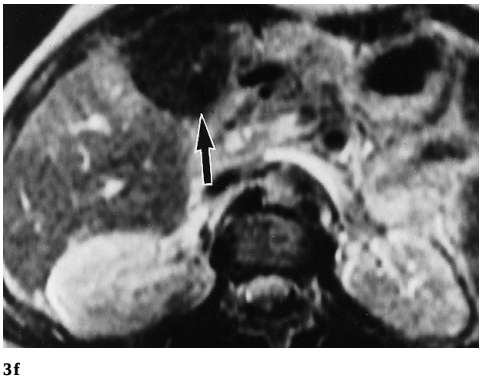

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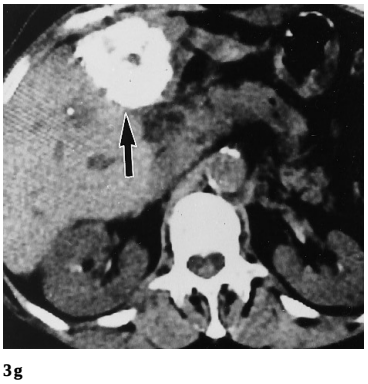


3g

tients are still alive; 7 of these underwent repeated treatment because of clinical and MRI findings of recurrent tumour. Three patients are currently tumour-free.

At 7 days and 3 months after TACE the changes in tumour signal intensity on T1W and T2W images were not predictive factors for survival, but in the patients who are still alive, a reduction of the C/N ratio on T1W images and a reduction in T2 relaxation time of the nodules were predominant findings.

In 8 patients within the first week after TACE we compared the S/N ratio of non-neoplastic lipiodol-permeated hepatic parenchyma with that of non-neoplastic hepatic parenchyma not permeated with lipiodol. In 4 cases T1W images showed a reduction in signal intensity compared with images performed prior to TACE, in 1 case an increased of signal and in 3 cases no difference in signal intensity (Fig. 4; Table 5). In 5 cases on T2W images we observed no variation in T2 relaxation time, in 1 case a reduction and in 2 cases an increase (Table 6).

Both for signal intensity of T2W images and for T2 relaxation times we observed no statistically significant difference between tests performed before TACE and those performed the first week after TACE.

The S/N ratio on SE T1W images of pure lipiodol was very high. The T2 relaxation time of pure lipiodol was 32 ms, less than that of cirrhotic liver hepatic parenchyma (average 38 ms in our patients) and of subcutaneous fatty tissue (44–46 ms).

Table 4. Variations in T2 relaxation time in embolized hepatocellular carcinoma by the first week after TACE

No. of patients	No. of lesions	Change in T2		
		Increase	Decrease	No change
8	8	3	5	0

$p = 0.1235$ (NS)

Table 5. Variations in the signal-to-noise ratio (S/N) on SE T1-weighted images of hepatic parenchyma permeated with lipiodol as compared with non-lipiodol-permeated parenchyma

No. of patients	Change in S/N		
	Increase	Decrease	No changes
8	1	4	3

$p = 0.345$ (NS)

Table 6. Variations in the T2 relaxation time of hepatic parenchyma permeated with lipiodol as compared with non-lipiodol-permeated parenchyma

No. of patients	Change in T2		
	Increase	Decrease	No change
8	2	1	5

$p = 1.00$ (NS)

Discussion

MRI is increasingly being used in patients for the follow-up of HCC after TACE [10, 11]. One reason why MRI is preferred to CT is the presence of artefacts in the latter due to the high density of lipiodol concentrated in the tumour, which can make it very difficult to recognize a possible recurrence of the disease [11], even though lipiodol CT is fundamental for recognizing lesions smaller than 1 cm, missed at MRI. Moreover, there is proof that lipiodol does not modify the MRI signal intensity of the tumour in which it deposits [7, 8]. Buckwalter et al. [12] state that ethiodol (ethiodized oil, Abbot, North Chicago, Ill.), a liposoluble iodate contrast medium, when used in lymphangiography, modified lymph node signal by reducing T1 and increasing T2 relaxation times, thus making them indistinguishable from those of subcutanous fatty tissue.

Pantopaque (iophendilate, Lafayette Pharmacal, Lafayette Ind.) is an oil iodate used as a contrast agent for myelography; it gives high signals due to a short T1 relaxation time, which may be mistaken for fat or haemorrhaging in the subarachnoid space on MRI [13].

