Bile Duct Injuries Leading to Portal Vein Obliteration after Transcatheter Arterial Chemoembolization in the Liver: CT Findings and Initial Observations

PURPOSE: To document the computed tomographic (CT) findings of transcatheter arterial chemoembolization (TACE)–induced, localized bile duct injuries leading to portal vein branch obliteration in the liver and to elucidate the clinical implications with retrospective review of the authors’ experiences.

MATERIALS AND METHODS: Follow-up CT scans obtained in 11 patients with TACE-induced intrahepatic bile duct dilatation were reviewed retrospectively to evaluate serial changes in the adjacent portal vein branches and hepatic parenchyma. Clinical data, including time between TACE and CT and serum alkaline phosphatase levels, also were analyzed.

RESULTS: Of 11 patients with marked (n = 8) or mild (n = 3), lobar (n = 4) or segmental (n = 7) bile duct dilatation with or without bile collection in the tissue sheaths of the Glisson capsule or hepatic parenchyma, nine (82%) had bile duct changes at the first CT follow-up, within 1 month after TACE. Marked narrowing or obliteration of the adjacent intrahepatic portal vein branches in 10 (91%) patients resulted in progressive atrophy of the corresponding hepatic parenchyma in nine (82%) at variable times after TACE. The serum alkaline phosphatase level increased to more than 200 U/L in eight (89%) of nine patients 1 month after TACE.

CONCLUSION: TACE-induced intrahepatic bile duct injury resulting in obliteration of the adjacent portal vein branch seems to be one cause of hepatic parenchymal atrophic changes after TACE.

Transcatheter arterial chemoembolization (TACE) has been used widely to treat hepatocellular carcinoma and less frequently to treat other malignant tumors of the liver (1–5). Among the various complications related to TACE, bile duct injury has been sporadically reported on since the introduction of hepatic arterial embolization therapy (6–10). During our routine computed tomographic (CT) examination of patients with TACE-related bile duct injuries, narrowing or obliteration of portal vein branches adjacent to the dilated bile ducts has been observed intermittently. We believed that TACE-related bile duct dilatation or biloma could possibly compromise the coinciding portal venous perfusion and induce subsequent ischemic injuries to the nontumorous hepatic parenchyma. Thus, our study was undertaken to document the CT findings of TACE-induced, localized bile duct injuries leading to the obliteration of gross portal vein branches in the liver and to elucidate the clinical implications of this condition by means of retrospective review of our experiences.

MATERIALS AND METHODS

During the past 5 years, more than 500 patients at our institution have undergone one or more TACE sessions to control primary or metastatic liver malignancy. Our routine
protocol for TACE includes the administration of an emulsion that consists of 1–20 mL of iodized oil (Lipiodol; Andre-Guerbet, Aulnay-sous-Bois, France) and 10–50 mg of doxorubicin hydrochloride emulsion (Adriamycin; Kyowa Hakko Kogyo, Tokyo, Japan), with the dose dependent on the size, extent, and vascularity of the tumor. If possible, the emulsion is injected exclusively into the segmental or subsegmental arterial branches feeding the tumor, and then gelatin sponge fragments (Gelfoam; Upjohn, Kalamazoo, Mich) are administered. Liver function tests, including those to measure serum aminotransferase and alkaline phosphatase levels, are routinely performed 1 or 2 days after TACE, before the patient’s discharge, regardless of the clinical course. In the current study, the first follow-up examination was routinely CT performed 3 or 4 weeks after TACE, and in those patients without marginal tumor recurrence or newly developed tumors, further follow-up CT studies were performed 3 and 6 months after TACE. At follow-up imaging, liver function tests also were performed in the majority of patients.

For follow-up imaging, nonenhanced and contrast-material–enhanced CT images were obtained by using a conventional scanner (HiLite Advantage; GE Medical Systems, Milwaukee, Wis) or a helical scanner (HiSpeed Advantage; GE Medical Systems) that, to our knowledge, has been available since June 1999. An intravenous drip infusion of 150 mL of iodinated contrast agent (iopromide [Ultrasound]: Schering, Berlin, Germany) was administered during conventional contrast-enhanced CT scanning. For two-phase (ie, arterial phase and parenchymal or delayed phase) dynamic helical CT imaging, 100 mL of iopromide was injected intravenously at a rate of 3 mL/sec by using an automatic injector.

