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MDCT findings after hepatic chemoembolization with DC-beads: What the radiologist needs to know

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Abstract

Transcatheter arterial chemoembolization with drug-eluting beads (TACE-DC-beads) is a new local treatment for primary or metastatic liver tumors. Despite technical efforts to achieve highly selective embolization of the tumor-supplying vessels, small, or large insults to the non-tumorous parenchyma are inevitably induced by the embolic materials or procedure itself. Parenchymal changes following TACE-DC-beads include bile duct injuries (bile duct dilatation, periportal edema, and bilomas), obliteration of intrahepatic portal vein branches, hypodense ill-defined areas, and perilesional parenchymal enhancement. The radiologist must be familiar with the changes induced by this treatment in order to distinguish therapeutic effect and collateral findings from complications and residual or recurrent tumor.

Key words: Transcatheter arterial chemoembolization—Drug-eluting bead—Liver tumors—Biloma—Bile duct injury

Transcatheter arterial chemoembolization (TACE) has been used widely to treat hepatocellular carcinoma and other primary or metastatic tumors of the liver. This technique either with or without drug-eluting beads combines local and targeted drug delivery with concurrent tumor-feeding artery embolization, offering the ability to expose tumors to high local chemotherapeutic agent concentrations with minimal systemic drug bioavailability favoring induction of tumor necrosis [1, 2].

Conventional-TACE is performed by injecting an emulsion of the drug and iodized oil (lipiodol) and then the embolic material [3, 4]. Drug-eluting beads (DC-beads) loaded with chemotherapy have recently been developed to increase the intensity and duration of blood flow blockage to the target tissue while delivering a local and sustained dose of drug directly to the tumor [5].

The most common complication after traditional TACE is the postembolization syndrome that consists of transient abdominal pain, fever, and laboratory changes lasting few days [6]. Other TACE-related complications occur infrequently and include liver failure, renal impairment, ischemic cholecystitis, hepatic abscesses, and biliary strictures [6, 7].

Bile duct injury including subcapsular biloma, focal strictures of hepatic bile ducts and diffuse dilatation of the intrahepatic ducts, has been reported with a 0.5–2% incidence after conventional TACE [8]. Despite technical efforts to achieve highly selective embolization of the tumor-supplying vessels, small or large insults to the non-tumorous parenchyma are inevitably induced by the embolic materials or procedure itself (Table 1).

The purpose of this pictorial essay is to describe the spectrum of MDCT findings and parenchymal changes detected in patients with primary or metastatic liver tumors treated with transcatheter arterial chemoembolization with drug-eluting beads (TACE-DC-beads).

Bile duct injury changes

It has been suggested that small embolic material diameter and repeated TACE procedures are related to ischemic bile duct injuries with or without chemical arteritis of the small vessels supplying the bile duct wall [9]. In contrast to normal liver parenchyma, the

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Table 1. Findings at follow-up CT after TACE-DC-beads

Bile duct dilatation
Periportal edema
Obliteration of adjacent intrahepatic portal vein branches
Bilomas
Parenchymal hypodense ill-defined areas
Parenchymal infarcts
Abscess
Perilesional parenchymal enhancement
Parenchymal atrophy
Tumoral necrosis
Tumoral residual enhancement
Intratumoral air

intrahepatic bile ducts do not have a dual blood supply and are fed exclusively from the hepatic arterial branches that give off a vascular plexus (peribiliary capillary plexus) around the bile ducts (Fig. 1). Therefore, ischemia of the intrahepatic bile ducts can easily occur after chemoembolization [10], causing bile duct strictures and dilatation. Bile duct injury changes are represented as bile duct dilatation, periportal edema, and bilomas, probably reflecting liver damages that occur gradually over time (Fig. 2).