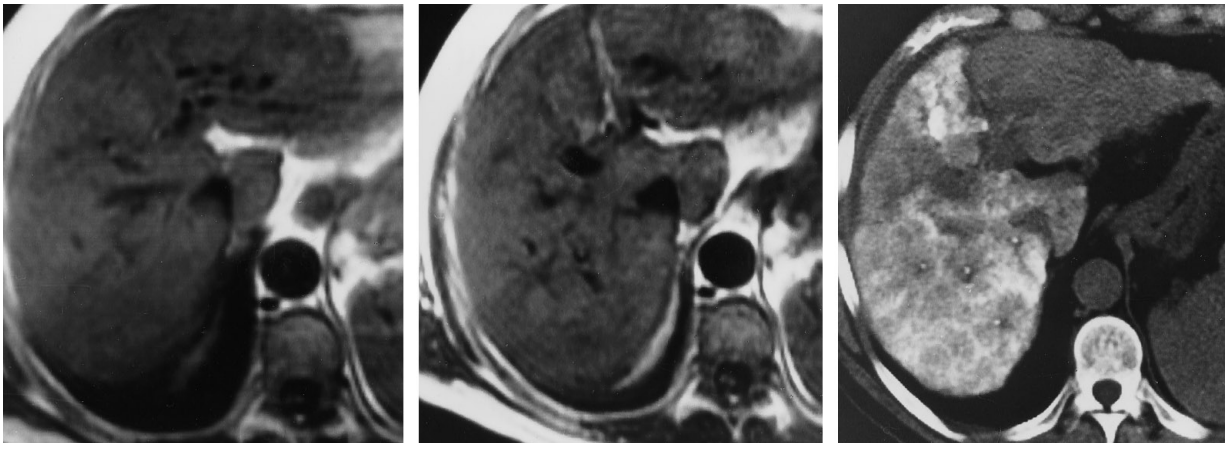
If lipiodol had the same effects on hepatic parenchyma and the HCC in which it accumulated, we would have obtained an increase in both T1 and T2 signals. Lipiodol is a mixture of ethyl esters of iodurate fatty acids of plant origin. The pure lipiodol used in the tests we conducted showed a high signal intensity on T1W images (higher than that of subcutaneous fatty tissue) and a low signal intensity on T2W images (lower than that of subcutaneous fatty tissue). In our patients, lipiodol distributed in the hepatic parenchyma in the first days following TACE did not change the hepatic signal intensity on either T1W or T2W images. This is probably due to the low concentration of lipiodol relative to the volume of tissue to be studied.

The increased signal intensity we observed on T1W images within the first week following TACE in all neoplastic nodules permeated with lipiodol, which was highly statistically significant, indicates that highly concentrated lipiodol in tissue causes a reduction in the T1 relaxation time and increased signal intensity.

Based on the observation that the T2 relaxation time of pure lipiodol is lower than that of hepatic parenchyma, and consequently of HCC, we would have expected a reduction in the T2 relaxation time in all tumours examined within the first week following TACE. Instead, we observed this only in 5 of 8 cases, while in the remaining cases we observed non-significant increase in T2.

It is difficult to establish whether this is due to the missed effect of lipiodol on T2 signal intensity in tumours in which it has accumulated or to early concomitant modifications brought on by TACE to the tumour, such as ischaemia and coagulative or colliquative initial necrosis, or to intratumoral haemorrhage or oedema.

In the MRI study obtained 3 months after TACE, we observed on T1W images no statistically significant difference in the C/N ratio of embolized neoplastic nodules containing lipiodol compared with pre-TACE values; the C/N ratio increased in 13 of 30 patients, was reduced



a **b** **c**

Fig. 4 a, b. The T1W images before (**a**) and 1 day after TACE (**b**) show no difference in the signal intensity of liver parenchyma. **c** CT scan performed 1 day after TACE shows lipiodol distribution in the liver

in 14 and remained the same in 3 patients. Moreover, it was interesting that 4 of the 8 nodules with an increase in the C/N ratio 7 days following TACE showed a reduction in the C/N ratio compared with the pre-TACE test when checked 3 months later. These data allow us to assume that either lipiodol does not affect signal intensity 3 months after TACE or that it is only one of several factors, alongside modifications induced by TACE in tumoral tissue.

Type of lipiodol fixation is a good predictive factor for determining the effect of TACE. Complete retention of iodized oil in encapsulated tumour demonstrated the best therapeutic effect [14].

On MRI HCCs that showed decreased signal intensity on both T1W and T2W images after TACE did not recur [8]. In this study the changes in C/N ratio on T1W images, or the T2 relaxation time within 7 days or 3 months after TACE, were not predictive factors for survival, even if the patients with the longer survival showed a reduction of both the C/N ratio on T1W images and the T2 relaxation time on MRI 3 months after TACE.

