DIAGNOSTIC NUCLEAR MEDICINE

Tc-99m IDA Cholescintigraphy in Acute Pancreatitis: Concise Communication

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Recently it has been suggested that cholescintigraphy is unreliable in the detection of acute cholecystitis when acute pancreatitis is present. During a recent 17-month interval, twenty-one patients with a firmly established diagnosis of acute pancreatitis underwent cholescintigraphy in our laboratory. The gallbladder failed to visualize in only five cases, all of whom had acute cholecystitis. These data, and those available in the literature, lead us to conclude that cholescintigraphy is useful in the diagnosis of acute cholecystitis whether or not acute pancreatitis is present.

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Acute upper abdominal pain, nausea, vomiting, and fever are presenting symptoms common to acute cholecystitis and acute pancreatitis. Evaluation of a patient by medical history, physical examination, and appropriate blood tests may not be sufficient to differentiate the two diseases. Furthermore, they may be present simultaneously. Cholescintigraphy with Tc-99m iminodiacetic acid derivatives has been reported to be useful in distinguishing acute cholecystitis from other causes of abdominal pain (*1–3*), but Edlund et al. recently questioned its ability to detect acute cholecystitis in the presence of acute pancreatitis (*4*). Analysis of cholescintigrams of seven patients with acute pancreatitis led them to conclude that the gallbladder frequently fails to visualize when pancreatitis is present, unless the patients are premedicated with cholecystogogues. Their report prompted us to review the results of cholescintigraphy in patients with acute pancreatitis who have been examined at our institution during a recent 17-month interval.

MATERIALS AND METHODS

Four hundred fifty patients were referred for cholescintigraphy to our Section of Nuclear Medicine between August 1, 1980 and December 31, 1981. Thirty-two patients who had acute abdominal pain, nausea, elevated serum amylase, and a provisional diagnosis of acute pancreatitis at the time of cholescintigraphy were identified by means of a preliminary survey. Their hospital charts were reviewed, and on this basis a diagnosis of acute pancreatitis was considered firmly established in 21 cases. All 21 patients had serum amylase levels at least twice the upper limit of normal in our laboratory (normal = 70–300 IU/l) on at least three consecutive days. Serum lipase levels were above normal (normal = 8–21 IU/l) in 13 of 13 patients so tested. The remaining eight patients included: five who were found at laparotomy to have signs of acute pancreatitis, such as pancreatic edema, induration, or areas of necrosis (*5*); one who had an elevated amylase-to-creatinine clearance ratio; and two who had severely elevated serum amylase levels (>6000 IU/l) that returned to normal within one week. We did not consider the results of cholescintigraphy in diagnosing acute pancreatitis. The presumed causes of pancreatitis in the 21 patients were as follows: cholelithiasis in 12; chronic alcoholism in seven; and treatment with thiazides two.

All patients fasted for at least 2 hr before cholescintigraphy. Following injection of 5 mCi of Tc-99m disofenin,* sequential images of the liver and abdomen were taken with a large-field-of-view scintillation camera fitted with a high-resolution collimator. Each scintiphoto contained one million counts. The study was terminated

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when the gallbladder, common bile duct, and small bowel were clearly seen on the images. Delayed images were obtained as long as 4 hr after injection of the tracer when the gallbladder was not seen during the first hour (*6*). Interpretations of the cholescintigrams used in this retrospective study were by an experienced observer at the time the examinations were performed.

RESULTS

The gallbladder was visualized normally (i.e. within 60 min after tracer injection) in 14 of the 21 patients with firmly established acute pancreatitis; it was visualized 90 min after injection in two cases, but was not seen as long as 4 hr after injection in the remaining five patients. These five were found to have gallstones on ultrasonic examination. Four underwent cholecystectomy, and all were found to have acute cholecystitis by surgical and histological criteria. The fifth patient was treated medically for acute cholecystitis and responded well to intravenous fluids and antibiotic therapy. Five of the 16 patients whose gallbladders visualized on cholescintigraphy (including the two with delayed visualization) also underwent cholecystectomy, and all five were found to have cholelithiasis and chronic cholecystitis.

