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PITFALLS IN GASTROINTESTINAL DIAGNOSIS

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INTRODUCTION

The discussion of pitfalls or errors in diagnosis is a more effective method of improving diagnostic accuracy than focusing attention upon the achievements of various diagnostic procedures. Diagnostic errors should be and usually are invaluable although painful stepping stones to real and lasting progress.

Frequently, misdirected treatment, both medical and surgical, in the field of gastroenterology is due to the incorrect interpretation of gastrointestinal symptoms and attributing them to the wrong underlying condition. This occurs especially where several underlying abnormalities exist, as so often occurs in the gastrointestinal tract.

Confused thinking usually can be avoided if one makes the classical threefold anatomical, functional and etiological diagnosis, outlining clearly in one's mind the evidence for each. The functional diagnosis should attribute the several symptoms to the correct underlying cause or causes. If the ultimate etiology of each of the several conditions is not known, it is essential at least to clarify in one's mind the important contributing or aggravating factors. The therapeutic emphasis will then be directed toward the correct underlying condition or conditions and from an etiological approach. To evaluate the functional diagnosis correctly, it is necessary to know the various differential points in the symptomatology of the several underlying conditions.

It seems worth while to summarize briefly, without going into unnecessary details, those errors in gastrointestinal diagnosis which are made from time to time and may lead to unfortunate and sometimes disastrous results. These errors are not made as often as they were made twenty years ago, but they still occur more often than they should. They may be summarized concisely in outline form as follows:

I. *Confusion Between Gallbladder and Chronic Irritable Colon Pain and Tenderness*

Failure to interpret correctly right upper quadrant pain and tenderness may lead to cholecystectomy for relief of colon pain. This error is due to failure to

appreciate (1) the several characteristics of irritable colon distress, (2) the importance of examining such patients in the sitting position in order to identify true gallbladder or liver tenderness and differentiate it from irritable colon tenderness and (3) the fact that chronic irritable colon is a much more common cause of right upper quadrant pain and tenderness than chronic inflammation of the wall of the gallbladder or gallstone colic.

II. *Errors in Intestinal Diagnosis*

1. The assumption is often made that the association of amoebiasis with colon distress means that the colon distress is due to amoebiasis, whereas 65% of the cases of amoeba histolytica infestation are latent and do not cause symptoms. Patients whose stools no longer reveal amoebae frequently receive unnecessary repeated courses of amoebicidal drugs for relief of persistent colon distress. Such cases usually are not actually relieved of their distress until they receive irritable colon therapy.

2. Many cases of latent amoebiasis are not found because warm stools are not examined routinely.

3. Numerous cases of early, atypical, nontropical sprue are misdiagnosed as chronic irritable colon. The fairly frequent occurrence of sprue is not always appreciated and often sprue is not even considered in differential diagnosis when it should be. In such cases, therefore, there is a failure to conduct the oral glucose tolerance test, other absorption studies, stool studies for unabsorbed fat and roentgen-ray studies of the small intestine.

4. Rectal polyps, which are frequent precursors of rectal carcinoma, are often overlooked and not removed because proctoscopic examinations are not done frequently enough.

5. Rectal and colonic cancer are still missed because rectal bleeding is not always considered as an indication for a proctoscopic examination and barium enema.

6. Early carcinoma of the distal colon, with recent constipation as the only symptom, still may be treated temporarily only for constipation, because a barium enema is not obtained always before treating constipation as a symptom.

7. Diagnosis of regional ileitis is often missed because x-ray studies of the small intestine often are not conducted when they should be.

8. Fortunately, the fallacious clinical diagnosis of chronic appendicitis, as a cause of symptoms, has waned.

III. *Misinterpretation of Liver Function Tests*

1. The degree of functional hepatic impairment may be interpreted incorrectly as indicating a corresponding degree of organic impairment.

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(a) In acute hepatitis impairment of liver function and structure do tend to run rather parallel.

(b) In chronic liver disease there is often considerable dissociation of impaired function and structure. Some cases with moderately well-developed cirrhosis may exhibit only slight impairment of function. Conversely, certain cases with only slight organic impairment, as indicated by liver biopsy, may show rather marked functional impairment.

(c) Therefore, if comprehensive as well as accurate information about the liver is desired, both liver function tests and liver biopsy studies are indicated.

2. A second common error in interpreting liver function tests occurs in jaundiced patients and consists of drawing the same deductions from the results of function tests in patients with early and late jaundice. In late jaundice the usual interpretation of these tests is unreliable for obvious reasons.

IV. Causes of Errors in Differentiating "Medical" and "Surgical" Jaundice

1. Failure to observe and study jaundiced patients for at least 7 to 10 days, or longer, if there is any disagreement between diagnostic data, before attempting to make a final diagnosis.

2. Failure to appreciate that complete suppression of bile flow in acute hepatitis *occasionally* persists for 10 to 14 days, instead of only 2 to 3 days. This may mislead one into an incorrect diagnosis of obstructive jaundice.

3. Failure to appreciate that a painstaking history and physical examination lead to a correct diagnosis in more than half of the cases.

