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MESENTERIC VASCULAR OCCLUSION

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Mesenteric vascular occlusion bears a very serious prognosis. Maingot[1] found 700 cases in the literature and, of those subjected to resections, only 8% survived. In 1938 Whittaker and Pemberton[2] described 19 cases of which 16 died.

In 1940 Thomas Moore,[3] in his masterly article on this condition wrote: 'The mortality resulting from this disease is still very high. This review is presented in the hope that further attention may be drawn to the condition. The present high mortality can only be reduced by earlier diagnosis and treatment'; yet, today the prognosis is not materially improved.

In the index of the Johannesburg General Hospital for the 6 years from 1953 to 1958, there are 24 cases listed under 'mesenteric thrombosis'; of these 24 cases 21 died.

HISTORY

Mesenteric vascular occlusion was first described by Tiedman in 1843 (quoted by Trotter[4]). In 1894 Elliott (quoted by Lower and Glazier[5]) described the first successful resection for this condition. In 1921 Klein[6] found only 24 cases of successful resection for the same condition. Since then a large number of cases has been reported. In 1940 Brown[30] found 722 cases in the literature.

INCIDENCE

Mesenteric vascular occlusion is not a rare disease; it is estimated to occur in 0·1% of all surgical admissions. This figure is, in all probability, an under-estimation, since many cases of transient abdominal pain, due to minor episodes of thrombosis, go undiagnosed.

The condition may be classified as follows:

Acute. (1) Arterial — due to embolism or thrombosis; (2) venous — due to thrombosis.

Chronic. Intestinal angina.

It is, therefore, quite incorrect to use the term mesenteric thrombosis when referring to this whole group of conditions. An effort should be made to use more precise nomenclature.

ANATOMY

Points of Interest

Mesenteric arterial occlusion may involve either the superior or the inferior mesenteric artery. Harkins[7] found that 1 in every 9 cases involved the inferior mesenteric artery. The greater incidence in the superior mesenteric artery is explained by the greater diameter of that vessel and the fact that it runs more nearly parallel to the aorta at its origin.

The superior mesenteric artery supplies the gut from the duodenum to the proximal two-thirds of the transverse colon. It may, however, be the only mesenteric artery and supply the whole of the small and large gut. Harkins reports a case in which this artery was occluded and the whole of the gut including the rectum was gangrenous.

The first branch of the superior mesenteric artery is the inferior pancreaticoduodenal artery which anastomoses with the superior pancreaticoduodenal artery to supply the duodenum. This branch may form a channel for the development of a collateral circulation when there is occlusion of the proximal part of the superior mesenteric artery. Several such cases are on record.

The venous drainage of the bowel is *via* the portal system. There are 2 significant points: (1) The portal system contains no valves, so that, if the pressure in the arterial system falls, due to arterial occlusion, blood flows back along the veins to cause congestion of the bowel; and (2) normally the pressure in the portal system is very low (*vide infra*).

PATHOLOGY

The effect of cutting off the flow of blood from an organ is dependent upon the rapidity with which a collateral circulation is established. A sudden occlusion is far more likely to produce an infarction than a gradually developing occlusion, because the collateral circulation may have time to develop before permanent damage occurs.

It may be thought that in the mesenteric vessels a small blockage could always be overcome, because of the extensive branching and anastomoses of the vessels. This is not so, since spreading secondary thrombosis usually occurs in these cases.

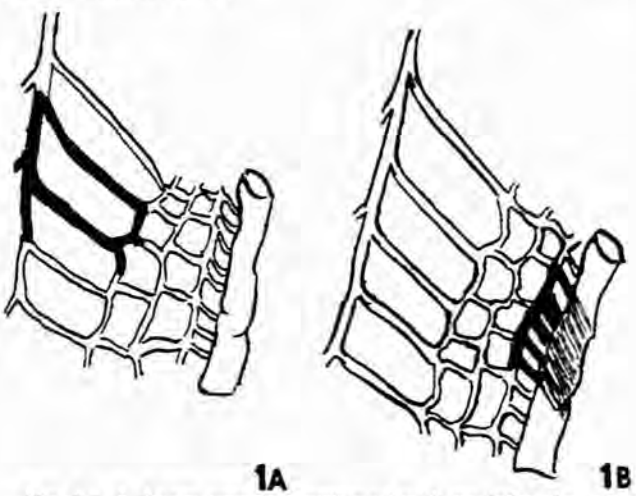

Fig. 1.[10] *A.* Large vessel thrombosed with no infarction of bowel. *B.* Vasa recti thrombosed with infarction of bowel.
[10] Adapted from Johnston and Baggenstos.[10]

