EDITORIAL

The Scintigraphic Evaluation of Sphincter of Oddi Dysfunction

In this issue of *The Journal of Nuclear Medicine*, Sostre et al. (*1*) describe an interesting scheme, combining visual and computer data, for the scintigraphic evaluation of sphincter of Oddi (SOD) dysfunction. This is an elusive condition primarily diagnosed in post-cholecystectomy patients and characterized by biliary type pain without structural abnormalities of the biliary channels.

It is relevant to understand the pathophysiology of SOD. A review article by Steinberg summarizes much of what is known about this entity (*2*). The SOD is a stubby muscular bundle regulating the flow of bile and pancreatic secretions into the duodenum through pressure changes caused by its grip on the confluence of the common bile and pancreatic ducts. Dysfunction of the sphincter manifests manometrically as increased basal pressure from spasm or stenosis, an increased frequency of contractions or uncoordinated ones, and a paradoxical response to cholecystokinin (CCK). These disorderly contractions and/or the poor emptying of the common bile duct may trigger ductal dilatation and abdominal pain since, postcholecystectomy, there is no gallbladder to serve as a reservoir to accommodate these pressure and volumetric changes.

Treatment of SOD consists essentially of sphincterotomy. Nevertheless, diagnosing the condition to institute therapy has proven difficult as many of the available tests are not sufficiently sensitive nor specific (*2*). These examinations have included noninvasive studies to identify sonographically a dilated common bile duct post-cholecystectomy, fatty meal sonography and also hepatobiliary scintigraphy with the ^{99m}Tc-IDA derivatives. More aggressively, provocative testing such as the morphine-prostigmine challenge can be carried out, while at endoscopic retrograde cholangiopancreatography(ERCP)delayed emptying of the common bile duct may be looked for. Otherwise SOD endoscopic manometry has been growing in acceptance although it may be an imperfect standard which may cause pancreatitis.

Qualitative hepatobiliary scintigraphy (HBS) originally showed promise as a simple screening test for SOD and for the assessment of response to therapy as elevated pressure at the sphincter retards the hepatobiliary transit of the radiotracer (*3,4*). Retention of a ^{99m}Tc-IDA derivative at 2 hr in visually prominent ducts was noted to be the best predictor of abnormal biliary drainage (*3*). Further refinements in HBS for sphincter dysfunction have included computer-assisted techniques, rather than structures such as the common bile ducts, to evaluate time to peak activity and percentage washout of radioactivity at a fixed time interval, both of which are delayed in SOD (*5–7*). While results have been mixed (*6–8*), nuclear scintigraphy is overall helpful in investigating the condition though it falls short of being the definitive screening test since overlaps may exist between normal populations, patients with hepatocellular disease or cholestasis and those with SOD.

The current clinical trial of Sostre et al. (*1*) differs from other attempts to screen for SOD with HBS in the routine use of a cholecystogogue, the timing of its administration and the duration of its infusion. Under normal circumstances, CCK will cause contraction of the gallbladder while simultaneously relaxing the sphincter of Oddi and increasing bile flow. The authors have applied a physiological test, with premedication with CCK, to a functional disturbance to accentuate differences in bile flow rates between the normal post-cholecystectomy population and the one with SOD. It is open to discussion however as to how much the administered CCK accounted for their excellent results considering the short 3-min length of the infusion and the relatively limited period of action of the agent which has a half-life of 2.5 min in the blood stream (*9*). Conceivably, as the authors suggest, a longer CCK infusion period might be more beneficial in lowering sphincter pressures and promoting biliary flow in more sustainable fashion in normals. The role and mode of administration of CCK in SOD still merits more thorough investigation therefore as the authors' concept in attempting to reduce false positives is pleasing. The possibility of CCK eliciting a paradoxical response at the sphincter and a greater delay in biliary transit in SOD, which could further facilitate the screening, needs more clarification as well. In fact, this paradoxical effect cannot always be demonstrated (*6*).

The authors also introduce a scintigraphic score when evaluating their data. By using qualitative and quantitative interpretive criteria they appear to have developed a useful scoring scheme which is subject to little interobserver variation and which targets accurately patients with SOD. Nevertheless, although this is a promising effort, their premise that this scoring system may also differentiate consistently between cholestasis and SOD still needs to be tested on a greater number and variety of patients (*10*). It should be noted that 46% of the patients they studied had SOD, which is a figure totally out of proportion to the much lower prevalence of <1% expected in the usual post-cholecystectomy population (*2*). This high occurrence of SOD in their small sample of patients could account substantially then for their perfect sepa-

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ration of SOD patients as a natural outcome of Bayes' theorem. Still, their scoring scheme is an initial and elegant step which unifies all the data gathered from HBS and which should help screen for a disabling condition which while not very common is yet amenable to a curative procedure. It would also be of interest to explore whether a clinical index based on the reproduction of pain or abdominal discomfort, after CCK injection, might be integrated with the scintigraphic score proposed by Sostre et al. in further separating patients with SOD from others.

The combined qualitative and quantitative approach by Sostre et al. to this clinical entity also suggests that their analysis may be used serially and may assist in distinguishing SOD patients from those with strictures and common bile duct stones who might have identical findings on HBS. Such a diagnostic application of HBS with a reproducible and accurate scoring system first with a baseline study to be followed by a repeat examination with smooth muscle relaxants, if initial results are positive, should enhance the value of scintigraphy as a screening test for SOD if abnormal findings reverse with relaxants. Of parallel interest as well, are those patients with end-stage chronic cholecystitis and functional cholecystectomy who might benefit from evaluation for SOD using the approach of Sostre et al. A study of these groups of patients would help further establish the validity of their scintigraphic score.

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