Introduction to ABDOMINAL SURGERY

Fifty Clinical Studies

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PREFACE

These case studies are for beginners in surgery. They are the substance of certain teaching conferences at the Montefiore Hospital Medical Center and the Albert Einstein College of Medicine, directed to first-year house staff, third-year medical students, physicians' associates, and certain nursing personnel.

These cases deal with the common disorders and their usual presentations. They are not intended to parallel the esoteric CPC conferences given by the specialists in footnote ibids.

Their purpose is to transmit information and, by example, to indicate how the collection of data by history and by a symptom-directed physical examination is rationalized into a provisional differential diagnosis. That deduction is then discriminated by pertinent laboratory data.

This approach does not reward the inspired snap diagnosis. It favors logic based on a reasonable physiologic, pathologic interpretation of the data. In the art of surgical diagnosis the ugly duckling rarely gets haphazardly embraced by the prince of chance to become what it is not.

I believe the greatest value of the introductory teaching conferences is in the "listen, feel, and rationalize," evaluation of the patient, to which the advanced expensive diagnostic machinery is secondary.

These three faculties have to be retained, indeed have to be cultivated, if we are to remain the masters and not the vassals of detached computers. Like the bikini, the confirmatory tests are as important for what

they conceal as for what they reveal. A computer may not tell you that the patient has fallen out of bed.

If there is some repetition in these case studies, that is part of the art of teaching. If there is dogmatism, that is the legacy of experience. If there are differences of opinion, that is the art of medicine to which surgery is the handmaiden.

TO THE STUDENT-READER

I hope you will view these cases as an adventure in surgical diagnosis. I have tried to preserve the informal conversational atmosphere in which these conferences took place.

Try to make a provisional diagnosis from the chief complaint and history. Listen to patients; they will often tell you the diagnosis. The first page of each of these cases is a sort of quiz, "Can you diagnose the disorder?" from the mini data. This is to emphasize a direct, on-target, no-nonsense, no-fuss approach to the problem of abdominal diagnosis in an age that strains to bypass the brain. Remember that most things occur most commonly. It is more frequent to see an unusual presentation of a common disorder than to see something truly rare.

Direct and particularize the physical examination so as to confirm or to refute your initial impressions. Request such pertinent laboratory data (x-ray and laboratory services) as are most likely to be of value, with the attitude that a shotgun approach diminishes acumen and the critical faculty. One well-thought-out procedural determination is likely to be of greater value than doing tests pro forma so that you don't miss anything. We in medicine are also obligated to participate in cost containment.

The illustrative roentgenographic material has been specifically chosen to be typical of the entity under discussion. In a few instances it pertains to the entity, but not to the specific individual under consideration. This reinforces my belief that recognition of the uncommon is facilitated if one knows the details of the typical.

The student participants at these conferences have chosen the suggested readings, without reference to their date of publication, as meeting their needs.

The table of contents in the front matter of the book lists the presenting symptoms. The impatient reader can turn to the rear of the book for an index of these case histories arranged according to final diagnosis.

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ACKNOWLEDGMENTS

The opinions and attitudes expressed here, many admittedly controversial, are my own. No other direct or inadvertent participants in these conferences are to be included under the umbrella upon which criticism may fall.

I want to acknowledge my debt to those who have contributed to the weekly functioning of these conferences. Dr. Marvin L. Gliedman initiated these sit-down luncheon conferences and often determined their direction, from the corner seat of a long, sandwich-laden, U-shaped table.

The radiologic material derives from several sources. Doctors Thomas C. Beneventano and Bertha Rubenstein provided the gastrointestinal studies; Dr. Seymour Sprayregen, the angiographic material; Dr. Ruth Rosenblatt, the echographic interpretations; Dr. Marc Goldman presented the CAT scans.

Stanley Waine, who did the drawings for three of my previous monographs, continues to provide first-rate illustrations that clarify and epitomize.

The aid of the Palestine family is appreciatively acknowledged.

None of this would have been possible without the 15 years of presentors of these cases. For many it was their first formal case presentation. I hope they remember us in the image of Mr. Chips. Certainly these sessions have been the most rewarding and stimulating part of my own academic teaching experience.

The cooperation, far beyond the perfunctory, of the publisher and

the editorial staff of the Medical Department of Harper & Row is again appreciated.

Once again, the arduous task of assembling all the material into a viable manuscript fell upon Mrs. Carol Grossman, still, and again, no vocalization, but great appreciation.

