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Letters

Acute renal failure after transarterial chemoembolization progressing to chronic renal failure in hepatocellular carcinoma

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Sir,

Hepatocellular carcinoma (HCC) has been widely treated with transarterial chemoembolization (TACE) by using various combinations of chemotherapeutic agents and embolic agents [1,2]. However, there have been no reports on the development of acute renal failure (ARF) after TACE in patients with HCC. We present a case of ARF after TACE, who did not recover normal renal function but instead progressed to chronic renal failure.

Case.

A 67-year-old female was admitted to hospital on July 2, 1998 with persistent nausea, vomiting and poor oral intake after TACE 9 days previously. On past history, she had been followed-up since 1993 due to liver cirrhosis by chronic viral hepatitis C infection. In May 1996, HCC of 4 cm size was detected on posterior inferior area of the right lobe of the liver and TACE was successfully performed with 30 mg of adriamycin without any complications. In June 1998, another 2 cm sized hepatic masses adjacent to the previous HCC were detected. She was admitted in June 18, 1998 for a second TACE. On admission, blood pressure was 130/70 mmHg, pulse rate was 88 per min, and body temperature was 36.3°C. There were no jaundice or ascites. White blood cell (WBC) count was 2200 cells/µl, haemoglobin (Hb) and haematocrit (Hct) were 10.7 g/dl and 30.5%, and platelets were 53 000 µl. Urinalysis was normal. Serum total protein was 5.3 g/dl, albumin 2.6 g/dl, total bilirubin 0.7

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ng/dl, AST 84 IU/l, ALT 74 IU/l, alkaline phosphatases 100 IU/l, BUN 11.9 mg/dl, creatinine 0.8 mg/dl, sodium 147 mM, potassium 4.2 mM, prothrombin time 15 s (INR 1.44), activated partial prothrombin time 35 s (normal 23–33 s), and α -fetoprotein 287 ng/ml (normal <20 ng/ml). On abdominal computerized tomography, previous HCC with lipiodol uptake remained unchanged on segment 6 and low attenuated masses of 2x1.5 cm were found. Kidneys were normal. On the sixth hospital day, TACE with cisplatin 100 mg, lipiodol 10 ml, and gelfoam was performed on right posterior inferior branch of hepatic artery. Total duration of TACE was 2 h and 20 min. The patient complained of persistent nausea and vomiting 20 min after TACE, which was controlled with intravenous metoclopramide injection. On the seventh hospital day, BUN 14.4 mg/dl, creatinine 1.0 mg/dl, total bilirubin 1.4 mg/dl, and AST and ALT were 98 and 72 IU/l respectively. She was discharged after 10 days in hospital but 5 days later she was readmitted due to persistent nausea, vomiting and severe weakness. On re-admission, blood pressure was 180/100 mmHg, pulse rate 64 per min, and body temperature 36.3°C. Skin turgor was poor and tongue was severely dry. Hb was 11.6 g/dl, Hct 37.2%, WBC 3600 cells/ μ l, platelet, 70 000 cells/ μ l, BUN 85.2 mg/dl, creatinine 4.3 mg/dl, AST 65 IU/l, ALT 53 IU/l, total bilirubin 2.2 mg/dl, amylase 153 U/dl, sodium 118 mM/l, potassium 3.0 mM, chloride 72 mM, prothrombin time 13.8 s (INR 1.18) and activated PTT 35 s. On arterial blood gas analysis, pH was 7.56, PaCO₂ 23.9 mmHg, PaO₂ 118.4 mmHg, and HCO₃⁻ 21.3 mM. Urinalysis was normal. On spot urine analysis, sodium was 43.9 mM, potassium 32.1 mM, chloride 49.4 mM, creatinine 85.0 mg/dl and osmolality 452 mosm/kg. Renal ultrasonography and blood flow examination was normal. Despite adequate hydration serum creatinine did not return to normal from 4.3 mg/dl on the first day in hospital to 2.7 mg/dl on day 16. After discharge, serum creatinine rose to 4.0 mg/dl in September 1998, and the changes of serum creatinine until May 1999 are shown in Figure 1.

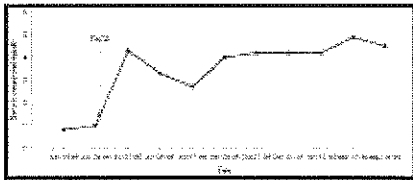


Fig. 1 Serial changes of serum creatinine concentrations after TACE.

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Comment.

Side effects of TACE include postembolization syndrome, abdominal pain, liver damage, and acute gastric mucosal lesions. Postembolization syndrome includes daily intermittent fever below 39°C, abdominal pain, nausea and vomiting, abdominal fullness, and appetite loss [2]. Those symptoms usually diminish spontaneously within a couple of weeks. A severe abdominal pain may result from gall bladder infarction resulting from cystic artery embolization, pancreatitis, gastroduodenal ulcer, or others. Liver damage is transient and liver function tests generally return to original level subsequently. Acute gastric mucosal lesions occur frequently and require medication.

TACE expose to several risks for the development of ARF. First, considerable amounts of radiocontrast are used for TACE. Thus, contrast media-associated nephrotoxicity can be developed in patients with volume depletion. In a number of patients with HCC, liver cirrhosis in which patients are vulnerable to the development of ARF are also present. Second, postembolization syndrome which includes fever, abdominal pain, nausea, vomiting, abdominal fullness, and appetite loss can induce volume depletion and thereby lead to the development of ARF. Third, acute hepatic failure during which ARF

In the present case, we could not perform renal biopsy due to patient's poor condition. There was no evidence of renal infarction. We suspect both the use of radiocontrast and the postembolization syndrome as possible causes of ARF in this case. The patient developed severe nausea, vomiting and appetite loss without adequate hydration after TACE. After discharge, she ate almost nothing due to vomiting for 5 days. Thus, prolonged volume depletion led to the development of prerenal ARF which progressed to ischaemic acute tubular necrosis and irreversible CRF. We could not find any report on ARF after TACE in MEDLINE search. This is, as far as we know, the first case of ARF after TACE. We suggest that the renal function of patients with HCC should be cautiously monitored after TACE.

References

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