Comment

Super Glue is a cyanoacrylate monomer made by condensation of formaldehyde and a cyanoacetate. It undergoes rapid anionic polymerisation in the presence of even trace amounts of weakly basic substances; the reaction proceeds unless acid is encountered.² It thus bonds tissue in seconds. Higher alkyl derivatives (butyl, decyl) have lower tissue toxicity and have found therapeutic use in the fashioning of temporary tarsorrhaphies and in the temporary patching of corneal defects.^{2,3}

The figure shows the similarity between the bottle of adhesive and the bottle of eye drops. Indeed, both containers were made by the same manufacturer. Other bottles in our ward drug store were also similar. This surely represents an unnecessary hazard, especially for patients with deficient eyesight, which is easily avoidable.

The manufacturers of the adhesive (Bostik), who have not heard of a case similar to ours, were asked to comment and to consider changing their packaging. They responded, "It so happens that we are . . . planning changes to the bottle for other reasons and in doing so we shall certainly take the point made . . . into consideration."

We would like to see a ban imposed on the sale of non-pharmaceutical chemicals in small plastic dropper bottles, as many alternative packagings are available.

¹ Margo CE, Trobe JD. Tarsorrhaphy from accidental instillation of cyanoacrylate into eye. *JAMA* 1982;247:660-1.

² Refojo MF, Dohlman CH, Koliopoulos J. Adhesives in ophthalmology: a review. *Surv Ophthalmol* 1971;15:217-36.

³ Ellis RA, Levine AM. Experimental sutureless ophthalmic surgery. *Am J Ophthalmol* 1963;55:733-41.

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Primary liver cell carcinoma complicating secondary biliary cirrhosis

Over recent years there have been reports of primary liver cell carcinoma complicating most types of cirrhosis, including autoimmune chronic active hepatitis¹ and primary biliary cirrhosis,² but none in relation to secondary biliary cirrhosis. We report a case of primary liver cell carcinoma in a patient with secondary biliary cirrhosis due to a postoperative biliary stricture.

Case report

A 68 year old woman presented to this hospital in September 1982 with jaundice and gastrointestinal bleeding. She was a teetotaler and had never taken potentially hepatotoxic drugs. At the age of 40 she had undergone a cholecystectomy for gallstone disease. Over the next three years she had two further operations in which attempts were made to repair a benign stricture of the proximal common bile duct. She remained well until 1978, when she again developed recurrent bouts of cholangitis. On each occasion her symptoms settled with oral antibiotics.

In August 1982 she was admitted to her local hospital because of vomiting, increasing jaundice, itch, and abdominal distension. Examination showed gross ascites and peripheral oedema but none of the peripheral stigmata of chronic liver disease. Haemoglobin concentration was 9.8 g/dl, and red cells showed morphological features of iron deficiency. Liver function tests yielded bilirubin concentrations of 147 $\mu\text{mol/l}$ (8.6 mg/100 ml) (normal < 23 $\mu\text{mol/l}$ (< 1.3 mg/100 ml)); serum aspartate transferase activity 157 IU/l (normal < 50 IU/l); serum alanine transferase 67 IU/l (normal < 50 IU/l); alkaline phosphatase 256 IU/l (normal < 110 IU/l); γ glutamyltransferase 157 IU/l (normal 0-40 IU/l); albumin concentration 25 g/l (normal > 35 g/l); lactate dehydrogenase activity 686 IU/l (normal < 300 IU/l); and prothrombin time 70%. Autoantibodies and hepatitis B surface antigen were not detected. Abdominal paracentesis yielded an evenly bloodstained transudate that was negative on cytological examination. Ultrasound examination of the liver showed mild dilatation of the intrahepatic ducts. After treatment with diuretics had reduced her ascites a percutaneous liver biopsy showed established cirrhosis, with regenerative nodules of parenchyma separated by broad bands of fibrous tissue containing numerous bile ducts and acute and

chronic inflammatory cells. This was thought to be consistent with biliary cirrhosis.

Her clinical course was complicated by haematemesis, which was shown by endoscopy to be due to oesophageal varices. Further investigation showed portal hypertension (splenic pulp pressure 16 mm Hg) and patent splenic and portal veins. A percutaneous transhepatic cholangiogram established that biliary drainage was completely blocked at the level of the proximal common bile duct. As variceal bleeding persisted portosystemic shunt surgery was performed to achieve decompression. Her postoperative course was compli-

Postmortem liver biopsy specimen showing hepatocytes separated from tumour cells by a broad band of fibrous tissue. Haematoxylin and eosin $\times 99$.

cated by increasing jaundice. Resuscitation was slowly reduced when the preoperative serum α fetoprotein concentration was found to have been 74 mg/l, and she died one week later. Necropsy disclosed a small cirrhotic liver with multifocal primary liver cell carcinoma invading intrahepatic vascular channels (figure) and a benign stricture of the proximal common bile duct.

Comment

Primary liver cell carcinoma as a complication of secondary biliary cirrhosis has not been reported previously. Though Purtilo and Gottlieb described 10 cases of malignant liver disease (primary liver cell carcinoma and cholangiocarcinoma) complicating unspecified biliary cirrhosis, they included patients with cholangiocarcinoma in whom the tumour may have been the cause of the cirrhosis. Moreover, serological evidence that might have excluded primary biliary cirrhosis was not available in most of their patients.

The diagnosis of secondary biliary cirrhosis and primary liver cell carcinoma in this patient was supported by the consistent clinical history of biliary surgery and recurrent biliary sepsis; the presence of a benign stricture of the proximal common bile duct, which was confirmed radiologically and histologically; compatible hepatic and tumour histology; and an appreciably raised serum α fetoprotein concentration.

It appears that primary liver cell carcinoma may complicate the clinical course of any patient with cirrhosis. Thus this diagnosis should be considered in patients with secondary biliary cirrhosis in whom hepatocellular function deteriorates without obvious cause.

¹ Jakobovits AW, Gibson PR, Dudley FJ. Primary liver cell carcinoma complicating auto-immune chronic active hepatitis. *Dig Dis Sci* 1981;26:694-9.

² Krasner N, Johnson PJ, Portmann B, Watkinson G, MacSween RNM, Williams R. Hepatocellular carcinoma in primary biliary cirrhosis: report of four cases. *Gut* 1979;20:255-8.

³ Purtilo DT, Gottlieb LS. Cirrhosis and hepatoma occurring at Boston City Hospital (1917-1968). *Cancer* 1973;32:458-62.

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