A retrospective search of the computerized reports of 381 pre-TACE and 1,307 post-TACE follow-up CT examinations performed in 381 consecutive patients, documented between September 1996 and June 2000, revealed that 42 patients had developed TACE-related intrahepatic bile duct dilatation, intraparenchymal fluid collection, and/or cyst formation with or without segmental or lobar parenchymal infarction or atrophic changes. Before reviewing the CT reports, we excluded the data on more than 120 patients treated by using TACE for whom no pre-TACE or follow-up CT data were available during the period of case collection. The imaging criteria for the diagnosis of TACE-induced bile duct injury were a disproportionately dilated bile duct with lobar or segmental distribution that developed after TACE or a newly developed cystic lesion accompanied by segmental bile duct dilatation.

The film hard-copy follow-up CT scans and pre-TACE CT scans serially obtained in the 42 selected patients were reviewed by four radiologists (J.S.Y., K.W.K., M.S.P., S.W.Y.) at consensus to determine the presence of newly developed marked portal vein obliteration adjacent to the dilated bile duct. In 22 of the 42 patients, the bile duct injuries were localized and limited to small subsegmental branches and the identification of the portal vein was not possible in the intensely opaque area of iodized oil uptake, with or without segmental infarction or necrosis resulting from selective TACE. Another five patients had tumor thrombosis in dilated portal veins, and another four were thought to have direct bile duct invasion caused by tumor progression during the follow-up period. After exclusion of these cases, a total of 11 patients (six men and five women aged 32–62 years) were selected for detailed analysis of CT and clinical findings.

The study was approved by our institutional review board, and informed consent was obtained from the patients or their family members. The degree and location of the TACE-induced bile duct dilatation, degree and level of portal vein narrowing, and liver parenchymal changes (including newly developed foci lesions, segmental or lobar perfusion variation, and/or decreased volume of the liver suggestive of parenchymal atrophy) depicted on CT images were determined and recorded by two radiologists (J.S.Y., K.W.K.) at consensus. The bile duct dilatation after TACE was graded as mild or marked, as compared with the diameter of the adjacent portal veins at pre-TACE CT. Mild dilatation was defined as an increased diameter of the bile duct smaller than the diameter of the adjacent portal vein at pre-TACE CT. When the dilated bile duct diameter was similar to or greater than those of the adjacent portal veins before TACE, we regarded this as marked dilatation. In addition, only narrowing of the portal vein with preservation of the intraluminal blood flow was distinguished from complete obliteration of the portal vein branches.

The clinical results reviewed were final diagnosis of the treated tumor; previous history of abdominal surgery or underlying liver disease; time between TACE and bile duct, portal vein, and/or liver parenchyma changes at CT; and serum alkaline phosphatase levels at follow-up. The collected data concerning the TACE method included the location of the catheter tip during the infusion of embolic materials, the amount of doxorubicin hydrochloride and iodized oil administered, the use of gelatin sponge fragments, and the total number of TACE procedures performed before the appearance of bile duct injuries. For patients who underwent more than one TACE session, the data obtained during the last session just prior to the development of bile duct injury were used.

The clinical data and CT findings of the 11 patients with TACE-induced intrahepatic bile duct injuries adjacent to the first- or second-order branches of the intrahepatic portal vein are summarized in Table 1. One TACE session in six patients, including five patients with metastatic tumors and one with hepatocellular carcinoma; two sessions in four patients (patients 1, 3, 8, and 11); and three sessions in one patient (patient 10) were performed before the CT appearance of TACE-induced bile duct injury.

