—Report on Experiments and Clinical Cases—

Spontaneous Disappearance of a Hepatic Cyst

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Abstract

The spontaneous disappearance of a hepatic cyst is described. A 62-year-old woman presented with symptoms of general fatigue in August 1992. Her past medical history was significant for chronic hepatitis, which was diagnosed in 1990 but not treated. Initial laboratory tests revealed mild liver dysfunction with a positive serologic test for hepatitis C. In August 1992, ultrasonography and computed tomography disclosed a cystic lesion along the middle hepatic vein in the right anterior segment of the liver, which was 40 mm in diameter. Repeat radiologic studies in June 1994 demonstrated that the size of the cyst was unchanged. In May 1995, the cyst was only 25 mm in diameter, and it continued to decrease in size thereafter, to 10 mm in September 1995 and 7 mm in September 1996. No hepatic cyst was visualized in December 1996, though the region in which the cyst existed was hyperechoic. Laboratory data were essentially unchanged from August 1992 to December 1996. Clinically the patient remained asymptomatic. (J Nippon Med Sch 2001; 68: 58—60)

Key words: spontaneous disappearance, liver cyst

Introduction

Before ultrasonography and computed tomography (CT) were generally available, hepatic cysts were thought to be rare because they were encountered only when they were exceptionally large or caused symptoms[1]. Systematic surveys using advanced noninvasive imaging techniques have revealed that asymptomatic hepatic cysts are in fact common. This report describes the spontaneous disappearance of a hepatic cyst.

Case Report

A 62-year-old woman presented with symptoms of general fatigue in August 1992. Her past medical history was significant for chronic hepatitis, which was diagnosed in 1990 but not treated. Initial laboratory tests revealed the following: glutamic oxaloacetic transaminase, 160 IU/L (normal, < 31 IU/L), glutamic pyruvic transaminase, 226 IU/L (normal, <31 IU/L), alkaline phosphatase, 235 IU/L (normal 98 to 279 IU/L), lactic dehydrogenase, 523 IU/L (normal, 199 to 486 IU/L), gamma glutamic transpeptidase, 54 IU/L (normal, 8 to 45 IU/L), zinc sulfate turbidity test, 21.6 K-U (normal, 4 to 12 K-U), and platelet count, 40,000/μL (normal 200,000 to 400,000/μL). A serologic test for hepatitis C was positive. In August 1992, ultrasonography and CT revealed a cystic lesion along the middle hepatic vein in the right anterior segment of the liver, which was 40 mm in diameter. Periodic studies for chronic hepatitis were perfomed as follows Repeat radiologic studies in June 1994 revealed no

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change in the size of the cyst. However, despite the absence of treatment, in May 1995 the cyst was only 25 mm in diameter, and it continued to decrease in size thereafter, to 10 mm in diameter in September 1995 and 7 mm in diameter in September 1996. No hepatic cyst was visualized in December 1996, though the region in the area of the cyst appeared hyperechoic (**Figs. 1 and 2**). Laboratory data were essentially unchanged from August 1992 to December 1996. The patient never had symptoms attributable to her cyst.

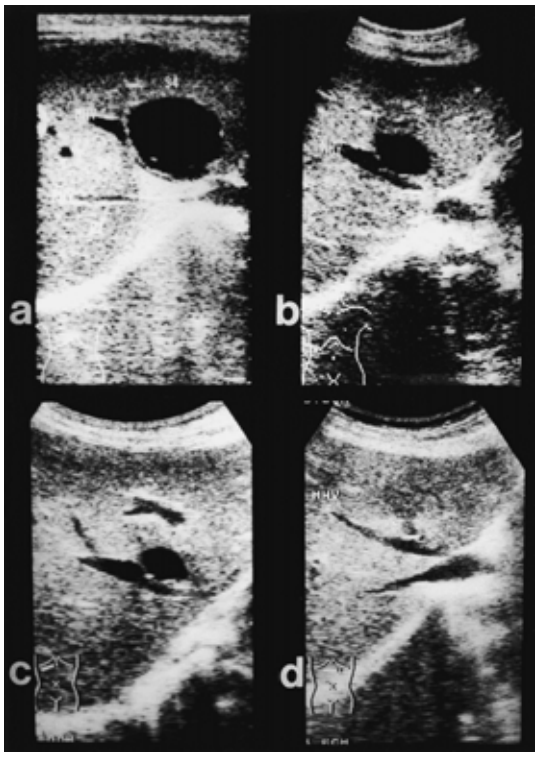

Fig. 1 Ultrasonography (a) 40 mm in diameter in June 1994. (b) 25 mm in diameter in May 1995. (c) 10 mm in diameter in September 1995. (d) No hepatic cyst was visualized in December 1996, though the region in the area of the cyst appeared hyperechoic.