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1 ESOPHAGUS

Case 1. Vomiting, Dyspnea, and Collapse

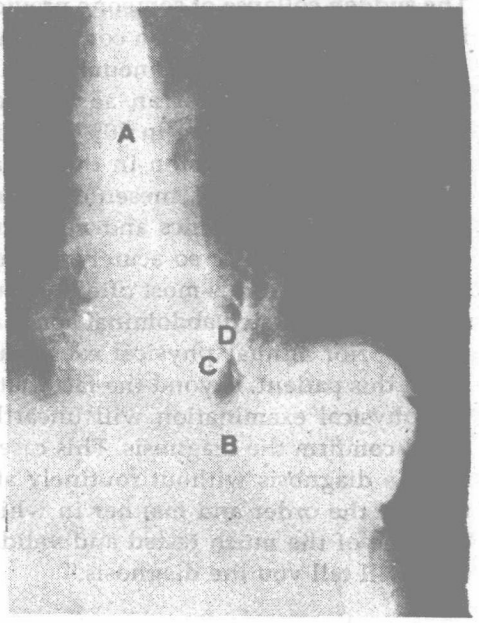

FIG. 1-1

HISTORY

A 54-year-old man is admitted to the emergency room in a state of collapse. Six hours previously, he had attended a banquet, where he had gorged himself on duck and whiskey. One hour later, he started to retch and vomit repeatedly and complained of pain in the epigastrium and left back. A physician friend attributed this to a bad gastric reaction—gastritis. The patient was escorted home. Three hours later the pain became worse, and he became increasingly short of breath. He was brought to the emergency room with a diagnosis of myocardial infarction.

This is a somber business man with no prior digestive complaints; he had done no heavy lifting that day. At a routine annual checkup 1 week before he had been declared "clean" (Fig. 1-1).

PROVISIONAL DIAGNOSIS: _____

Discussion

The sudden collapse of someone previously healthy suggests a catastrophic physiologic insult. When considering the chest, one thinks of a pulmonary embolism, massive pneumothorax, or myocardial infarct. The abdomen can be the site of an aortic aneurysm complication, a perforated peptic ulcer (presenting in 20% of patients with no history), fulminating pancreatitis (less common in the nonalcoholic than in the alcoholic, although it does occur), mesenteric thrombosis (unusual in patient with normal cardiac dynamics and no arteriosclerotic problem). Strangulated bowel rarely evolves so acutely. A ruptured dissecting aneurysm might do this, although it is most often encountered in hypertensive persons. A large uncomplicated abdominal aortic aneurysm might have been evident at the prior annual physical examination. The important clinical point about this patient, beyond the fact that he has a catastrophic illness is that the physical examination will unearth a specific clue, one finding that could comfirm the diagnosis. This case illustrates the fact that one cannot make a diagnosis without routinely attempting to dissect out the symptoms in the order and manner in which they presented. This is another example of the much tested and validated aphorism, "Listen to patients; they will tell you the diagnosis."

Symptom-Directed Physical Examination

The physical examination requires a systematic search for clues to each of the possibilities. One specifically wants to know the physical findings upon auscultation and percussion of both lungs and heart, especially the cardiac rhythm, rate, and quality of the heart sounds. Are the lungs clear? On abdominal examination, one should define the degree and site of the peritoneal reaction and look for blood in the gastric aspirate and on rectal examination. Meanwhile, a nasogastric tube should be put down; a catheter should be inserted into the bladder and a central venous line established for monitoring the need for fluids or cardiac support measures as well as for the administration of colloids.

Fig. 1-1. The Gastrografin-filled esophagus (A) is opacifed in the thorax. The contrast material enters the stomach (B). However, just above the diaphragm, the contrast material has leaked outside of the esophageal lumen and appears as puddled areas at sites C and D. This indicates a perforation of the esophagus into the mediastinum.

PHYSICAL EXAMINATION

This patient is in severe pain and collapse. Blood pressure is 90/60, pulse 130, respiration 40 and extremely shallow. The temperature is subnormal at 97° F. The skin is cold and clammy. There is upper abdominal rigidity with surprisingly little tenderness. The cardiac rate is normal, the heart sounds are faint, and there are no murmurs. Examination of the chest reveals diminished breath sounds and dullness at the left base. No rales. There is no blood on the rectal examining finger and there is no blood in the gastric aspirate.

Discussion

The clinical syndrome here is that of a previously healthy man who presents with sudden collapse, subnormal temperature, upper abdominal rigidity, and auscultatory findings in the left chest. This is obviously more than the suggested gastritis.

Differential Diagnosis

The differential diagnosis has to include severe acute alcoholic pancreatitis. This would fit with the recent Lucullan gluttony and alcohol consumption and with the abdominal findings, collapse, and assumed left pleural reaction. Acute pancreatitis can be this severe and even terminate fatally with the first episode.

Rupture of the gallbladder or of the appendix without any prodome occurs but is extremely unusual. There is no arteriosclerotic problem here to indicate that this man has a ruptured aortic aneurysm or a mesenteric vascular occulsion. The presumptive diagnosis therefore is acute fulminating catastrophic peritonitis due to an undefended free perforation.

Pertinent Diagnostic Methods

In addition to the usual blood studies, this patient requires a chest film, an electrocardiogram (ECG), and a plain film of the abdomen. One would think that this would lead to the diagnosis. If the electrocardiographic and enzymatic changes do not indicate a pulmonary embolus or a myocardial infarct, it is probable that he is going to require prompt abdominal surgery. It should be remembered that we are now into this man's illness for about 2 hours and that he is being sent directly from the emergency room to the x-ray department.