At CT, eight patients (73%) had a newly developed linear, low-attenuating area alongside the portal tract at CT; this was suggestive of marked dilatation of the lobar (n = 3) or segmental branches (n = 5) with or without bile extravasation in the connective tissue sheaths of the Glisson capsule surrounding the portal triads. The widths of the linear low-attenuating areas were greater than those of the corresponding portal vein branches before TACE (Figs 1, 2). Three patients (27%) had mild dilatation of the lobar (n = 1) or segmental (n = 2) bile ducts (Fig 3). In six patients (54%), the bile duct dilatation was limited to the area distal to the location of the catheter tip during TACE. In the other five patients (45%), however, the area of bile duct dilatation was more extensive and involved the neighboring segments.

With respect to the time of appearance of the bile duct dilatation after TACE, nine patients (82%) had mild (n = 4) or marked (n = 5) bile duct dilatation at the first CT follow-up, within 1 month after TACE. Two patients (patients 4 and 11) had no definite imaging findings that suggested bile duct injury at initial CT follow-up, but they had such findings at the second CT follow-up 11 and 16 weeks after TACE, respectively. The time-related progression of bile duct dilatation was well demonstrated on the CT scans ob-
<table>
<thead>
<tr>
<th>Patient No./Sex/ Age</th>
<th>Final Diagnosis</th>
<th>Underlying Liver Disease or Abdominal Surgery History</th>
<th>TACE Regimen</th>
<th>Bile Duct Dilatation*</th>
<th>Portal Vein Narrowing*</th>
<th>Hepatic Parenchyma Changes*</th>
<th>Tumor Progression*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/M/55 HCC</td>
<td>Chronic BHV, partial hepatocarcinoma</td>
<td>50 mg DXH, 5.0 mL IO, and GF to right RHA</td>
<td>Marked dilatation in anterior and posterior segments of right lobe at 3 weeks; marked dilatation in left lobe at 17 weeks</td>
<td>Mild narrowing in right lobe at 4 weeks; complete vein obliteration in right lobe at 13 weeks</td>
<td>Right lobe atrophy at 13 weeks</td>
<td>Multiple nodular tumor recurrence in left lobe at 13 weeks</td>
<td></td>
</tr>
<tr>
<td>2/F/50 Rectal cancer metastasis</td>
<td>Lower anterior resection of rectum</td>
<td>30 mg DXH and 3.0 mL IO to RHA</td>
<td>Marked dilatation in right lobe at 3 weeks; marked dilatation in right lobe at 8 weeks; mild dilatation in left lobe at 17 weeks</td>
<td>Complete vein obliteration in right lobe at 4 weeks</td>
<td>Right lobe atrophy at 18 weeks</td>
<td>Multiple tumor nodules in entire liver at 40 weeks</td>
<td></td>
</tr>
<tr>
<td>3/F/58 HCC</td>
<td>Chronic BHV</td>
<td>20 mg DXH, 2.0 mL IO, and GF to RHA</td>
<td>Mild dilatation in right lobe at 3 weeks; marked dilatation in right lobe at 8 weeks; mild dilatation in left lobe at 17 weeks</td>
<td>Mild narrowing in right lobe at 4 weeks; complete vein obliteration in right lobe at 4 weeks</td>
<td>Right lobe atrophy at 17 weeks</td>
<td>No tumor recurrence at 237 weeks</td>
<td></td>
</tr>
<tr>
<td>4/M/62 Stomach cancer metastasis</td>
<td>STG-GJ</td>
<td>40 mg DXH, 4.0 mL IO, and GF to right posterior segmental artery</td>
<td>Marked dilatation in posterior segment of right lobe at 11 weeks</td>
<td>Mild narrowing in posterior segment of right lobe at 11 weeks; complete vein obliteration in posterior segment of right lobe at 54 weeks</td>
<td>Cholangiohepatic abscess in right lobe at 19 weeks; atrophy of posterior segment of right lobe at 54 weeks</td>