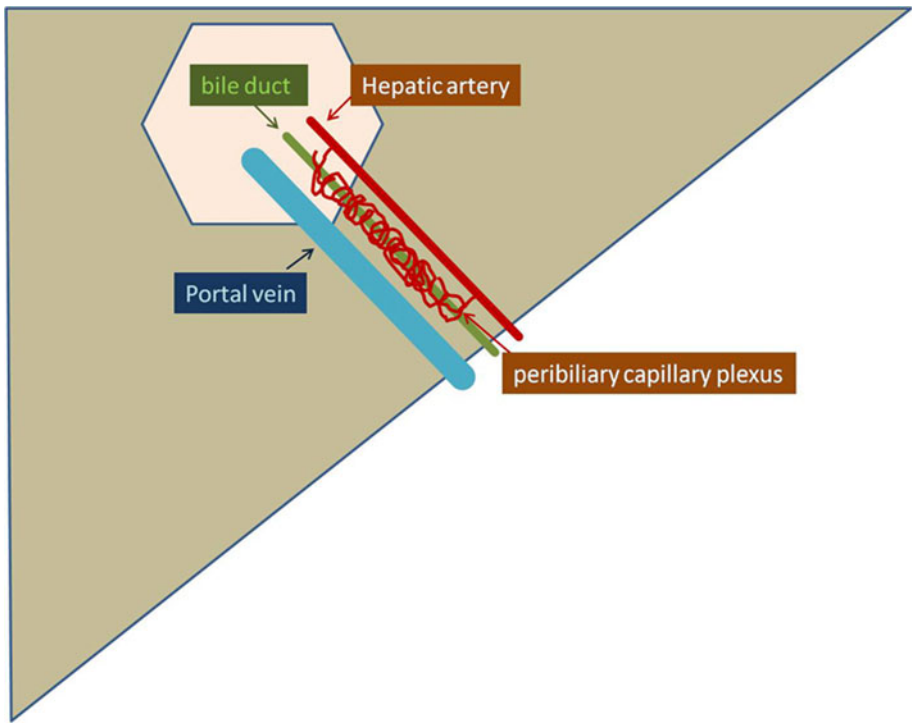

Fig. 1. Illustration of normal intrahepatic bile duct irrigation.

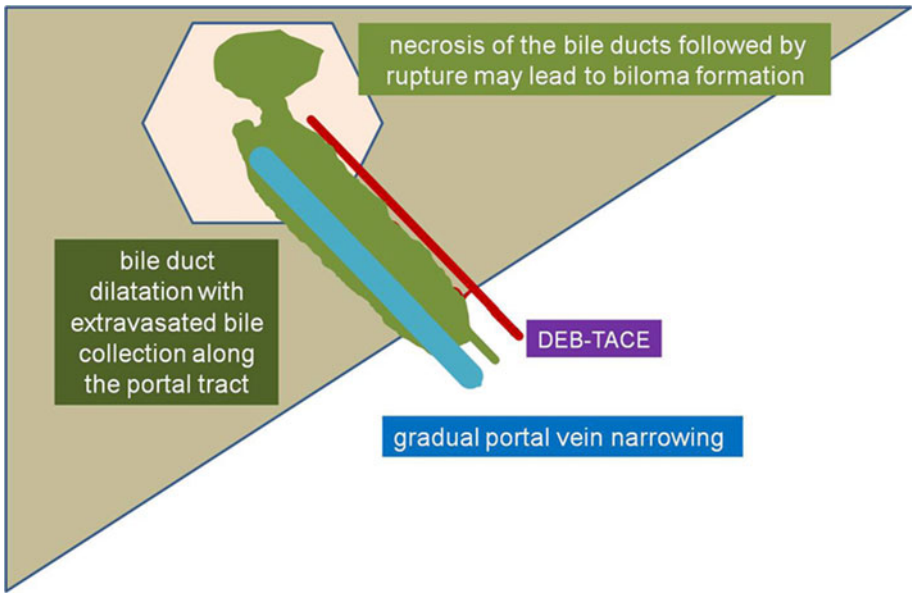

Fig. 2. Illustration of ischemic bile duct injuries: bile duct stricture and dilatation, periportal edema, and bilomas.