INVESTIGATIVE DATA

SMA 6 and 12 and CBC are normal. The flat film of the abdomen reveals no free air or ileus pattern. The radiologist calls back an emergency reading on the chest film. He reports hydropneumothorax on the left and mediastinal emphysema. He suggests that the patient be reevaluated with this in mind.

Reexamination reveals subcutaneous crepitus in the supracavicular fossa and in the supersternal notch. Furthermore, auscultation of the precordium reveals a crunching sound with the heart beat, Hamman's sign. The diagnosis now seems obvious, especially when the sodium diatroziode that the radiologist instills into the nasogastric tube leaks out of the esophagus into the left pleural space (See Fig. 1–1).

Integration and Final Differential Diagnosis

It is now apparent that this patient has a perforation in the intrathoracic portion of the lower end of the esophagus. This is not an intra-abdominal disorder despite the rectus muscle rigidity. Without the chest film, this man's abdomen might have been opened without disclosing the pathologic process. This disease is 100% fatal if untreated; if treated late it still is associated with a mortality of about 30%. We can reconstruct this as being a typical Boerhaave's syndrome. According to the oft repeated story about that clinician of Leiden, he reported in 1724 in Latin, the case of an admiral in the Dutch navy who gorged himself on roast duck, vomited, and died. At the autopsy Boerhaave predicted a full-thickness disruption of the lower third of the esophagus, hence the eponym.

In his time, Boerhaave (1668–1739) was the most famous clinician in Europe. It was said that if a letter were addressed to him from China with only "to Boerhaave in Europe," it would come to the correct place. He was a prominent physician who is said to have accumulated more than 10 million florins from private practice. His other legacy to our time was his emphasis on direct bedside teaching in preference to impersonal amphitheater lectures. He stressed the physical examination and collected his clinical observations, regarding diagnosis and treatment, in a group of aphorisms.

This patient vomited repeatedly, and, for an obscure reason that pertains to the syndrome generally, there must have been an impediment to the outflow of the vomitus, so that a disruptive high-pressure zone was built up in the lower esophagus. Ordinarily, as is known from experiments done on cadavers, the pressure required to rupture the esophagus is four times greater than that required to rupture the stomach. It is hard

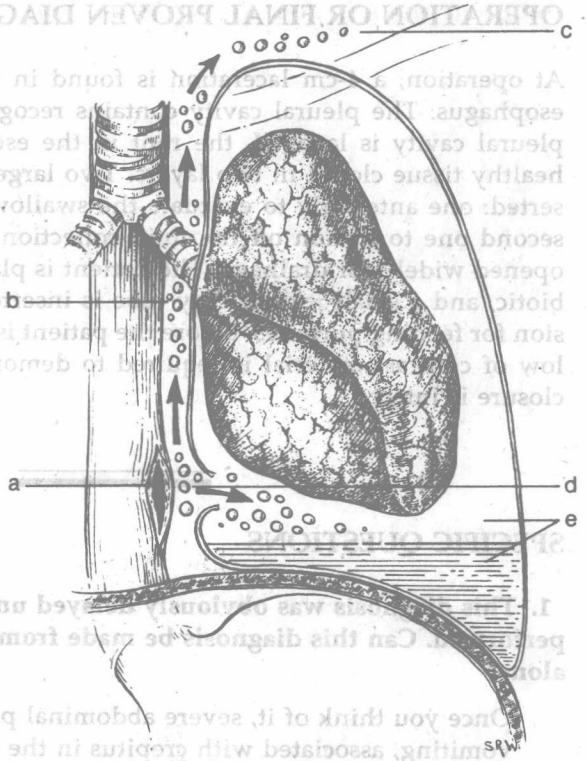

Fig. 1-2. Sequence of events in the Boerhaave syndrome. There is a longitudinal tear in the lower third of the esophagus, **a**; initially the mediastinal pleura is intact (**vertical arrows**), and the swallowed air as well as some of the fluid in the esophagus escapes into the posterior mediastinum (**b**) and presents as supraclavicular crepitus, **c**. When the pressure increases, the mediastinal pleura ruptures, **d** (**horizontal arrow**), and a pneumohydrothorax ensues, **e**. The latter soon becomes infected, becoming an empyema originating in an esophagopleural fistula.

to understand why, in these instances, the esophagus ruptures rather than the stomach. Perhaps there is some more proximal obstructive factor. The pressure alone does not determine whether the organ will rupture. The rapidity with which the pressure builds up also plays a role. Why do some esophagi rupture and others not? There are circular muscle deficiencies in some esophagi. The vast majority of these organs, however, are not grossly diseased to start with. In other words, this is a rupture of a normal esophagus. It is not an acid–pepsin-linked phenomenon (Fig. 1-2).

THERAPEUTIC ALTERNATIVES

The therapeutic implication of this diagnosis is that it requires prompt surgery. Initially, a thoracentesis may relieve the intrapleural hydropneumothorax that is responsible for the dyspnea. A temporizing closed thoracotomy may be advisable, but the important thing is that a transthoracic operation be performed immediately.