<td>No tumor recurrence at 54 weeks</td>
<td></td>
</tr>
<tr>
<td>5/M/50 Stomach cancer metastasis</td>
<td>STG-GJ</td>
<td>30 mg DXH, 3.0 mL IO, and GF to subsegmental artery 8</td>
<td>Mild dilatation in anterior segment of right lobe at 4 weeks</td>
<td>Complete vein obliteration in anterior segment of right lobe at 4 weeks</td>
<td>Atrophy of anterior segment of right lobe at 4 weeks</td>
<td>No tumor recurrence at 110 weeks</td>
<td></td>
</tr>
<tr>
<td>6/M/57 Stomach cancer metastasis</td>
<td>STG-GJ</td>
<td>30 mg DXH and 3.0 mL IO to subsegmental artery 8</td>
<td>Marked dilatation in anterior segment of right lobe at 3 weeks</td>
<td>Complete vein obliteration in anterior segment of right lobe at 3 weeks</td>
<td>Atrophy of anterior segment of right lobe at 22 weeks</td>
<td>Intraabdominal carcinomatosis at 47 weeks</td>
<td></td>
</tr>
<tr>
<td>7/F/32 Stomach cancer metastasis</td>
<td>STG-GJ</td>
<td>20 mg DXH, 2.0 mL IO, and GF to subsegmental arteries 7 and 8</td>
<td>Mild dilatation in right lobe at 4 weeks; mild dilatation in right lobe at 7 weeks; mild dilatation in right and left lobes at 16 weeks</td>
<td>Mild narrowing in right lobe at 4 weeks; complete vein obliteration in right lobe at 7 weeks; complete vein obliteration in right lobe and mild narrowing in left lobe at 17 weeks</td>
<td>Decreased perfusion in right lobe at 7 weeks</td>
<td>Systemic carcinomatosis with bile duct dilatation and PTBD at 16 weeks</td>
<td></td>
</tr>
<tr>
<td>8/M/54 HCC</td>
<td>None identified</td>
<td>50 mg DXH, 5.0 mL IO, and GF to right anterior segmental artery 2</td>
<td>Marked dilatation in anterior segment of right lobe at 3 weeks</td>
<td>Complete vein obliteration in anterior segment of right lobe at 3 weeks</td>
<td>Atrophy of anterior segment of right lobe at 5 weeks</td>
<td>Hepatic failure after three additional TACE sessions, at 17 weeks</td>
<td></td>
</tr>
<tr>
<td>9/F/53 HCC</td>
<td>Chronic BHV and cirrhosis</td>
<td>15 mg DXH, 1.5 mL IO, and GF to subsegmental artery 3</td>
<td>Mild dilatation in lateral segment of left lobe at 3 weeks</td>
<td>Mild narrowing in lateral segment of left lobe at 3 weeks</td>
<td>None identified</td>
<td>Spontaneous regression of bile duct dilatation at 10 weeks</td>
<td></td>
</tr>
<tr>
<td>10/M/51 HCC</td>
<td>Chronic BHV and cirrhosis</td>
<td>20 mg DXH, 2.0 mL IO, and GF to subsegmental artery 8</td>
<td>Marked dilatation in anterior segment of right lobe at 3 weeks</td>
<td>Complete vein obliteration in anterior segment of right lobe at 3 weeks</td>
<td>Atrophy of anterior segment of right lobe at 12 weeks</td>
<td>Progressive right lobe atrophy after four additional TACE sessions, at 96 weeks</td>
<td></td>
</tr>
<tr>
<td>11/F/45 HCC</td>
<td>Chronic BHV and cirrhosis</td>
<td>50 mg DXH, 5.0 mL IO, and GF to RHA</td>
<td>Marked dilatation in anterior and posterior segments of right lobe at 16 weeks</td>
<td>Complete vein obliteration in anterior and posterior segments of right lobe at 16 weeks</td>
<td>Right lobe atrophy at 135 weeks</td>
<td>Hepatic failure after three additional TACE sessions, at 135 weeks</td>
<td></td>
</tr>
</tbody>
</table>

Note.—BHV = hepatitis B, DXH = doxorubicin hydrochloride, GF = gelatin sponge fragments, HCC = hepatocellular carcinoma, IO = iodized oil, PTBD = percutaneous transhepatic biliary drain, RHA = right hepatic artery, STG-GJ = subtotal gastrectomy and gastrojejunostomy.

