INTRA-ABDOMINAL APOPLEXY

J. EDWARD BERK, M.D., M.Sc. (MED.)., N. S. ROTHSCHILD, M.D.,
AND
J. C. DOANE, M.D., F.A.C.P.
PHILADELPHIA, PA.
FROM THE MEDICAL AND SURGICAL SERVICES OF THE JEWISH HOSPITAL, PHILADELPHIA, PA.

SPONTANEOUS HEMOPERITONEUM is not too unusual an occurrence. Even excluding spontaneous hemoperitoneum, of genital origin in the female, Morridor and Olivier[1] were able to collect 432 cases from the literature. On the other hand, massive spontaneous intraperitoneal hemorrhage resultant from the rupture of a small intra-abdominal blood vessel independent of any direct trauma to the abdomen, is a rare accident. The condition has very aptly been designated "intra-abdominal apoplexy," to denote its close resemblance both in spontaneity and nature to the much more frequent and better known cerebral apoplexy. An exhaustive study of the literature has revealed but 19 cases which might be so classified.* To these 19, we have added a case of our own and briefly tabulated (Table I) what seemed to us the essentials in each. It is only through the repeated reportings and tabulations of such cases, that characteristics, if any, will become recognized which may serve in the diagnosis and treatment of future cases.

Case Report.—The patient, male, age 52, presented himself at the hospital at 10:00 P.M., May 2, 1939, complaining of dull, persistent pain in the abdomen since April 29, 1939, which had become steadily worse. The pain, which began insidiously, was constant, dull, and diffuse over the midabdomen without any radiation. Vomiting had occurred but once, and that was on the day of admission. The vomitus was of a nondescript type. The bowels had been regular and the appetite undisturbed. There were no urinary symptoms.

The patient had known for several years that he had hypertension. For the past two months he had been receiving treatment for attacks of precordial pain and dyspnea which would occur while walking home from work. He had had a bilateral herniorrhaphy and appendicectomy in 1935. For the past few years he had noted failing vision. There was no history of any digestive disturbances. The remaining past history and the family and social history were irrelevant.

Physical Examination.—As seen on admission, the patient was resting flat in bed apparently fairly comfortable, despite his description of pain of a rather severe character. The state of nutrition was fair. There was no cyanosis, jaundice or dyspnea. He appeared alert, oriented and cooperative. Temperature 98° F.; pulse 90; respirations 21; blood pressure 240/170.

The heart was markedly enlarged. There was a presystolic gallop rhythm present with a systolic murmur at the apex and base and a split pulmonic second sound. The aortic second sound had a tambour quality. The abdomen was flat and the wall was soft and relaxed. Tenderness was obtained in the epigastric area on deep pressure. The

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* Since this paper was submitted an additional case has been reported (Crile, G., Jr., and Newell, E. T., Jr.: Abdominal Apoplexy, J.A.M.A., 114:1155, 1940).

peripheral blood vessels were firm and thickened. Ophthalmoscopic examination revealed a blurring of the disk margins and hypertensive neuroretinopathy, Grade III. Because of the marked hypertension, the apparent cardiac and arterial changes and the essentially negative abdominal findings, a tentative diagnosis of "abdominal angina" was made and the patient was kept at rest in bed under observation.

Laboratory Data.—W.B.C. count on the morning following admission was 16,000, 86 per cent neutrophiles. Repeated counts yielded figures ranging between 10,000 and 14,200 W.B.C.

An electrocardiogram revealed myocardial disease with an intraventricular conduction defect. A flat roentgenogram of the abdomen was negative and a gastro-intestinal roentgenologic examination revealed nothing more than spasticity in the pyloroduodenal region. Repeated uranalyses showed a low specific gravity with a profusion of casts and W.B.C. Repeated kidney function tests, likewise, showed impaired renal function. The blood Wassermann was negative. All other studies yielded results within normal limits.