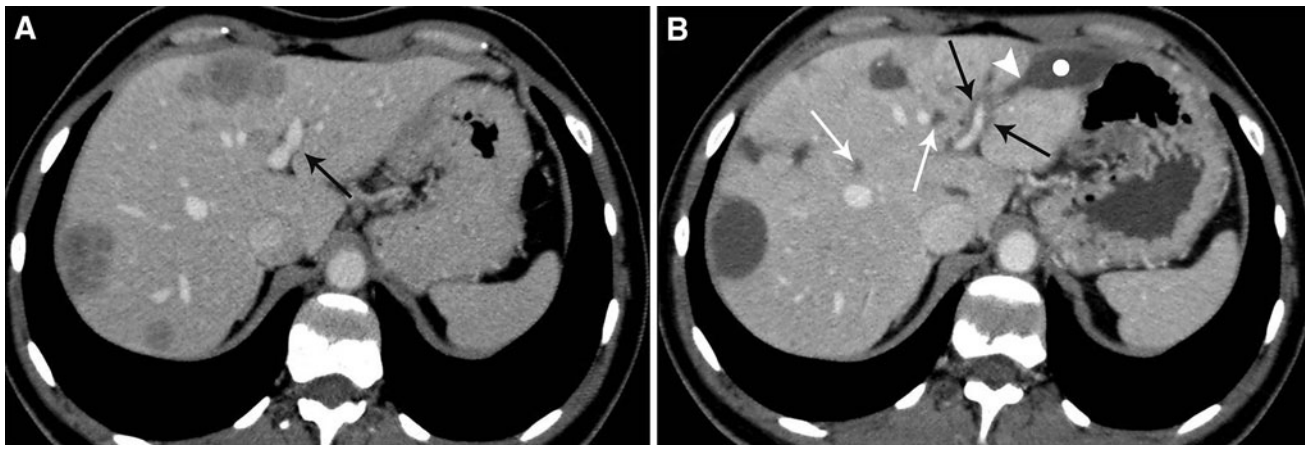

Fig. 3. A 45-year-old male patient with melanoma liver metastases. **A** Before TACE-DC-beads, the left portal vein branch (*arrow*) is well demonstrated on portal venous phase CT. Note metastases in segment IV and VII. **B** Two months follow-up CT after TACE-DC-beads. Portal venous phase CT depicts periportal edema (*black arrows*), bile duct dilatation (*white arrows*) obliteration of left portal vein for segment III (*arrowhead*) and a fluid collection, suggesting biloma (*white circle*). Note also necrotic changes in liver metastases after treatment.

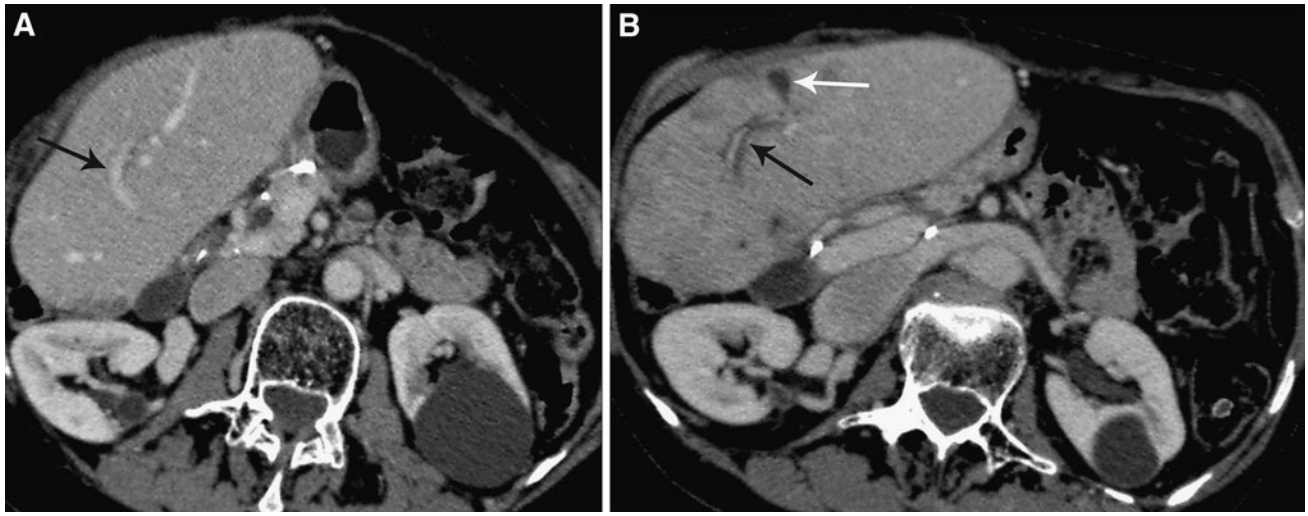

Fig. 4. A 69-year-old female patient with carcinoid liver metastases (not shown). **A** Before TACE-DC-beads, the left portal vein branch (*arrow*) is well demonstrated on portal venous phase CT. **B** Two months follow-up portal venous phase CT after TACE-DC-beads depicts periportal edema (*black arrow*). A small biloma is also seen (*white arrow*).

The incidence of TACE-DC-beads related bile duct injury is higher in patients who had a normal liver than in those with a cirrhotic liver. This can be the result of inadvertent retention at the hepatic arteries or capillary network of some of the small microspheres loaded with a high concentration of the chemotherapeutic agent causing stasis of blood flow and chemical irritation of the vascular endothelium favoring vasculitis and/or ischemic injury (Fig. 3) [11]. In cirrhotic livers, the peribiliary capillary plexus usually becomes hypertrophied resulting in an increased capacity of collateralization that could protect the bile ducts from ischemic injury.