* TACE-induced bile duct dilatation, intrahepatic portal vein narrowing, hepatic parenchyma changes, and tumor progression depicted at CT at different intervals after the last TACE.
tained in three patients (patients 1, 3, and 7) (Fig 1).

Marked narrowing or obliteration of the first- (n = 4) or second-order branches (n = 6) of the intrahepatic portal vein was demonstrated in 10 patients (91%) and was associated with progressive atrophy of the corresponding hepatic parenchyma in nine patients (82%) at subsequent follow-up CT (Figs 1, 2). In eight patients (73%), narrowing of the portal vein was accompanied by marked bile duct dilatation with or without extravasated bile. However, three patients (patients 5, 7, and 9) had portal vein narrowing, even though the degree of bile duct dilatation was mild without extravasated bile collection (Fig 3). The timing of the hepatic parenchymal atrophy in nine patients was variable, from 4 to 54 weeks (mean + SD, 21 weeks ± 18) after TACE. One patient (patient 7) with mild bile duct dilatation and progressive portal vein narrowing had a marked decrease in portal venous perfusion and died owing to multigian failure caused by systemic carcinomatosis; this patient was not properly followed up. Another patient (patient 9) had no remarkable parenchymal changes at CT, and the bile duct dilatation had spontaneously regressed by the time of 10-week follow-up CT.

The serum alkaline phosphatase level was elevated to more than 200 U/L in eight (89%) of the nine patients in whom this enzyme level was tested within 1 month after TACE (Table 2, Fig 4). In four patients (patients 1, 2, 6, and 8), the serum alkaline phosphatase level decreased after 1 or 2 months, whereas other patients had progressive elevations or fluctuations that included biliary dilatation progression in the right and left lobes of the liver (patient 3), superimposed cholangiohepatic abscess (patient 4), progressive extrahepatic biliary obstruction due to intraperitoneal carcinomatosis (patient 7), and gradual parenchymal atrophy with overt cirrhosis (patients 10 and 11). In another two patients (patients 5 and 9), in whom serum enzyme testing was not performed at the initial 1-month CT follow-up after segmental TACE, the serum alkaline phosphatase levels increased moderately 3 months after TACE.

DISCUSSION

Hepatic parenchymal atrophy, a well-known complication of TACE, is related to ischemic injury, especially in patients with decreased portal venous perfusion caused by thrombosis or in those who have undergone repeated embolization (10,11). In daily practice, however, parenchymal atrophic changes after TACE are not infrequently seen, even in patients without risk factors for these changes.

Bile duct injury has been reported intermittently as a complication of TACE since the introduction of hepatic arterial
embolization therapy (6–10). It has been suggested that small embolic material diameter (7) and repeated TACE procedures (10) are related to ischemic bile duct injuries with or without chemical arteritis of the small vessels supplying the bile duct wall. The majority of patients selected for the present study had undergone only one or two TACE sessions before the appearance of bile duct injuries at CT. However, the CT findings of overt bile duct injury developed in the intrahepatic first- or second-order branches.

In a previous study (12), the incidence of TACE-related bile duct injury was substantially higher in patients who had an otherwise normal liver than in those with a cirrhotic liver, regardless of the number of TACE procedures or the quantities of embolic materials. In the majority of patients in our study, the liver was not cirrhotic. During cirrhotic changes of the liver, the peribiliary capillary plexus becomes hypertrophied, and the resulting increased capacity for collateralization protects the bile ducts from ischemic injury (13). In the noncirrhotic liver, however, even a small amount of embolic material injected into the normal nonhypertrophied capillary network can induce stasis of blood flow and chemical irritation of the vascular endothelium that result in vasculitis and/or ischemic injury.

With regard to extent of bile duct injury, five patients in our study had bile duct dilatation that extended beyond the area distal to the tip of the catheter during TACE. In patients with normally small or spastic hepatic arteries, there is the possibility of proximal backflow of the embolic materials into the hepatic arteries during TACE. If the catheter tip were wedged to a small arterial branch, the positive injection pressure would contribute to a proximal backflow of embolic materials into the peribiliary capillary plexus.