Course in Hospital.—The presenting complaint of diffuse midabdominal pain of a dull nonradiating type persisted after admission until May 8, 1939. On the evening of May 8, 1939, the temperature began to mount to reach a high of 103° F. at noon of May 9, 1939, then declined to return and remain normal about noon of May 10, 1939. The patient was completely asymptomatic and examination revealed nothing to account for this temperature elevation other than slight distention of the abdomen. A repeat roentgenogram of the chest and electrocardiogram were both negative. Abdominal distention remained, despite regular bowel evacuations and a lack of any complaints on the part of the patient.

Shortly before 3:00 A.M., May 16, 1939, the patient complained of sudden severe, nonlocalized abdominal pain and requested a bedpan. While on the bedpan he vomited about ten ounces of a dark material that seemed blood-tinged, following which he went into a partial collapse. Examination revealed a temperature of 98° F. (rectal), pulse 98; respirations 28; blood pressure 90/70. The skin was cold and clammy. The abdomen was moderately distended without tenderness or rigidity. There was no dulness in the flanks and peristalsis seemed somewhat hyperactive. Shock treatment was instituted and about one-half hour later the blood pressure had risen to 110/80. The abdomen continued to become more distended and peristalsis became entirely inaudible. Moderate generalized tenderness appeared over the abdomen and the walls were felt to become more tense. A flat film of the abdomen, taken at this time, was negative. It was our impression that the patient had suffered a mesenteric thrombosis and an operation was decided upon as soon as he had responded to the shock treatment that had been instituted.

Operation.—May 16, 1939 (N. S. R.): Under ether anesthesia, an upper right rectus incision was made into the peritoneal cavity. A considerable amount of blood exuded. It was dark in color and apparently free of any stomach or intestinal contents. The blood was gradually, and slowly, removed. In excess of 1,000 cc. of blood was aspirated and considerably more was lost from the wound primarily. The abdominal wound was enlarged to obtain a better view.

Operative Pathology.—The mesentery of the small bowel showed no point of bleeding. Examination of the omentum showed no evidence of bleeding. The stomach (only the pyloric and fundal portions could be examined) showed no evidence of a bleeding point. The duodenum appeared normal. There was no evidence of bleeding from the liver, although there seemed to be just as much blood immediately below the liver as there was in the pelvis. The spleen appeared intact and gave no evidence of bleeding in or about it. With practically all of the blood in the peritoneal cavity removed, and inspection revealing no bleeding point or site of continued oozing, the abdomen was closed with through and through silkworm sutures, without drainage. The skin was closed with silkworm gut. During this extensive exploration, the patient went into

deeper shock, despite the fact that intravenous administration of glucose and saline, acacia and blood were administrated during the operative procedure.

Postoperative Course.—Postoperative recovery proceeded uneventfully with the exception of abdominal distention, for which more or less continual duodenal suction syphonage (Wangensteen) was maintained. It was found necessary to keep the Wangensteen apparatus attached and tube suction continued for about 15 days, during which time there was, of necessity, a deficiency in protein and vitamin intake. On June 6, 1939, moderate anasarca appeared with bilateral hydrothoraces and abdominal ascites. The R.B.C. count just prior to this time was 3.1 million, with 55 per cent hemoglobin. Although the total plasma protein was at the lower limits of normal, 6.45 grams per cent, it was felt that some of this represented hemoconcentration from dehydration and that the status of the patient represented primarily an advanced degree of postoperative nutritional and vitamin deficiency. Following paracentesis of sanguineous-tinged effusions from both pleural sacs and from the abdomen, a high protein and vitamin diet was instituted, forti-