Periportal edema

Periportal edema, shown as bilateral linear low attenuating areas alongside the portal vein, is the result of extravasated bile or reactive fluid collection in addition to bile duct dilatation (Fig. 4).

Bilomas

A biloma is an extrabiliary collection of bile. The possible mechanism of biloma formation after TACE-DC-beads is considered to be the development of

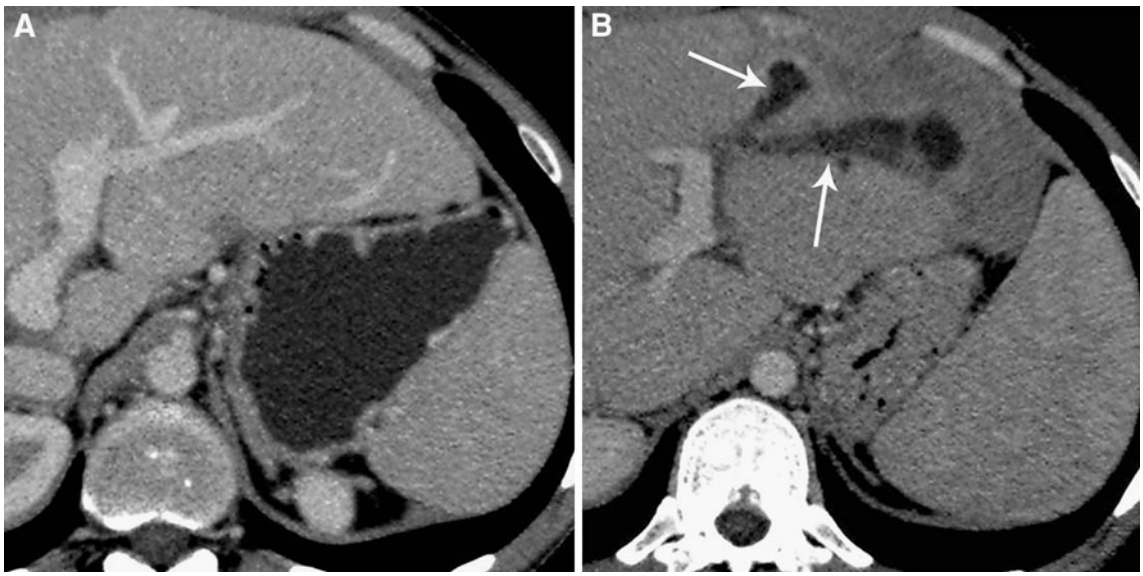

Fig. 5. A 30-year-old male patient with carcinoid liver metastases (not shown). **A** Portal venous phase CT obtained before treatment shows patent left portal vein. **B** 5 months follow-up portal venous phase CT after TACE-DC-beads through the lobar branch of the left hepatic artery shows periportal edema associated with bile duct dilatation and complete obliteration of left portal vein branches.