The patients with CT findings of gross portal vein thrombosis before TACE were excluded from case selection for the present study. Therefore, the cases of hepatic parenchymal atrophy observed in our study should be regarded as consequences of decreased portal venous perfusion after TACE. The results of numerous investigations have demonstrated that biliary tree obstruction causes a decrease in portal venous inflow, which is perhaps related to the dilated intrahepatic bile duct radicles compressing the lower-pressure portal venous radicles (14,15). To our knowledge, however, only one report (16) on the direct relationship between TACE-induced bile duct dilatation and occlusion of the adjacent portal vein has been published.

In the cases examined in this study, there were linear or branching low-attenuating areas suggestive of bile duct dilatation with or without an extravasated
bile collection along the portal tract. As demonstrated by the patients with gradual portal vein narrowing, localized periportal tracking—that is, bilateral linear low-attenuating areas alongside the portal vein—also was revealed on the CT scans and was suggestive of an extravasated bile or reactive fluid collection in addition to bile duct dilatation. Extravasated bile from the disruption of the necrotized bile duct after TACE can track along the low-resistance connective tissue sheaths of the Glisson capsule that surrounds the portal triads. We believe that the dilated bile duct and the extravasated fluid collection in the Glisson capsule can gradually compress and compromise the adjacent portal vein branches.

There is also the possibility of a periportal inflammatory process that is related to the effect of high concentrations of chemotherapy and embolization materials administered to the periportal tissue without direct extravasation of the bile (17). An additional inflammatory process related to the chemical vasculitis of peribiliary and periportal vascular plexus can induce phlebitis that results in portal vein thrombosis. The inflammatory process can induce portal vein narrowing and decreased perfusion, especially in patients without marked bile duct injuries at CT who have marked narrowing of the portal vein branches and subsequent parenchymal atrophy (16).

Although no acute parenchymal infarction was observed to be related to the simultaneous cutoff of arterial and portal flow, as is usually seen with subsegmentally selective TACE, gradually decreased portal venous flow can induce functional impairment and subsequent atrophy of hepatocytes, or the so-called Zahn infarct (18).

The time between TACE and the appearance of atrophic changes at CT was variable, and the mean time observed in our study, 20 weeks, was longer than that previously reported by Yamashita et al (11) (2–3 months after TACE). This difference might be attributed to the makeup of the study groups or to our exclusion of patients with portal vein thrombosis or segmental infarction, which might in itself induce rapid parenchymal atrophic change. Ischemic bile duct injury, bile duct dilatation with portal vein narrowing, and the resultant parenchymal atrophy observed in our study cases seem to have occurred more gradually than did those reported previously (11). The cause of the variability in time of hepatic atrophy (4–54 weeks after TACE) is not clear; however, on a case by case basis, the degree of bile duct dilatation, severity of the periportal inflammation, repeated TACE, and/or timing of the follow-up imaging could have contributed to the timing of overt parenchymal atrophic changes at CT.

In the absence of bone disease or pregnancy, elevated alkaline phosphatase ac-
tivity usually reflects impaired biliary tract function. The most dramatic increases in the level of this enzyme occur in cases of extrahepatic biliary tract obstruction or intrahepatic functional cholestasis. The enzyme may be elevated even in the presence of incomplete biliary obstruction or when there is obstruction of only one hepatic duct—conditions under which the serum bilirubin level often is normal or only slightly elevated (19). In the present study, the majority of the patients had an elevated enzyme level of more than 200 U/L within 1 month after TACE. Although we did not compare the enzyme levels in these patients with those in control subjects, which properly would have included patients with small segmental infarctions or those without bile duct dilatation, we believe that a substantially elevated serum alkaline phosphatase level at about 1 month after TACE may be an indicator of TACE-induced bile duct injury. In three patients (patients 4, 7, and 11), the elevated enzyme level was checked before the CT appearance of bile duct dilatation, and the results suggest that a marked elevation of serum alkaline phosphatase is more sensitive than bile duct dilatation visualized at CT. However, at later follow-up examinations in the present study, a relationship between serum enzyme level and degree of bile duct dilatation at CT was not seen. It is believed that in the chronic stage of bile duct injury, the enzyme level depends on the enzyme-producing capability of atrophied hepatocytes and the degree or level of the bile duct strictures.