There are 3 factors which favour the development of this spreading secondary thrombosis: (1) There is a fall in the pressure in the vessels following the block; (2) the pressure in the portal system is normally low; and (3) in the more extensive cases there is a loss of blood into the bowel causing oligaemic shock, with a further fall of blood pressure in these vessels.

If a number of vasa recti are blocked, the bowel supplied by them will become infarcted, but if some of the larger mesenteric vessels are occluded there may or may not be infarction of the bowel (Fig. 1).

There must indeed be many cases of this type which recover without operation or without being diagnosed.

Karcher[5] described a case which bears this out. Seven weeks after an attack of mesenteric vascular occlusion, the patient died of another cause. At autopsy there was a large thrombus in the superior mesenteric artery, but no infarction in the bowel.

This consideration suggests 2 interesting possibilities: (1) There may be sufficient blood supply to prevent infarction, but not enough to allow bowel function, thus causing a localized intestinal ileus; and (2) although the blood supply may be just adequate with a normal blood pressure, any fall in blood pressure at a later date may then cause an infarction of the bowel.

The infarction produced by mesenteric arterial occlusion is usually haemorrhagic. The bowel is infiltrated with blood, the mucosa is plum-coloured and swollen, and the lumen is filled with altered blood. Gradually areas of gangrene develop and infection spreads through the devitalized wall. There are similar changes in the mesentery if the obstruction is some distance from the gut. There is a blood-stained effusion into the peritoneal cavity.

The infarction may rarely be anaemic, and Trotter[4] reported 7 of these cases in the 359 cases he reviewed. In venous thrombosis the infarction is always haemorrhagic.

AETIOLOGY

The causes of mesenteric vascular occlusion may be summarized as follows:

A. Mesenteric Arterial Occlusion

1. *Embolism* may arise from: mitral valves, patent ductus, left auricular fibrillation, septic infarcts of lung, or atheromatous plaques on aorta.

2. *Thrombosis* is rare. It occurs as a result of: atherosclerosis, thrombo-angiitis obliterans, pressure by a tumour, diabetes, or dissecting aneurysm.

B. Mesenteric Venous Occlusion

1. *Primary*. "There is no doubt that the number of primary cases will diminish the more complete and detailed is the search for the cause" — Moore.[3]

2. *Secondary*. Secondary cases may be the result of: portal obstruction, cirrhosis of the liver, polycythaemia vera, splenectomy, abdominal sepsis (such as acute appendicitis, ulcerative colitis, or strangulated bowel), or carcinoma of the rectum.

Gordin and Laurent[9] reviewed 47 cases of mesenteric vascular occlusion. Of 42 cases operated on, 37 were due to occlusion of the superior mesenteric artery and only 5 were due to occlusion of mesenteric veins.

In a review of 99 cases of mesenteric venous occlusion coming to autopsy, Johnston and Baggenstos[10] gave the aetiology as follows: infection, 28; neoplasms, 27; abdominal operations, 23; cirrhosis of the liver, 21; splenomegaly, 17; cardiac failure, 9; splenectomy, 5; and no cause found, 8. In some instances several probable causes were found in the same case.

AGE INCIDENCE AND MORTALITY

The age incidence is shown in Table I and the mortality rates in Table II.