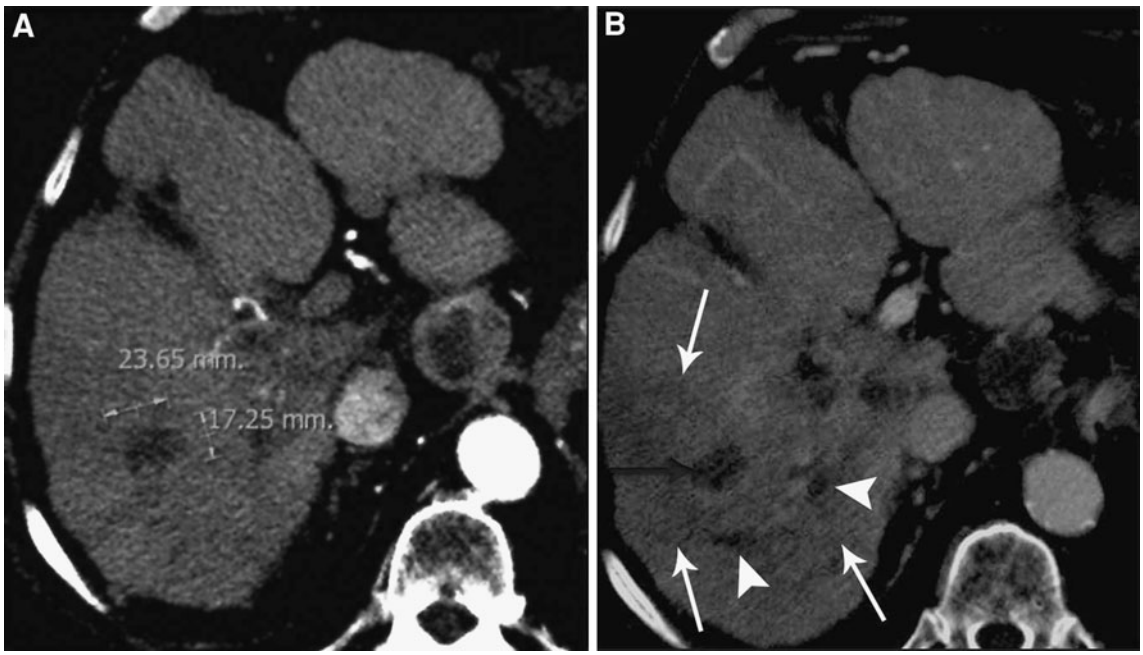

Fig. 6. A 70-year-old male patient with HCC. **A** Arterial phase CT after TACE-DC-beads shows necrotic changes in HCC with minimal peripheral enhancement. B Portal venous phase CT after TACE-DC-beads shows necrotic changes in HCC with minimal peripheral enhancement more evident during portal venous phase. Ill-defined hypodense areas are seen around the treated lesion (*white arrows*). Bile duct dilatation is also depicted (*white arrowheads*).

peripheral bile duct necrosis with bile leakage caused by microvascular damage of the peribiliary capillary plexus (Fig. 4) [12]. Occasionally bilomas become infected and progress to abscess formation, which may require an urgent drainage procedure [13, 14].

Obliteration of intrahepatic portal vein branches

TACE-induced bile duct injury, including focal dilatation of the intrahepatic bile duct with or without

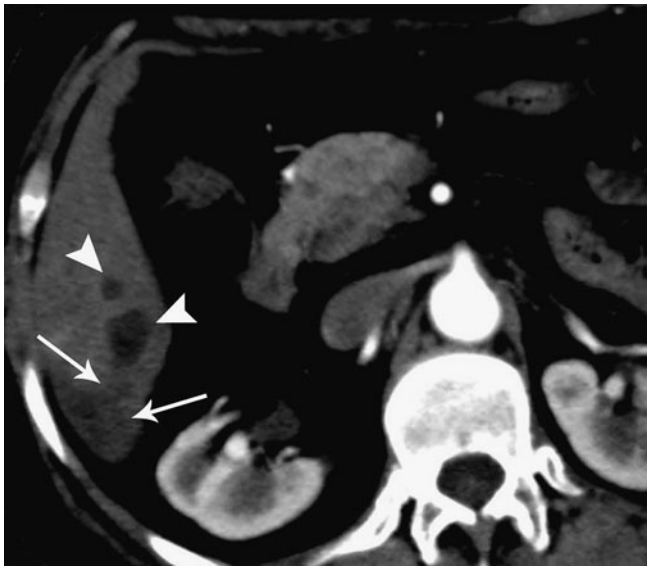

Fig. 7. A 69-year-old male patient with HCC (not shown). Portal venous phase CT after TACE-DC-beads shows a wedge-shaped, non-enhancing low-density area suggesting sub-segmental parenchymal infarction (*arrows*). Small bilomasare also seen (*arrowheads*).

extravasation of bile along connective tissue sheaths of Glisson capsule, may obliterate the adjacent portal vein branch [16].

Portal venous narrowing and thrombosis can result from extravasated fluid collection due to disruption of a necrotized bile duct that can gradually compress and compromise the adjacent portal vein branches (Fig. 5). An additional inflammatory process due to chemical vasculitis could also be involved in this process [15].

Parenchymal hypodense ill-defined areas

Parenchymal hypodense ill-defined areas are found around the treated lesions or coinciding with the segmental or lobar distribution of the treatment. This finding is also more frequent in normal livers than in cirrhotic patients. The combination of simultaneously decreased arterial and portal venous perfusion secondary to the procedure itself and to the bile duct injury that favors obliteration of adjacent portal vein branches, contributes to the appearance of these areas that could represent reversible hypoperfusion (Fig. 6). Otherwise, the typical appearance of parenchymal infarction appears as a low-density area on all phases of dynamic CT most of which extended to the periphery of the liver and are wedge-shaped [17] (Fig. 7). Finally, segmental or subsegmental volume reduction with parenchymal atrophy can develop in these areas.