4. Failure to emphasize sufficiently the *direct type* of laboratory evidence for bile duct obstruction, namely,

(a) repeated chemical examination of the stools for bile and blood,

(b) quantitative urinary urobilinogen test and (c) the blood prothrombin determination before and after parenteral vitamin K administration.

5. Failure to interpret the indirect laboratory evidence from other liver function tests in relation to the duration of the jaundice.

6. Failure to search for primary carcinoma elsewhere in the body as a source for possible metastatic carcinoma in the liver.

7. Problem of differentiating the two types of obstructive jaundice (intrahepatic and extrahepatic).

(a) Chronic cholangiolytic hepatitis tends toward more or less normal liver function with associated complete and persistent intrahepatic obstruction to the bile

flow. It may be extremely difficult or impossible to differentiate it from extrahepatic obstruction of the common bile duct. Fortunately, no harm is done by exploring patients with the cholangiolytic type of hepatitis, since liver function as a rule is essentially normal.

V. *Erroneous Diagnosis of Duodenal Ulcer*

1. Functional pylorospasm may be diagnosed as a duodenal ulcer.
2. This error can be avoided by a painstaking history and a critical interpretation of x-ray findings.
3. The eight characteristics of uncomplicated peptic ulcer distress, which if present differentiate it from pylorospasm and other types of epigastric distress, should be inquired for in every patient with epigastric distress.
4. A typical ulcer history indicates the presence of a peptic ulcer. An atypical history throws grave doubt on the diagnosis of ulcer, although a small per cent of patients with ulcer complications yield a somewhat atypical history.
5. A probable clinical diagnosis of active ulcer or no ulcer should be made in every suspected case, since the x-ray examination often does not portray an ulcer crater as direct evidence of an active ulcer even when an ulcer exists. The more frequently found, generalized x-ray deformity of the duodenal bulb indicates either a present or previous ulcer.

VI. *Fatal Error of Diagnosis of a Malignant Gastric Ulcer as Benign, with Prolonged Medical Treatment*

1. Initial history and x-ray studies of malignant ulcer may simulate benign ulcer.
2. The three classical points of differential diagnosis, namely, the prompt disappearance under therapy of the distress, the x-ray defect and the occult blood from stools in benign gastric ulcer have value, but also distinct limitations.
3. Repeated follow-up x-ray and gastroscopic examinations during treatment over a considerable period are absolutely essential in medical treatment of gastric ulcer.
4. Even the complete growth of epithelium over a healing gastric ulcer, gastroscopically noted, does not rule out a malignant type of ulcer, with subsequent recurrence of ulceration in this location.
5. The only way to avoid the above serious diagnostic error is to advise surgery promptly, if there is *any suggestion* of malignancy either in the original or follow-up studies and observation.

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VII. *Problem of Determining the Cause of Hematemesis*

1. Oesophageal varices of hepatic cirrhosis, as the probable cause of hematemesis, are suggested if one can demonstrate either (a) clinical evidence of cirrhosis, (b) appreciable retention of bromsulfalein dye, or (c) a reduction of the serum albumen. Bleeding from varices is usually profuse and at times fatal.

2. Bleeding from a *chronic* peptic ulcer is suggested if a history of previous ulcer-like distress or of previous x-ray evidence of an ulcer is obtained.

3. Bleeding from an *acute* peptic ulcer is suggested if none of the findings noted above under (1) or (2) are present.

4. Hypertrophic gastritis occasionally causes hematemesis, as has been demonstrated gastroscopically. (Unless this latter examination is conducted, bleeding from hypertrophic gastritis cannot be differentiated from that of a single, acute peptic ulcer.)

5. The more rare causes of hematemesis will not be discussed here.

VIII. *Confusing Perforated Duodenal Ulcer with Acute Pancreatitis*

1. Both conditions present severe epigastric pain and protective muscle spasm, with fever and leukocytosis.

2. History of previous ulcer-like distress and previous x-ray evidence of ulcer, if present, and the demonstration by x-ray of free air in the peritoneal cavity make the diagnosis of perforated ulcer in such cases.

3. Presence of elevated blood diastase and absence of any of above evidence of ulcer point to acute pancreatitis in cases of this type.

4. Prompt operation is essential for perforated ulcer, whereas our statistics show that most cases of acute pancreatitis progress better without surgery.

5. If occasionally early differential diagnosis is impossible, prompt laparotomy is indicated for obvious reasons.

IX. *Causes of Failure to Make Relatively Early Diagnosis of Primary Carcinoma of Stomach*

1. Failure of symptoms to appear early.

2. Failure of patient to consult physician when symptoms first appear.

3. Failure of physician in some instances to obtain repeated stool examinations for occult blood and *repeated* follow-up roentgen-ray examinations of stomach in all patients with recent onset of epigastric distress.

4. Failure to conduct a gastroscopic examination when other studies yield negative results in such cases.

The above array of pitfalls and possible errors in diagnosis is not intended to detract in any way from the remarkable progress made by the medical profession in recent years in advancing the accuracy of medical diagnosis, nor to imply that the above errors are made frequently. Rather, it is hoped that the above comments simply may call attention to those areas of gastrointestinal diagnosis where one should stop, look and listen.