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# Gallbladder contraction in biliary atresia: a pitfall of ultrasound diagnosis

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**Abstract** In 3 (9%) of 34 children with biliary atresia, US revealed gallbladder contraction following an oral feed, given on admission, but not with subsequent feeds. Surgery revealed a Kasai type IIIa biliary atresia with a patent communication between the gallbladder and duodenum. We propose that the bile ducts may initially have been patent, but then gradually became obliterated secondary to inflammation. These cases may explain the development of one type of biliary atresia.

## Introduction

Advances in US imaging have facilitated the differentiation of neonatal hepatitis from biliary atresia [1–6]. In biliary atresia, the biliary tree is occluded and gallbladder contraction is unlikely after oral feeding [1, 5, 6]. Conversely, in neonatal hepatitis, US will reveal contraction of the gallbladder after oral feeding [6]. However, in some patients with neonatal jaundice, it can be difficult to distinguish these entities. We report three children with biliary atresia in whom the gallbladder contracted after the first feed. Mechanisms of gallbladder contraction and pitfalls in the US diagnosis are discussed.

## Materials and methods

From June 1986 to November 1996, 72 Japanese infants with neonatal jaundice were investigated. The mean total bilirubin level was over 2.0 mg/dl. On admission, all underwent serial US to differentiate neonatal hepatitis from biliary atresia. US was performed before, during and after a milk feed. The fasting period before feeding was termed "before feeding", and the 2–5 min after the start of this feed was termed "during feeding". The term "after feeding" represented the 15- to 45-min period after the end of the feed. The US examination involved identification of the gallbladder and observation of contraction after oral feeding, using a high-resolution, real-time B-mode ultrasound scanner (SAL 50 A with a 5-MHz probe, Toshiba, Tokyo, Japan).

## Results

Thirty-four (47%) of 72 children had biliary atresia, as confirmed at surgery, while the others had neonatal hepatitis. The gallbladder in all patients with neonatal hepatitis was identified before or during feeding, and it contracted after feeding. In 8 (24%) of the 34 cases of biliary atresia, the gallbladder could not be identified before, during or after feeding. In the remaining 26 infants, a small gallbladder, or a cystic lesion, was evident before feeding, but the size did not change following feeding. All underwent cholangiography (Fig. 1) and biliary atresia was confirmed.

In three infants (two boys, one girl, all aged 1 month) with biliary atresia, US showed a gallbladder of normal

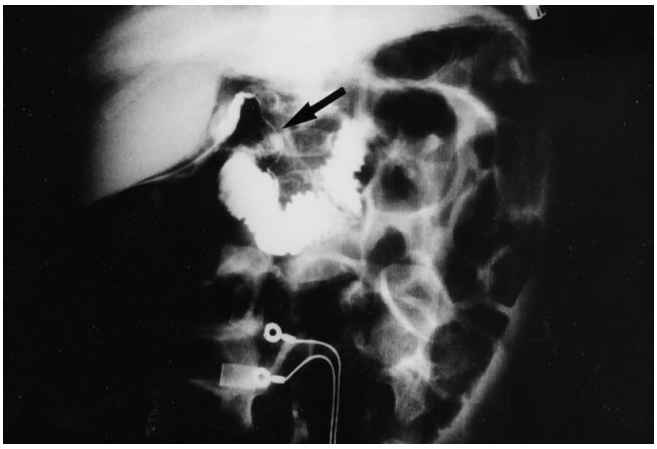

**Fig. 1** Intra-operative cholangiography showing a patent common bile duct. Retrograde filling of intrahepatic and common hepatic ducts did not occur. *Arrow* Common bile duct

size, which contracted after oral feeding (Fig. 2). Initially, these patients were diagnosed as having neonatal hepatitis, but jaundice did not decrease, despite medical treatment. When the US examination was repeated 2 weeks later, we found that the gallbladder did not contract after feeding (Fig. 3). At surgery, Kasai type IIIa biliary atresia was evident.

## Discussion

The precise aetiology of biliary atresia is unknown and was originally thought to be a congenital anomaly resulting from maldevelopment of the biliary tree, possibly due to failure of recanalization [7]. Danks suggested a genetic cause in at least a proportion of cases [8]. Malunion of the pancreatic and biliary systems has also been implicated [9], as has a fetal vascular accident [10]. Reovirus type 3 infection was suggested by Morecki et al. [11]. Some infants with definite bile-stained stools at birth eventually develop biliary obstruction, indicating that biliary atresia may be an acquired condition.