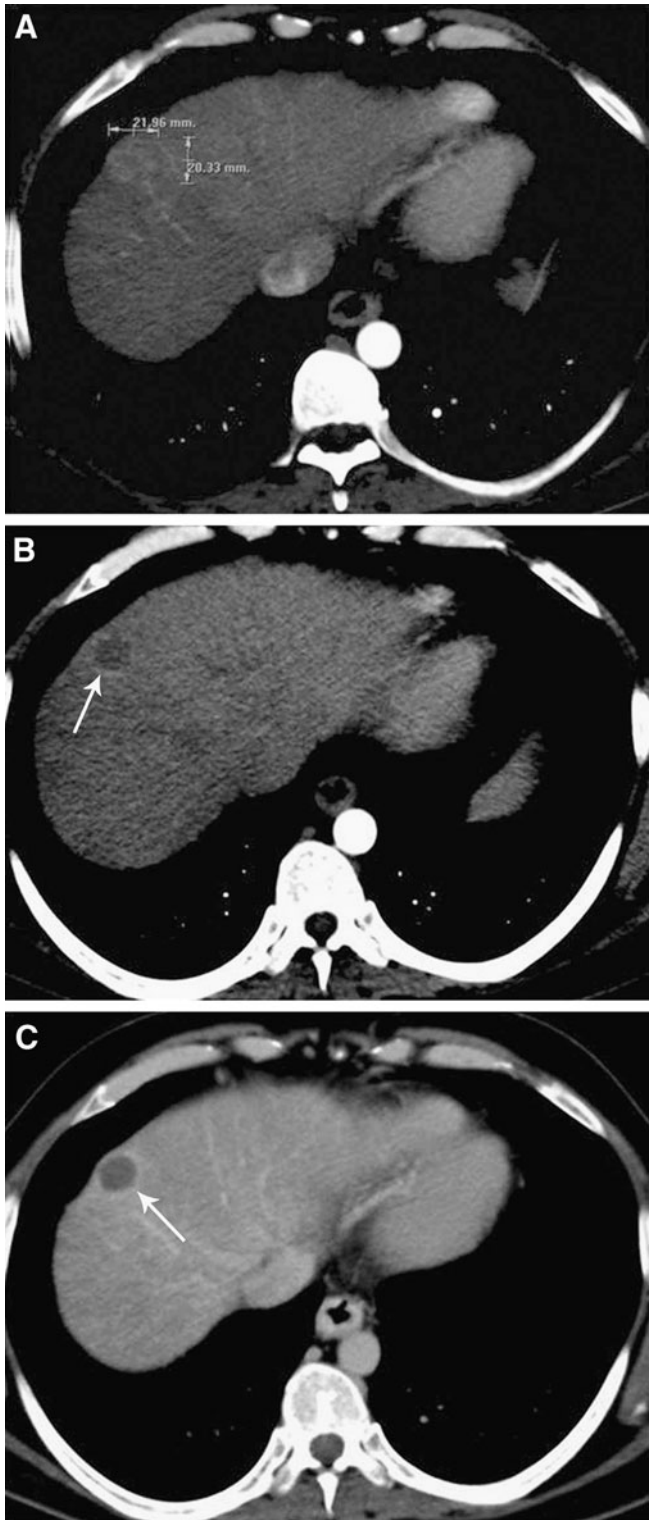

Fig. 8. A 50-year-old male patient with HCC segment VIII. **A** Arterial phase CT. **B** Two months follow-up arterial phase CT after TACE-DC-beads shows necrotic changes in HCC with minimal peripheral enhancement around the treated lesion (*arrow*). **C** Two months follow-up portal venous phase CT after TACE-DC-beads shows necrotic changes in HCC with minimal peripheral enhancement around the treated lesion.

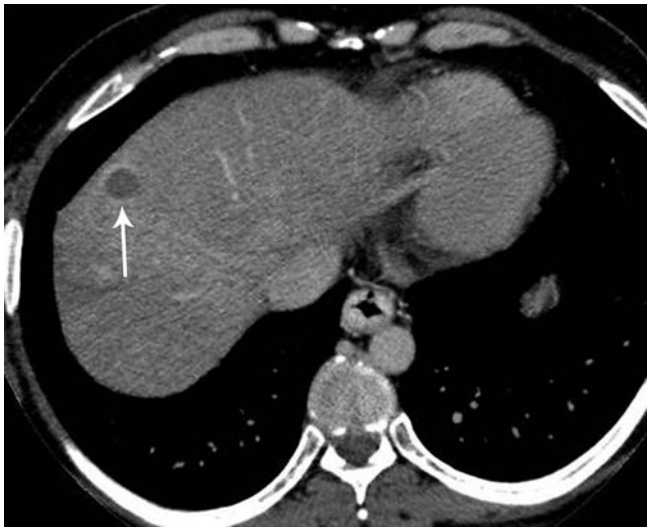

Fig. 9. Same patient of Fig. 8. Six months follow-up portal venous phase CT after TACE-DC-beads shows necrotic changes in HCC with minimal peripheral enhancement around the treated lesion.