Discussion

Large or multiple hepatic cysts can produce vague discomfort or upper abdominal pain. Recommended treatments for symptomatic hepatic cysts include surgery[1] and the injection of a sclerosing agent into the cyst[2–5]. However, small hepatic cysts are usually asymptomatic and are detected incidentally in radiologic studies of the liver.

The prevalence of congenital hepatic cysts is reported to be 2 to 4%[6]. Gaviser[7] has reported that the wall of a hepatic cyst consists of three layers: an inner layer of loose connective tissue lined with cylindrical or cuboidal epithelium; a middle layer of compact connective tissue containing blood vessels; and an outer layer of loose connective tissue with large blood vessels, bile ducts and occasional von Meyenburg complexes. The epithelial lining may necrose and slough if the intracystic pressure becomes too high. This anatomy accounts for the inability to enucleate hepatic cysts without causing significant bleeding.

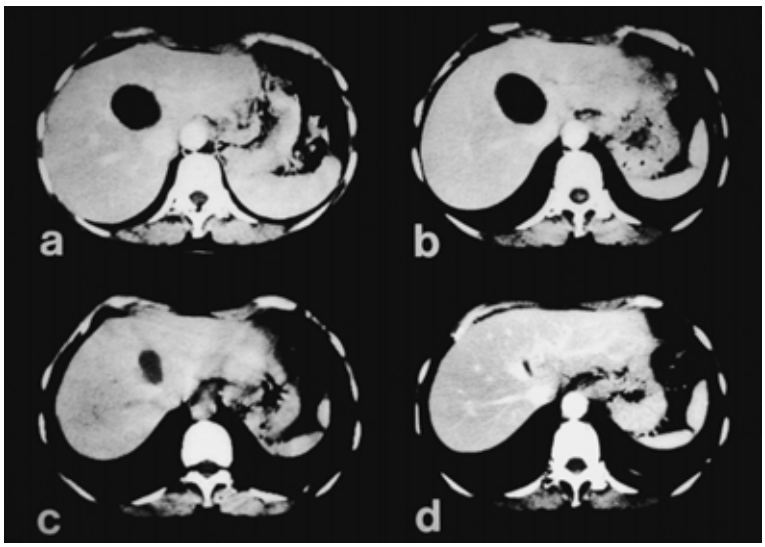

Fig. 2 Computed tomography (a) 40 mm in diameter in August 1992, (b) 40 mm in diameter in June 1994, (c) 25 mm in diameter in May 1995, (d) No hepatic cyst was visualized in Nobember 1996.

Moschcowitz[8] has attributed cyst formation to obstruction of aberrant bile ducts. Melnick[9] felt that the presence of many islands of ductular epithelium in the cyst wall was confirmation of this hypothesis. These islets of bile ducts that are discontinuous with the portal spaces are what are now called von Meyenburg complexes[10]. Kida et al.[11] reported that intrahepatic peribiliary glands in livers with adult-type polycystic disease are markedly dilated in contrast to normal livers, which show no or minimal dilatation. Uddin et al.[12] reported that most cysts are lined by flattened biliary epithelium and that von Meyenburg complexes are filled with inspissated bile and scattered throughout the intervening parenchyma. Sinusoidal dilatation is extensive, and congestion associated with hepatocyte loss affects acinar zones 2 and 3, and to a lesser extent, focal areas of zone 1. The lumens of small supralobular branches of the hepatic vein are narrowed or occluded in these areas by loose fibrous tissue containing entrapped red cells and demonstrate neolumena consistent with organized thrombi.

We have previously reported success in treating a symptomatic hepatic cyst by the injection of minocycline hydrochloride[2].The acidity of minocycline hydrochloride kills the cyst's secretory cells, leading eventually to reabsorption of the cyst[13]. In the present case, the cyst lay along the middle hepatic vein in the right anterior segment of the liver. Consequently it is possible that cystovenous communication might have accounted for cyst drainage. More likely, however, reabsorption was more active than secretion, and the cyst involuted spontaneously. This change may have been due to necrosis of the secretory cells by increased intracystic pressure, or possible ischemic necrosis due to pericystic scarification resulting from local inflammation, conceivably a consequence of cystic leakage.

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