TABLE I

ESSENTIAL DATA IN 20 CASES OF INTRA-ABDOMINAL APOPLEXY

Case No.	Author	Year	Sex	Age	Anatomic Location	Cardiovascular System	Operation	Result	Autopsy
1	Barber[2]	1909	F.	32	No definite bleeding point	Apparently normal	Yes	Rec.	—
2	Churchman[3]	1911	M.	48	No definite bleeding point	Apparently normal	Yes	Died	No
3	Florence & Ducuing[4]	1913	F.	—	Sup. mes. art.	—	No	Died	Yes
4	Hilliard[5]	1918	M.	48	N.D.B.P.—(transmesocolon)	Hypertension 160–190 (systolic)	Yes	Died	No
5	Starcke[6]	1923	M.	60	Gast.-duo. art.	Hypertension 155	Yes	Rec.	—
6	Budde[7]	1925	M.	27	L. gast.-epip. art. (aneurysm)	Normal	Yes	Rec.	—
7	Green & Powers[8]	1931	F.	54	L. gast. art.	Hypertension 270/145	Yes	Rec.	—
8	Morgue-Molines & Cabanac[9]	1933	M.	56	L. gast. art.	(Questionable)	Yes	Rec.	—
9	"	1933	—	—	L. gast. art.	Art.-sclerosis & hypertension 230/130	Yes	Rec.	—
10	" (Case of Rud's)	1933	F.	73	L. gast.-epip. art.	Marked art.-sclerosis at P.M.	Yes	Died	Yes
11	Hartley & McKechnie[10]	1934	M.	31	No definite bleeding point	Apparently normal	Yes	Died	Yes
12	Thompson & Dunphy[11]	1935	F.	62	L. gast. art.	Hypertension 170/100	Yes	Rec.	—
13	Buchbinder & Greene[12]	1935	M.	57	R. gast. art.	Hypertension 190/115	Yes	Rec.	—
14	Moorehead & McLester[13]	1936	M.	44	R. & L. gast. art.	Hypertension 220/140	No	Died	Yes
15	"	1936	M.	50	Sup. mesen. art.	Myocardosis with arteriosclerosis	No	Died	Yes
16	Bruce[14]	1937	M.	34	No definite bleeding point	Apparently normal	Yes	Rec.	—
17	"	1937	M.	75	Mid. colic art. (aneurysm)?	Abdominal vessels normal	—	—	—
18	Morton[15]	1938	M.	72	Sup. mes. art.	(Questionable) B.P. 100/70—155/96	Yes	Rec.	—
19	Silverstone[16]	1938	M.	52	No definite bleeding point	Hypertension 200	Yes	Died	—
20	Berk, Rothschild & Doane	1939	M.	52	No definite bleeding point	Hypertension 250/160. Myocardosis & art.-scler.	Yes	Rec.	—

fied by several repeated blood transfusions and daily administrations of vitamin concentrates. Under this regimen the patient made a slow but steady improvement. He was finally permitted out of bed on July 5, 1939, and was discharged in fairly good condition on July 12, 1939.

Follow-up.—The patient was last seen again on October 1, 1939. He had had no digestive disturbances and was free of all abdominal complaints. Since his discharge he had about four attacks of paroxysmal nocturnal dyspnea. He was ambulatory and his color and nutrition were good. His temperature was 97° F., pulse 76, respirations 20, blood pressure 250/160. As noted on earlier examinations, the heart was enlarged. There was a presystolic gallop and a systolic murmur at the apex and base. The aortic second sound had a tambour quality and the pulmonic second sound was split. Ophthalmoscopic examination revealed an hypertensive retinopathy, Grade III. These findings were exactly similar to those on admission and discharge from the hospital.

COMMENT.—An analysis of the 20 cases of intra-abdominal apoplexy, tabulated in Table I, reveals some interesting points. The accident occurred much more frequently in males, with the ratio of occurrence approximately three males to one female. The ages varied from 27 to 73, but the average age was 51.5 years, with the greatest frequency of occurrence between the ages of 45 to 55 (Table I).

Ten cases (50 per cent) gave evidence at some time or other of a definite hypertension. Two other cases, or 10 per cent, in which no blood pressure reading was noted, were described as showing marked arteriosclerosis at postmortem examination. Thus, 60 per cent of the cases in this series presented evidence of organic cardiovascular disease in the nature of a diffuse vascular sclerosis (Table II).