TABLE I. AGE INCIDENCE OF CASES OF MESENTERIC VASCULAR OCCLUSION ACCORDING TO VARIOUS REPORTS

Report	No. of cases	Ages: Under 20	20 - 30	30 - 40	40 - 50	50 - 60	60 - 70	70 —
Forty[19]	32	—	1	3	1	4	8	15
Johnston and Baggenstos[10] ..	99	5	10	8	19	32	19	6
Brown[20]	91	—	18	18	17	24	13	1
General Hospital, Jhbg. ..	24	—	1	—	6	4	7	6

TABLE II. MORTALITY OF CASES OF MESENTERIC VASCULAR OCCLUSION ACCORDING TO VARIOUS REPORTS

Date	Report	No. of cases	Deaths	%
1938	Whittaker[2] ..	19	16	84
1940	Moore[3]	8	4	50
1940	Brown[20]	104	?	68
1955	Maingot[1] ..	700	?	92
1957	Forty[19]	32	29	90
1959	Present series ..	24	21	87

CLINICAL FEATURES

Mesenteric vascular occlusion may present as follows: (1) symptomless — it may be discovered by aortography, (2) vague abdominal pain and nausea, (3) insidious onset with symptoms becoming worse until there is a fully developed infarction, (4) sudden abdominal catastrophe — this is the most typical type, or (5) intestinal angina.

The usual clinical presentation is an urgent abdominal catastrophe. There is sudden onset of persistent central abdominal pain, vomiting, and constipation or the passage of a little altered blood in the stool. There may be profound shock and sweating. The patient is pale and restless, with a low blood pressure, and a rapid pulse of poor volume. The abdomen becomes slightly distended, tenderness and rigidity are not marked, and bowel sounds are excessive. Later, signs of generalized peritonitis develop.

DIAGNOSIS

Trotter[4] reviewed 359 cases of mesenteric vascular occlusion. In only 4% was the diagnosis made preoperatively or before autopsy. Nevertheless, if the condition is kept in mind it should be recognizable.

Predisposing causes should be looked for, e.g. cardiac lesions or the history of loss of limbs or toes through vascular disease. The two-enema test may produce a little altered blood. The white-cell count is raised. X-ray of the abdomen may show an isolated dilated loop. Shaw and Rutledge[15] stated that there are few bowel shadows on X-ray.

TABLE III. CASES OF MESENTERIC VASCULAR OCCLUSION RECORDED IN THE INDEX OF THE JOHANNESBURG GENERAL HOSPITAL, 1953 - 58

Case	*Sex Age*	*Clinical picture*	*Diagnosed*	*Treatment*	*Result*
1	M 41	Acute appendicitis followed by generalized abdominal pain	At autopsy	IV drip and suction	Died on 8th day
2	M 43	Abdominal pain for 5 days. Vomiting	At laparotomy	44 in. of small bowel resected. Heparin	Died 24 hours postoperatively
3	M 46	Resection for mesenteric thrombosis 3 months before	Not confirmed	Symptomatic	Died 3 months postoperatively
4	M 66	Abdominal pain for 6 hours. Melaena	Not confirmed	Symptomatic	Died 2nd day
5	M 50	Abdominal pain for 3 days	Confirmed at autopsy	Symptomatic	Died 1st day
6	F 70	CCF, fibrillating. Abdominal pain	Not confirmed	Symptomatic	Died 1st day
7	M 44	CCF, mitral stenosis. Abdominal pain for 4 hours	Not confirmed	Symptomatic	Died 6th day
8	F 84	Abdominal pain for 3 days	Not confirmed	Symptomatic	Died 3rd day
9	F 66	CCF, aortic stenosis. Abdominal pain for 1 day	Not confirmed	Symptomatic	Died after 8 hours
10	M 44	Abdominal pain for 5 days	At laparotomy	18 in. of bowel resected. Heparin postoperatively	Died 6th postoperative day
11	M 87	Auricular fibrillation. Abdominal pain for 2 days	Not confirmed	Heparin	Died 2nd day
12	M 63	Abdominal pain for 2 days. Previous arteriosclerosis	At laparotomy	8 in. of bowel resected	Died 4 weeks postoperatively
13	F 80	Abdominal pain for 2 days	Not confirmed	Symptomatic	Died 2nd day
14	F 56	Mitral stenosis and fibrillation. Abdominal pain	At autopsy	Symptomatic	Died 10th day
15	F 24	Mitral stenosis. Sudden collapse. Melaena	Not confirmed	Symptomatic	Died in 6th week
16	F 71	Admitted comatose. Had complained of abdominal pain	Not confirmed	Symptomatic	Died after 1 hour
17	M 67	Coronary thrombosis. Arteriosclerosis. Abdominal catastrophe	Not confirmed	Had dicoumarol	Died in 2nd week
18	M 68	CCF. Melaena. Collapse	Not confirmed	Symptomatic	Died 1st day
19	M 65	Abdominal pain for 2 days. Vomiting	Confirmed at laparotomy	All except 18 in. of small bowel resected	Recovered
20	F 59	Cerebral thrombosis 2 days previously. Abdominal pain	Not confirmed	Symptomatic	Recovered
21	F 59	Rheumatic heart fibrillating. Abdominal pain. Melaena	Not confirmed	Heparin. Dicoumarol	Recovered
22	F 64	Abdominal pain for 2 days	At autopsy	Symptomatic	Died 2nd day
23	M 73	Abdominal pain for 7 hours. Melaena	Not confirmed	Symptomatic	Died 6th day
24	M 42	Obliterative vascular disease. Both legs previously amputated. Abdominal pain for 4 days	Pre-operatively	Resection of 4 ft. of small bowel	Died 2nd postoperative day

