Cardiac failure, hepatic congestion and increased level of serum carcinoembryonic antigen

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Summary

A 65-year-old woman undergoing chronic haemodialysis for chronic nephritis had transiently elevated serum carcinoembryonic antigen levels, up to 26 ng/ml for nearly 3 months. This elevation was most probably due to cardiac failure with hepatic congestion and pulmonary oedema, because the antigen level returned to normal when the cardiac failure was properly treated, and comprehensive examinations revealed no other cause. During follow-up for 18 months, the carcinoembryonic antigen level has remained normal.

KEY WORDS: carcinoembryonic antigen, cardiac failure, hepatic congestion, interstitial nephritis, renal failure.

Introduction

The serum concentration of carcinoembryonic antigen (CEA) may be increased in patients with various malignant neoplasms, such as cancers of the colon, stomach, oesophagus, pancreas, thyroid gland, lung, breast, ovary, prostate or urinary bladder, as well as occasionally in neuroblastoma, multiple myeloma, osteogenic sarcoma and melanoma (Zamcheck *et al.*, 1972; Shuster, Freedman and Gold, 1977; Char and Christensen, 1980; Madeddu *et al.*, 1980). Several more benign conditions, such as colorectal polyps, diverticulitis, ulcerative colitis, pancreatitis, hepatitis, acute toxic liver damage, liver cirrhosis, obstruction of extrahepatic bile ducts by gall-stones, chronic pulmonary diseases, benign breast diseases and hypothyroidism may cause elevation of CEA and so may smoking (Zamcheck *et al.*, 1972; Shuster *et al.*, 1977; Lurie, Loewenstein and Zamcheck, 1975; Bullen *et al.*, 1977; Wilkinson *et al.*, 1980; Amino *et al.*, 1981), although the elevation is then usually slight. Here we describe, to our knowledge for the first time, a patient who transiently had distinctly elevated serum CEA levels, for which no other cause was found except cardiac failure with liver congestion and pulmonary oedema.

Case report

A 65-year-old female, undergoing chronic haemodialysis, had had recurrent urinary tract infections for over 20 years and renal biopsy showed chronic interstitial nephritis and urography revealed deformed papillae. Because of uraemia, haemodialysis was started in November 1980. Simultaneously, arterial hypertension, mitral insufficiency and ischaemic cardiac disease resulting in cardiac failure were diagnosed. Since then medical treatment has included digitalis, metoprolol, prazosin, isosorbide, aluminium hydroxide and B vitamins.

In September 1981 the patient had increasing dyspnoea, and decompensated cardiac failure was detected. Bilateral rales were heard and the jugular veins were seen to be distended. Chest X-ray showed pulmonary oedema and pleural fluid on the left.

The previously normal liver function tests were abnormal: aspartate and alanine aminotransferases rose to 169 and 505 u/litre (normal upper limit 40 u/litre), alkaline phosphatase to 461 and gamma-glutamyl transferase to 364 u/litre (normal upper limits 280 and 46 u/litre). Serum bilirubin and lipase values remained normal, and no antibodies to mitochondria, smooth muscle cell, hepatitis A and B and cytomegalovirus were detected. No hepatitis B surface antigen was found. In May 1981, because of pain in the lower abdomen, CEA had been measured by radioimmunoassay as described by Rutanen *et al.* (1978), and had been normal (2.5 ng/ml) but now rose, being at the acute phase 15.0 ng/ml and 2 weeks later 26.0 ng/ml. Two months later, the concentration was 7.9 ng/ml and after 4 months the level was normal. Concomitantly, the pulmonary oedema and pleural fluid disappeared gradually, and the size of liver returned to normal when the cardiac failure was treated by more effective haemodialysis.

No neoplasm was found on the clinical examination or in the following examinations: X-rays of the colon, stomach, chest and bones, computerized tomo-

graphy of the trunk, bone marrow aspirate, and radioisotope liver scan. Ultrasound of the upper abdomen at the acute phase revealed a slightly enlarged liver, but no dilated bile ducts or tumours. The serum thyroxine and tri-iodothyronine levels were normal.

During follow-up for 18 months serum CEA, aspartate and alanine aminotransferase, alkaline phosphatase and gamma-glutamyl transpeptidase levels have been normal and no signs of neoplasms have been found.

Discussion

In our case nothing was found to explain the transient rise of CEA concentration apart from cardiac failure with resulting pulmonary oedema and liver congestion. To our knowledge no similar case has been reported earlier. It is not surprising that such damage should raise the level of serum CEA, for lesions of hepatic parenchyma and benign obstruction of the bile ducts have been shown to cause such an elevation (Lurie *et al.*, 1975; Bullen *et al.*, 1977; George *et al.*, 1982). However, the elevation observed here was remarkably high and long-lasting. Lurie *et al.* (1975) examined 29 patients with jaundice attributable to benign extrahepatic biliary tract obstruction and inflammation. In 48% the CEA was normal, in 35% 2.5–5.0 ng/ml and in 17% 5.1–10.0 ng/ml. After relief of the obstruction, the CEA level usually returned to normal within 3 weeks. Serum alkaline phosphatase levels showed a positive correlation with CEA levels. Using radioimmunoassay Bullen *et al.* (1977) observed slight but significant elevation of CEA levels in 50% of 16 patients with acute toxic or infectious liver damage. George *et al.* (1982) examined 14 persons suffering from fulminant hepatitis and in all except one the CEA, measured by radioimmunoassay, was found to be elevated. In only three did the value exceed 10 ng/ml, the maximum being 21 and the mean 7.0 ng/ml. No correlation was found between CEA level and serum bilirubin, alkaline phosphatase, aspartate aminotransferase or alanine aminotransferase.

The reason for raised CEA levels in liver diseases is not known. It may be that damaged liver cells release this antigen or are unable to metabolize it owing to liver cell damage or increased production elsewhere in the tissues. Another possible explanation would be

failure of excretion into the bile, or even increased hepatic synthesis during reparative growth (Lurie *et al.*, 1975; Bullen *et al.*, 1977; George *et al.*, 1982). Whatever the mechanism, our case suggests that even cardiac failure can cause significant elevation of CEA levels, probably mediated by liver damage and possibly also by pulmonary oedema. To exclude the possibility that elevation of CEA might be due to the decreased renal function, we measured the CEA levels in 10 other patients undergoing chronic haemodialysis as well as in another 10 uraemic patients not undergoing haemodialysis treatment, and found no abnormal values.

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