

Biliary scintigraphy with DISIDA

A simpler way of showing bile duct patency in suspected biliary atresia

M C DICK AND A P MOWAT

Department of Child Health, King's College Hospital, London

SUMMARY ^{99m}Tc -diisopropyl iminodiacetic acid (DISIDA) scintigraphy after oral phenobarbitone treatment accurately indicated bile duct patency or obstruction in 28 of 32 (87%) infants, aged less than 12 weeks, with suspected biliary atresia.

This investigation is more rapid than and as accurate as the ^{131}I Rose-Bengal faecal excretion test.

Demonstration of bile duct patency using radionuclides is often an essential investigation in distinguishing extrahepatic biliary atresia from intrahepatic disease causing severe cholestasis. The improved results from portoenterostomy for biliary atresia, particularly if performed in the first 2 months of life,¹ together with the deleterious effect of inappropriate laparotomy and biliary surgery for infants with hepatic disease,² make early accurate diagnosis essential.

Difficulties with the ^{131}I Rose-Bengal faecal excretion test, which requires a 72 hour stool collection, have prompted consideration of new isotope excretion tests. Iminodiacetic acid (IDA) compounds are rapidly removed from the circulation by the liver and excreted in bile and, when labelled with ^{99m}Tc , the biliary-intestinal excretion may conveniently be shown with a gammacamera.

A new IDA compound, diisopropyl IDA (DISIDA) with improved lipid solubility allowing hepatic uptake at a high serum bilirubin concentration and with a shorter hepatobiliary transit time, has theoretical advantages over earlier IDA compounds, which often fail to distinguish biliary atresia from hepatic disease.³ We report an assessment of the value of DISIDA scintigraphy in 50 consecutive infants investigated because of conjugated hyperbilirubinaemia in the first 12 weeks of life.

Patients and methods

All 50 infants were investigated for possible genetic, metabolic, infective, and structural causes of hepatobiliary disease. DISIDA scintigraphy was not carried out in 18 infants as they had either pigmented stools (13), α_1 antitrypsin deficiency (PiZ)

previously diagnosed by phenotyping (four), or the presence of a choledochal cyst on ultrasound (one). DISIDA scintigraphy was performed in 32 infants in whom distinction between biliary atresia and hepatic disease was difficult. Of these, 21 subsequently had ^{131}I Rose-Bengal faecal excretion studies.

Radionuclide tests. In 32 infants 40 mBq (1 mCi) ^{99m}Tc -DISIDA was injected intravenously after a three day course of oral phenobarbitone (5 mg/kg/day). Imaging was carried out at five minute intervals for the first hour and then at hourly intervals for 10 hours or until radioactivity was seen in the intestines. If excretion was still not apparent imaging was repeated at 24 hours. In 21 infants 0.2 mBq (5 μCi) ^{131}I Rose-Bengal was injected intravenously. Stools were then collected for three consecutive days, taking care to avoid urinary contamination.

Results

Diagnoses and results of the radionuclide tests are shown in the Table. There was no biliary excretion of DISIDA in 23 infants. Biliary atresia was confirmed at laparotomy in 20 of these. In two infants liver biopsy specimens showed features of bile duct hypoplasia, and their subsequent course confirmed the diagnosis. The other child had histological features of biliary atresia, but laparotomy was not performed because of advanced liver disease with ascites. No post mortem was performed. There was delayed excretion of tracer at 24 hours in three cases: one had a bile duct plug diagnosed at laparotomy, one had α_1 antitrypsin deficiency, and one had severe familial neonatal hepatitis of unknown aetiology. Excretion of DISIDA was evident by three hours in six infants, one with α_1 antitrypsin deficiency and five with hepatic disease suggested by liver biopsy and confirmed by subsequent clearing of jaundice. ^{131}I Rose-Bengal faecal excretion was less than 10% in all 14 with biliary atresia and in four others with acholic stools, one of whom had α_1 antitrypsin deficiency (delayed excretion of DISIDA) and three with intrahepatic disease (one of whom had no excretion of DISIDA). Diagnostic accuracy was therefore 87% (28 out of 32) for DISIDA and 81% (17 out of 21) for Rose-Bengal.

Table *Diagnoses and results of the radionuclide tests in the 50 infants*

Diagnosis	No	DISIDA				Rose-Bengal		
		No excretion	Rapid excretion	Delayed excretion	Not done	<10%	>10%	Not done
Biliary atresia	21	20	0	0	1	14	0	7
Bile duct plug	1	0	0	1	0	0	0	1
α_1 Antitrypsin deficiency	6	0	1	1	4	1	0	5
Hepatic disease	22	3	5	1	13	3	3	16
Total	50	23	6	3	18	18	3	29

Discussion

A skilfully interpreted percutaneous liver biopsy specimen and ^{131}I Rose-Bengal faecal excretion test have been shown to discriminate between biliary atresia and hepatic disease once genetic disorders have been excluded.³ Neither test alone is reliable. The use of ^{131}I as a tracer in infancy is not ideal as it is a beta emitter with a long half life, although the dose is very small. The test also involves collection of stools for three days, carefully avoiding urinary contamination that may invalidate the result. This study suggests that DISIDA scintigraphy after treatment with phenobarbitone is as accurate as the Rose-Bengal test. Whether it is necessary to use phenobarbitone is uncertain.^{4,5} It was used in this study because five of seven infants with hepatic disease had no excretion of paraisopropyl IDA when tested without phenobarbitone but did excrete after treatment with phenobarbitone.⁶

DISIDA scintigraphy has the drawback that it is subjective, allowance having to be made for isotope in the kidneys and bladder, and time consuming. Assessment of hepatic uptake of DISIDA may increase diagnostic accuracy.^{4,5} Nevertheless, when used in conjunction with ultrasonography, tests for infective and genetic causes of liver damage, and

percutaneous liver biopsy DISIDA scintigraphy represents a considerable advance in distinguishing complete from partial cholestasis in early infancy.

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Correspondence to Dr A P Mowat, Consultant Paediatrician, Department of Child Health, King's College Hospital, Denmark Hill, London SE5 8RX.

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Splenunculectomy in thrombocytopenic purpura

M M REID, P W G SAUNDERS, J D FENWICK, AND W WALKER

Departments of Haematology, Royal Victoria Infirmary and Newcastle General Hospital, and Regional Medical Physics Department, Newcastle General Hospital, Newcastle upon Tyne

SUMMARY In six patients with thrombocytopenic purpura not cured by splenectomy platelets fell to $<150 \times 10^9/l$ within six weeks of splenectomy. They 'failed to respond'. Two underwent splenuncu-

lectomy without improvement. Splenunculectomy offered little chance of improvement to published cases who failed to respond but may well be helpful after true relapse.