TABLE II

SUMMARY OF ANATOMIC FINDINGS IN 20 CASES OF INTRA-ABDOMINAL APOPLEXY

Cardiovascular Changes				Definite Bleeding Point					No Definite Bleeding Point				
Hypertension		Arteriosclerosis		No. of Cases	Per Cent	Average Age	Normal C-V System		No. of Cases	Per Cent	Average Age	Normal C-V System	
No. of Cases	Per Cent	No. of Cases	Per Cent				No. of Cases	Per Cent				No. of Cases	Per Cent
10	50	2	10	14	70	56.5	1	7	6	30	41.5	4	65.6

In six cases (30 per cent) it was specially noted that no definite bleeding point could be found. It is of interest that these cases occurred among the younger individuals—the average age of this group being 41.5 years compared with 56.5 years in the group in which a definite bleeding vessel was found. In four of these six cases (66 2/3 per cent) the cardiovascular state was noted as being apparently normal (Table II). To state it another way: Of the five cases in the reported series in which it was definitely noted that the cardiovascular state was apparently normal, there were four cases (80 per cent) in which no definite bleeding point could be found either at operation or at postmortem examination. This close association between a younger-age occurrence, a relatively normal cardiovascular apparatus and the inability to demonstrate a bleeding point was striking and noteworthy. The practical

implication of this association becomes apparent when the operative results are analyzed.

Sixteen of the 20 cases in the series (80 per cent) came to operation (Table III). Of these 16, 11 cases (69 per cent) recovered. Of the five (31 per cent), in which operation resulted in death, four were cases in which no definite bleeding point could be found. It would seem from this, that despite the occurrence of a massive intraperitoneal hemorrhage if, at opera-

TABLE III

SUMMARY OF OPERATIVE RESULTS IN 20 CASES OF INTRA-ABDOMINAL APOPLEXY

Operated		Recovered				Died			
				No Def. Bl. Pt.				No Def. Bl. Pt.	
No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent
16	80	11	69	1	9	5	31	4	80

tion, a definite bleeding point can be located and the bleeding vessel ligated, the chance of recovery is excellent. This is especially to be emphasized, since we have been unable to find any reported case of repeated occurrence of intra-abdominal apoplexy. Thus, it would appear that in this condition there might be some advantage favoring the hypertensive-arteriosclerotic individual, in that such patients are much more likely to present a definite bleeding point that would permit of ligation.

In no instance was a correct diagnosis made prior to operation. The majority of cases were felt to represent perforated peptic ulcers. Of the various diagnoses made, the one which would seem most nearly related to the actual condition found was "mesenteric thrombosis." In our case the latter was the preoperative diagnosis. All cases were considered as acute abdominal catastrophes, but in every instance the encountering of an abdomen full of blood occurred as a distinct surgical surprise. A close analysis of the symptoms and signs in each of the reported cases reveals no one thing pathognomonic of the condition. There seems little, if anything, that can be recognized with any degree of certainty, to distinguish it from other acute abdominal emergencies. Of course, the absence in the history of such gross things as trauma, dominant digestive complaints, or previous similar occurrences, are of some aid in the differential diagnosis.

Morridor and Olivier,[1] in their exhaustive review of spontaneous hemoperitoneum in men, resulting from various causes, call attention to several signs and symptoms that were common to most of the cases. They all showed abdominal signs of peritoneal irritation, an elevated leukocyte count (over 10,000 in most cases), and a lowered erythrocyte level.

The quantitative status of the erythrocytes calls attention to the usefulness, in these cases, of recognizing that the clinical picture of vascular collapse which they present is due to internal hemorrhage rather than to shock from other causes. Moon[18] has called attention, on many occasions, to the significance of hemoconcentration as a diagnostic finding in true shock. A

demonstration of hemoconcentration by either repeated hematocrit readings, specific gravity determinations, hemoglobin determinations, or red blood cell counts would indicate a state of shock due to causes other than hemorrhage, whereas the failure to demonstrate hemoconcentration by the same means might suggest hemorrhage as a causative factor. Such a differentiation would appear to be of paramount importance in cases of spontaneous hemoperitoneum, where the most frequent condition, suspected clinically, is ruptured peptic ulcer with shock and, not in a single instance, internal hemorrhage.

There was noted in several of the cases, including our own, a phenomenon, to which Morridor and Olivier[1] have also called attention, namely, that a number of these cases described the occurrence of indefinite abdominal pain of variable nature prior to the acute accident for which they were eventually seen. They, in fact, found that about 25 per cent of the cases which they reviewed, described such pains months or years preceding the acute attack. They also described what they termed "treacherous calm" in about 20 per cent of their cases during which, in a few instances, clinical remissions were so great as to permit return to work.