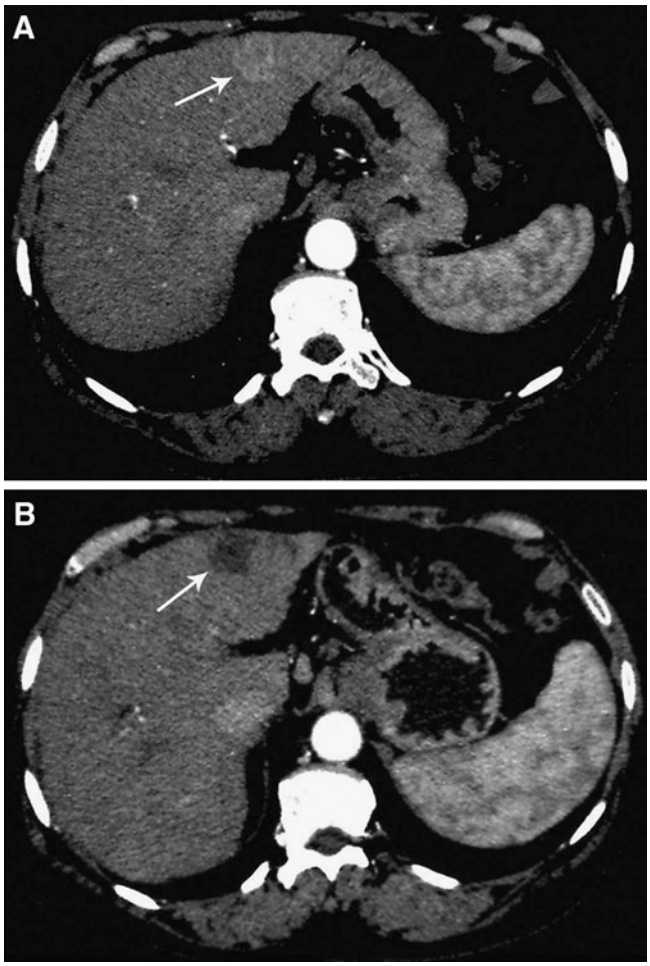

Fig. 10. A 63-year-old female. **A** Cirrhosis and HCC in left lateral segment (*arrow*). **B** Two-month CT follow-up shows complete tumor necrosis of the treated lesion.

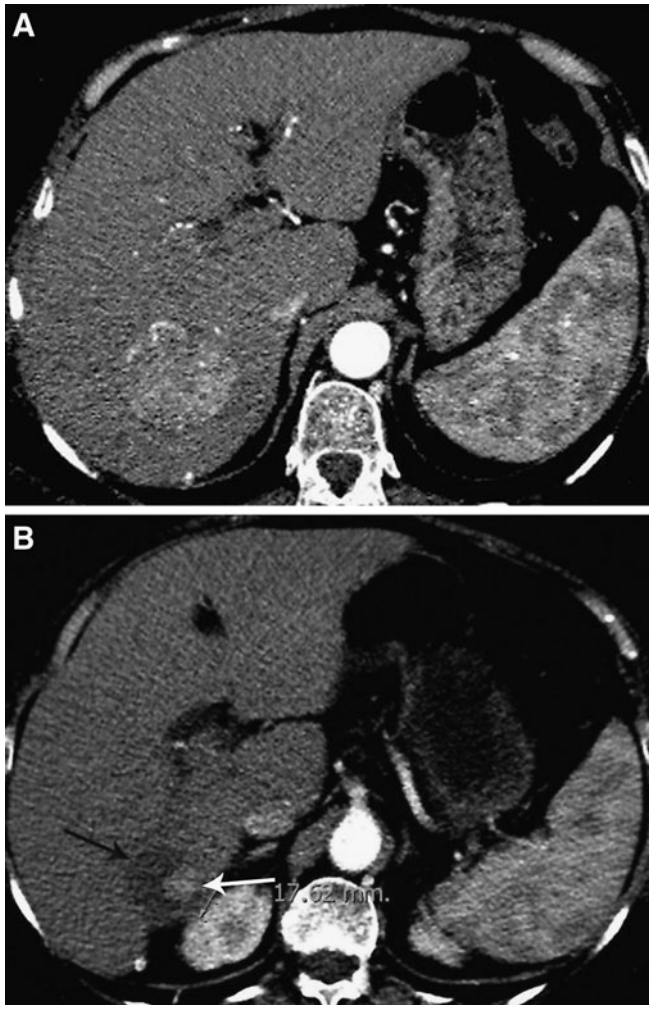

Fig. 11. A 64-year-old female. **A** Cirrhosis and HCC in segment VII–VI. **B** Follow-up arterial phase CT shows decrease in size, and lesion devascularization (*black arrow*) with a residual nodular enhancement suggesting viable tumor (*white arrow*).