Several diagnostic tests to differentiate neonatal hepatitis from biliary atresia have been proposed and evaluated [12–14]. Part, or all, of the bile ducts in infants with biliary atresia are obliterated, but in babies with neonatal hepatitis, the biliary tree is patent, allowing the gallbladder to contract after feeding and these entities to be differentiated [6]. In our experience, in 97/100 normal children and in all of 31 children with neonatal hepatitis, the gallbladder could be identified, and changes in size of the gallbladder following oral feeding were clear. In 8/13 infants with biliary atresia, we identified a small gallbladder, the size of which was not altered by oral feeding. In the other patients, the gallbladder was not identified before, during or after feeding. In the three cases presented here, US revealed a gallbladder which contracted following initial oral feeding but not on subsequent feeding. All proved to be cases of Kasai type IIIa biliary atresia in which the hepatic ducts are obliterated but the cystic and common bile ducts are patent [15]. Weinberger et al. have reported the gallbladder of a patient with Kasai type IIIa biliary atresia to contract after feeding [16]. Kasai type IIIa is common, and Ohi and Ibrahim have reported that 19% of patients with biliary atresia had a patent communication between the gallbladder and duodenum [17].

In our series, 12 (35%) of 34 cases of biliary atresia were Kasai type IIIa but the gallbladder contracted after the initial feed in only 3 patients, and 2 weeks later, the gallbladder no longer contracted. Based on these observations, we believe that the bile ducts were initially patent but then gradually became obliterated due to an inflammatory process. If this notion is valid, then acquired biliary atresia can be considered.

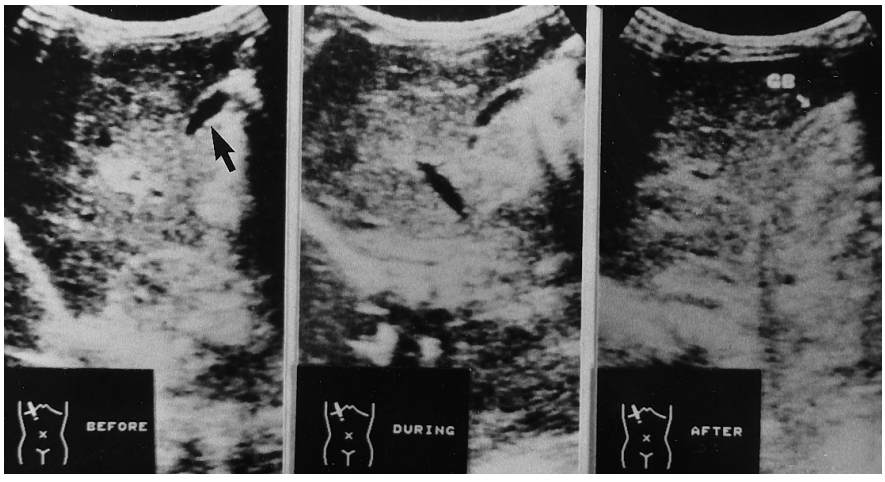


**Fig. 2a–c** US of the gallbladder in a patient with biliary atresia Kasai type IIIa, on admission. **a** Before feeding; **b** during feeding; **c** after feeding. The gallbladder contracted after feeding. *Arrow* Gallbladder

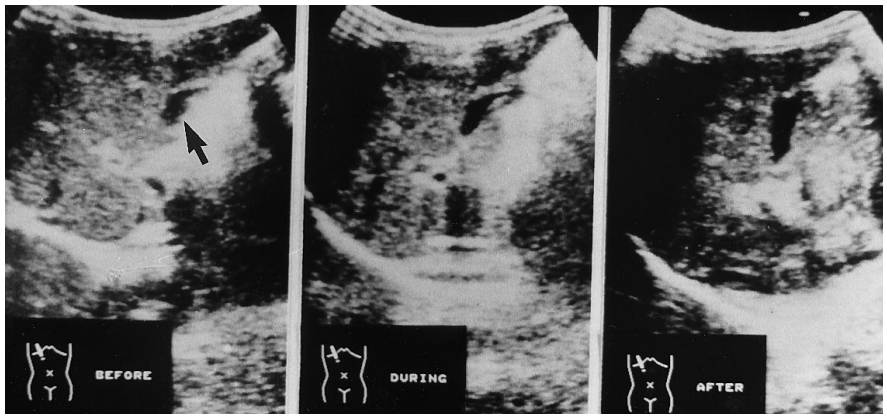


**Fig. 3a–c** US of the same patient as in Fig. 2, 2 weeks after admission. The gallbladder did not contract

To distinguish between biliary atresia and neonatal hepatitis, we suggest that if no change is detectable on US following oral feeding, a diagnosis of biliary atresia is likely to be accurate. Conversely, if the gallbladder can be detected and its size changes following oral feeding, most patients will have neonatal hepatitis but a few will have biliary atresia. In cases of neonatal hepatitis in which the gallbladder no longer contracts, urgent surgery will be required to exclude a possible diagnosis of acquired biliary atresia.

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