The use of abdominal paracentesis as a diagnostic procedure in the presence of suspected hemoperitoneum has been recently reapplied by Johnston.[19] Despite the indifferent success of others who have employed the procedure in the past, Johnston feels that the danger of visceral injury is negligible and that exploratory abdominal puncture with the use of a trocar can be of inestimable value in the diagnosis of hemoperitoneum.

The etiology of the condition affords much interesting speculation. The high incidence of hypertension and arteriosclerosis in the reported series of cases (60 per cent) would strongly suggest that local disease of the blood vessels must be the basic, underlying factor. The recent report of Morlock[17] would tend to substantiate such a conclusion. He was able to demonstrate that the blood vessels of the gastro-intestinal tract of hypertensives of all ages as compared to normal individuals, showed measurable thickening of the arteriolar wall and reduction of the ratio of wall to lumen. There occurred early in such blood vessels, hyperplasia of the nuclear elements of the media followed later by degeneration and fibrosis. It is much more difficult to account, on the same local vascular disease basis, for the hemorrhages occurring in the younger individuals with an apparently normal vascular tree. The close association was pointed out above of a younger-age occurrence, the presence of an apparently normal vasculature, and the inability to demonstrate a definite bleeding point (Table II). Bruce,[14] discussing the cause for intra-abdominal apoplexy, suggested the presence in the abdominal vessels of a congenital weakness, similar to that which leads to aneurysm of the basal cerebral arteries. He quoted the work of Forbus, whose study on the development of the mesenteric vessels indicated that at the branchings of the arterial tree a gap exists temporarily between the independent formation of muscle coats on each branch. It was felt that developmental defects at the branchings, resulting from deficiency in the formation of muscle coats, might

explain the occurrence of small aneurysmal formations. Small aneurysmal dilatations, similar to that seen in cerebral arteries, have been reported in the mesenteric, pulmonic and renal arteries. Such conditions would lend an easy explanation for the occurrence of a massive intraperitoneal hemorrhage in younger individuals, free of known organic vascular disease. There is, of course, no sure proof that such a condition actually exists, but the demonstration of small aneurysmal rupture in Cases 6 and 17, where the surrounding abdominal vessels were noted to be normal, would lend support to such a theory.

The spontaneous feature in all these cases of intra-abdominal apoplexy is an important one. The precipitating cause is, in most instances, hidden and unexplainable. Given a basic, underlying local disease or congenital defect whose very nature is such as to weaken the vessel at the local site, then the addition of anything that would tend to raise the intravascular pressure within that vessel might well result in a rupture at the weakened site. In that way, and for that reason, hypertension, possibly, is found in so large a percentage of cases. Likewise, too, the use of pitressin, as in Morton's[15] case, sudden emotional strain, excessive eating, coitus, heavy lifting, and anything else which might raise the blood pressure could well predispose to a "blowing out" of a blood vessel.

Summary

(1) A case of massive spontaneous intra-peritoneal hemorrhage ("intra-abdominal apoplexy") is reported.

(2) Nineteen similar cases reported in the literature are analyzed.

(3) The striking features of the condition are noted, and a discussion is made of the possible etiology, the treatment and its results.

Conclusions

(1) Massive spontaneous intraperitoneal hemorrhage resulting from the nontraumatic rupture of a small intra-abdominal blood vessel (so-called "intra-abdominal apoplexy") is a rare condition, the occurrence of which is being reported with greater frequency.

(2) There is nothing pathognomonic in either the symptoms or signs that permit a certain preoperative diagnosis.

(3) The condition should be considered in all cases in which there occurs sudden severe abdominal pain, shock, and signs of peritoneal irritation especially in a known hypertensive.

(4) Hypertension and arteriosclerosis seem to be the dominant factors in the etiology of the condition. The possibility of the occurrence of small localized aneurysmal dilatations, particularly in younger individuals, has been suggested.

(5) Early operation, with ligation of the bleeding point, if possible, is the only treatment. This treatment is one which offers a high chance of recovery if a definite bleeding point be found.

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