In conclusion, TACE-induced bile duct injury, including focal dilatation of the intrahepatic bile duct with or without extravasation of bile along connective tissue sheaths of the Glisson capsule, may obliterate the adjacent portal vein branch. Despite the limitation of having no reference standard such as cholangiography or pathologic findings to prove our beliefs, bile duct injury leading to obliteration of the adjacent portal vein branch should be regarded as one of the mechanisms of TACE-induced, gradual progressive parenchymal atrophic changes, as elucidated by CT findings. Bile duct injury associated with the obliteration of portal vein branches may occur in the noncirrhotic liver of patients after one TACE procedure and can be monitored by checking the serum alkaline phosphatase level 1 month after TACE.

### References

6. Doppman JL, Dunnick NR, Girton M, Fauzi AS, Popovska MA. Bile duct cysts

---

### Table 2

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Pre-TACE</th>
<th>Just after TACE</th>
<th>1 m</th>
<th>2 m</th>
<th>3 m</th>
<th>4 m</th>
<th>5 m</th>
<th>6 m</th>
<th>7 m</th>
<th>8 m</th>
<th>9 m</th>
<th>10 m</th>
<th>11 m</th>
<th>12 m</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>78</td>
<td>74</td>
<td>606</td>
<td>NA</td>
<td>151</td>
<td>119</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>2</td>
<td>54</td>
<td>103</td>
<td>310</td>
<td>310</td>
<td>NA</td>
<td>142</td>
<td>NA</td>
<td>87</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>3</td>
<td>167</td>
<td>190</td>
<td>215</td>
<td>NA</td>
<td>NA</td>
<td>497</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>295</td>
<td>NA</td>
<td>230</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>4</td>
<td>72</td>
<td>102</td>
<td>479</td>
<td>382</td>
<td>NA</td>
<td>583</td>
<td>386</td>
<td>NA</td>
<td>NA</td>
<td>404</td>
<td>NA</td>
<td>NA</td>
<td>289</td>
<td>NA</td>
</tr>
<tr>
<td>5</td>
<td>72</td>
<td>78</td>
<td>NA</td>
<td>NA</td>
<td>226</td>
<td>NA</td>
<td>205</td>
<td>NA</td>
<td>NA</td>
<td>138</td>
<td>NA</td>
<td>147</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>6</td>
<td>95</td>
<td>144</td>
<td>327</td>
<td>161</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>179</td>
<td>NA</td>
<td>741</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>7</td>
<td>84</td>
<td>87</td>
<td>215</td>
<td>358</td>
<td>606</td>
<td>2,314</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>8</td>
<td>142</td>
<td>144</td>
<td>396</td>
<td>NA</td>
<td>NA</td>
<td>207</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>9</td>
<td>79</td>
<td>109</td>
<td>NA</td>
<td>NA</td>
<td>137</td>
<td>NA</td>
<td>98</td>
<td>97</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>10</td>
<td>80</td>
<td>75</td>
<td>140</td>
<td>NA</td>
<td>202</td>
<td>119</td>
<td>103</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>11</td>
<td>135</td>
<td>137</td>
<td>201</td>
<td>NA</td>
<td>298</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>303</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

Note.—Data are serum alkaline phosphatase levels expressed in international units per liter. The data in columns 4–15 are serum alkaline phosphatase levels measured 1–12 months (m) after TACE. NA = not applicable—that is, enzyme levels were not tested at the given time.

---

![Graph](image-url)  
Figure 4. Graph illustrates serum alkaline phosphatase levels compared with time after TACE in nine patients in whom enzyme levels were tested within 1 month after TACE.