Lipiodol retention on the CT scan and T2 relaxation time on MRI are well correlated with necrosis of the tumour [14, 15], but necrosis is only one of the many factors that determine the survival of the patient.

On T2W images 3 months following TACE, we observed a reduction in the T2 relaxation time in 25 of 30 nodules, with high statistical significance. We do not believe that this result is due to the effect of lipiodol on the T2 of the tumour in which it accumulated, but rather to the coagulative necrosis induced by TACE [15, 16]. Similar results have been described following percutaneous ethanol injection therapy [17, 18].

In conclusion, we believe that lipiodol does not modify signal intensity in the non-neoplastic hepatic parenchyma in which it has deposited; furthermore, after 3 months it does not significantly affect the signal of the tumoral nodules that accumulated it. Instead it produces a high signal on T1W images over the first days following TACE in those tumours in which it deposited.

Given that MRI is used to determine the effects of TACE usually 2–3 months after the procedure, we believe that after this period of time lipiodol does not cause a significant modification of signal intensity of embolized neoplastic nodules.

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Book review

European Radiology

Bruel J.-M., Lopez F.-M.: Imagerie et Urgences. Flammarion Paris: Médecine-Sciences, 1996, ISBN: 2-257-15539-4 Language: French, 363 pages, approximately 650 illustrations, 685 FF

This book is a nicely illustrated brief and schematic survey of medical imaging in different emergencies. Conventional films, CT, MRI, and angiographic images are given to illustrate the most common conditions. Each chapter includes traumatic and non-traumatic emergencies.

The first chapter illustrates the brain and skull. It starts with a short review of the indications of imaging, and a short introduction about film reading. Then the authors give a nicely illustrated survey of the most common traumatic conditions, including subdural hematoma, epidural hematoma, intracerebral hematoma, subarachnoidal bleeding, etc. Next, the non-traumatic conditions are illustrated, including stroke, tumors, and infectious diseases of the brain.

The second chapter deals with head and neck emergencies, also including traumatic and non-traumatic conditions. Special attention is given to the complications of endoscopic surgery of the sinuses. Infections of the head and neck are nicely illustrated, including their complications.

The next chapter discusses emergencies of the spine. The traumatic conditions are described first, including a review of medical imaging in traumatic conditions, correlated with the clinical state of the patient. A few examples are shown. As non-traumatic conditions tumoral and discal cord compression and infectious diseases are discussed.

The fourth chapter is about thoracic emergencies. It starts with a short review of semiologic signs in chest imaging. Next, the most common traumatic conditions, including pneumothorax, pneumomediastinum, lung contusion, and lesions of the chest wall, are illustrated with conventional imaging and CT and a few ultrasound images. The angiographic imaging of aortic rupture is shown. Also, cardiac trauma is illustrated. The differential diagnosis and further workup in causes of traumatic pleural fluid is discussed. Next, diving and drowning accidents, and toxic fume inhalation, are described. A few words are written about chest tubes. Non-traumatic conditions include chest wall diseases, pleural and pulmonary diseases, including tumors and infections, pleural fluid, coronary diseases, aortic dissection and aortic aneurysm, a few conditions causing dyspnea, and emergencies in the postoperative chest.

The fifth chapter deals with abdominal emergencies. It also starts with a semiologic survey on plain films and ultrasound, followed by a review of the traumatic conditions. Plain films, CT, and ultrasound images of parietal, peritoneal, and retroperitoneal traumatic collections are shown. Also, traumatic lesions of the abdominal organs, bowel, and vessels are discussed. Next, the possible locations of foreign bodies are demonstrated. Non-traumatic conditions including gall bladder diseases, renal diseases, acute appendicitis, Crohn's disease, diverticulitis, adnexitis, peritonitis, free abdominal air, pancreatitis, and mesenterial infarctions are discussed, divided by the location of pain. A note of attention is given to intra- and retroperitoneal bleeding and to the female pelvis. Obstruction and gastrointestinal bleedings are discussed more in detail.

The last chapter is about emergencies in the extremities and also discusses traumatic and non-traumatic conditions. Traumatic conditions include fractures and luxations, but also labral tears of the shoulder, meniscal tears and cruciate ligament tears of the knee. Vascular and muscular damage is discussed. Non-traumatic conditions include thrombovascular diseases and infectious diseases including osteomyelitis, arthritis, and tendonitis.

In conclusion, I assert that this book is a nice survey with a lot of figures (for a relatively low price) of traumatic and non-traumatic conditions seen in the emergency room.

E. Geusens, Leuven