Perilesional parenchymal enhancement

Another frequent finding is the presence of perilesional parenchymal enhancement in patients after TACE-DC-beads treatment. These enhancing areas are usually seen during arterial or portal venous phase as ill-defined parenchymal enhancement or a continuous peripherical enhancement around the treated lesion (Fig. 8).

Unlike the familiar transient peripheral rim of enhancement reported by Lim et al. [18] in 79% of their cases after radiofrequency ablation that usually disappeared by the 1-month follow-up CT examination, after TACE-DC-beads treatment these areas are usually ill-defined and do not disappear by the first month (Fig. 9). We consider this finding probably as benign physiologic

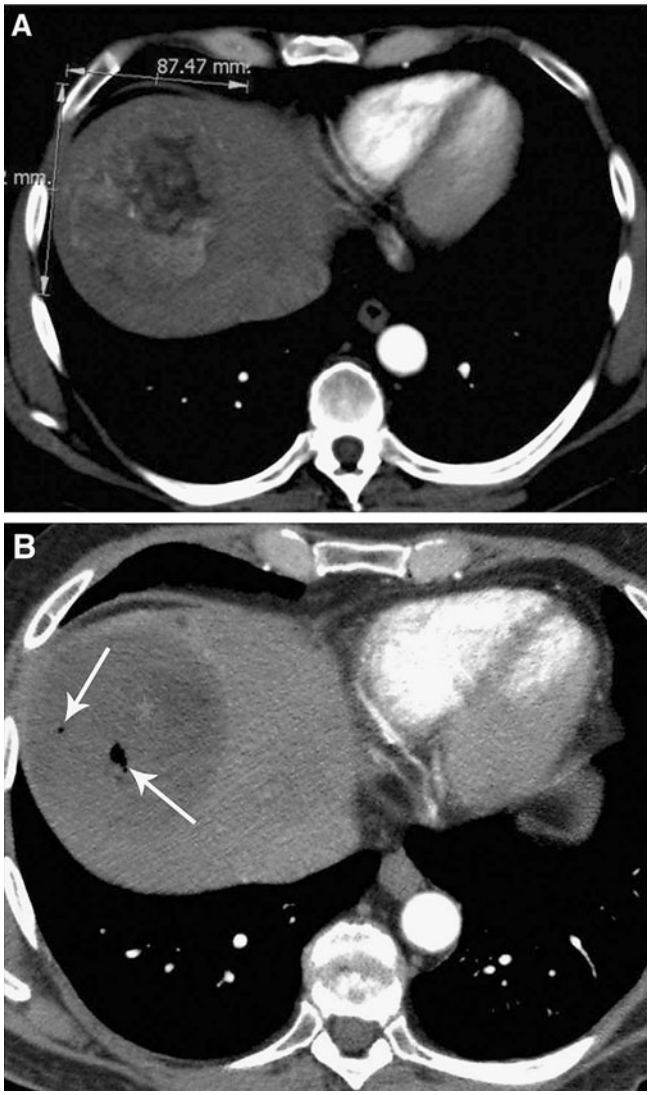

Fig. 12. A 64-year-old male. **A** HCC in segment VIII. **B** One-month follow-up arterial phase CT after TACE-DC-beads shows complete tumor necrosis in HCC and air bubbles within the tumor (*arrows*). The patient was asymptomatic with no clinical manifestations.

hemodynamic response to the other parenchymal changes previously described including bile duct injury, chemical arteritis, and obliteration of intrahepatic portal vein branches.

Intralesional changes

After TACE-DC-beads treatment, intralesional changes can be seen as necrosis, representing devascularization of the lesion (Fig. 10), or residual enhancement indicating persistence of disease (Fig. 11).

Sometimes, intratumoral air after treatment is seen as an incidental finding and should not be confused with an hepatic abscess (Fig. 12).

Conclusion

TACE-DC-beads is a new local treatment for patients with hepatocellular carcinoma and hypervascular liver metastases. Different findings, not only in the target lesion but also in the perilesional parenchyma, are frequently identified in these patients. Hence, it is important for the radiologist to be familiar with the spectrum of changes induced by this treatment in order to distinguish therapeutic effect and collateral findings from complications and residual or recurrent tumor.

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