DISCUSSION

Numerous reports have established cholescintigraphy as a useful procedure in the diagnosis of acute cholecystitis (*2,3*). Nonvisualization of the gallbladder within one hour after injection of Tc-99m-labeled iminodiacetic acid derivatives, in association with normal visualization of the biliary tree and bowel, has greater than 95% sensitivity and 85% specificity for acute cholecystitis (*3*). Weissmann and her associates have reported that the specificity of nonvisualization of the gallbladder as a sign of acute cholecystitis is approximately 99% if the finding persists in images made as long as 4 hr after administration of the imaging agent (*6*).

Nonetheless, Edlund et al. recently suggested that cholescintigraphy is unreliable in detecting acute cholecystitis if acute pancreatitis is present; they assert that in such cases normal gallbladders frequently fail to visualize (*4*). We have not been able to confirm their findings. The gallbladder failed to visualize in only five of our 21 patients with acute pancreatitis. Four of the five had histologically confirmed acute cholecystitis, and one was clinically presumed to have acute cholecystitis. Our results are similar to those of Frank, et al. (*7*) (Table 1), who reported nonvisualization of the gallbladder in seven of 22 patients with acute pancreatitis. Five of the seven had concomitant "cholecystitis," and radioactivity was not seen in the common duct or the bowel in the remaining two cases. Fonseca et al. (*1*) reported visualization of the gallbladder in 13 of 15 patients with acute pancreatitis, although in one case an image was achieved only after administration of cholecystokinin. The remaining two patients were thought to have chronic cholecystitis on the basis of sonographic findings. Edlund et al. cite one other series, that of Zeman et al. (*8*), in which the gallbladder failed to appear in four patients with acute pancreatitis. However, Zeman et al. did not specify the total number of patients with acute pancreatitis in their series, so their data cannot readily be brought to bear upon the question at hand. Thus, the gallbladders of patients with acute pancreatitis have been scintigraphically visualized in 46 (87%) of 53 published cases in which a) the common bile duct and bowel visualized normally, and b) acute cholecystitis has not been present. Contrary to Edlund's contention, the presence of pancreatitis per se does not seem to greatly affect the specificity of cholescintigraphy for acute cholecystitis.

One can only speculate on the reasons for the discrepancy between the experience of Edlund et al. and that reported by others. Differences in the radiopharmaceutical used or the amount of radioactivity administered conceivably may have been responsible. A more likely cause is that Edlund et al. did not obtain "delayed"

TABLE 1. CHOLESCINTIGRAPHY IN PATIENTS WITH ACUTE PANCREATITIS

Authors	NO. OF CASES	GALLBLADDER VIS†	Gallbladder not visualized		
			AC*	NO AC BOWEL NOT VIS	NO AC BOWEL VIS
Frank, et al. (*7*)	22	15	5	2	0
Fonseca, et al. (*1*)	15	13	0	0	2
Edlund, et al. (*6*)	7	2	0	0	5
Present series	21	16	5	0	0
Total	65	46	10	2	7

* Acute cholecystitis.
† Visualized.

images—those made later than 1 hr after administration of the tracer. Delayed views might have increased the rate of visualization of the gallbladder in their patients, as it did in our series. Chronic cholecystitis and cholelithiasis, which frequently result in delayed visualization of gallbladder (*9*), are very common accompaniments of acute pancreatitis. In Edlund's series a normal nonvisualized gallbladder was confirmed in only one of five patients, and it is possible that chronic cholecystitis was present in some or all of the remaining four. Furthermore, Edlund's series of seven was quite small, so their results cannot be expected to predict with accuracy the rate of nonvisualization of the gallbladder in other series of patients with acute pancreatitis.

We must conclude, on the basis of the data available to us, that cholescintigraphy is as useful for detecting acute cholecystitis in patients with acute pancreatitis as it is in patients without the latter disease. The gallbladder will visualize in a large majority of patients with acute pancreatitis if they do not have concomitant acute cholecystitis, particularly if delayed images are obtained (*7*). Additional specificity may be obtained in selected cases by the use of cholecystogogues (*1*), but usually this will not be necessary.

FOOTNOTE

* Hepatolite, New England Nuclear, Inc.

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