CCF = Congestive cardiac failure.

Abdominal Angina

In 1936 Dunphy and Whitfield[11] described 12 patients who had died of acute mesenteric occlusion. Seven of these patients had been seen, complaining of the following symptoms for a few months: recurrent abdominal pain after meals, weight loss, and occasional diarrhoea. Clinical examination and X-ray of the abdomen had shown nothing abnormal.

These patients had been suffering from abdominal or intestinal angina, due to ischaemia of the bowel. This is now a well-established clinical entity and an endeavour should be made to diagnose and treat this condition before the disaster of acute mesenteric vascular occlusion occurs.

Berman and Russo[12] recorded a case in which the diagnosis was made at laparotomy — no resection was done. The patient was alive and well 6 years later on anticoagulant therapy.

Endarterectomy is a practical procedure before the onset of an acute mesenteric vascular occlusion, whereas the mortality in emergency surgery may be greater than that of bowel resection.

TREATMENT

As may be expected in a condition with such poor prognosis, much has been written on the treatment of acute mesenteric occlusion and there is a great divergence of opinion.

Thomas Moore[3] recommends wide resection and anastomosis. Russell[13] says there is general agreement that if possible the bowel should be resected and anastomosed. Murray[14] recommends starting heparin therapy during the operation. Laufman and Scheinberg[15] make the point that heparin should only be given if bowel has been resected because there will be an increased blood loss into the bowel if the devitalized bowel remains. Aird[16] states that at Hammersmith Hospital, London, cases are treated on heparin, blood transfusion and antibiotics and only go to laparotomy if peritonitis develops. However, he admits that results of surgery are slightly better. Gordin and Laurent[9] state that heparin should be used postoperatively and, when there is massive infarction, heparin only should be used.

Mesenteric Embolectomy

Klass,[17] in 1951, was the first to undertake embolectomy in acute mesenteric vascular occlusion. His 2 patients died, 1 of ventricular failure and the other of haemorrhage from the mesenteric vein. Shaw and Rutledge[18] reported the first survival after mesenteric embolectomy in 1957. Since then they have had other survivals. Their cases were treated on anticoagulants given immediately after operation.

The success of embolectomy does not only depend on surgical technique but also, and primarily, on early diagnosis. The operation must, of course, be done before irreversible changes have occurred in the bowel. Immediate restoration to unquestionable viability is unlikely and so it is generally necessary to re-explore the patient's abdomen 24 hours later, to ascertain whether the bowel has survived.

CASES AT JOHANNESBURG GENERAL HOSPITAL 1953 - 1958

There were 24 cases of which 21 died. The diagnosis was confirmed by operation or autopsy in only 9 cases. However, in several of the others, there was very strong presumptive evidence of the correct diagnosis having been made (Table III).

Of the 9 confirmed cases 5 had bowel resections with 1 survival, while 4 cases treated conservatively came to autopsy.

It would be misleading to exclude the unconfirmed cases from this series as that would exclude the cases treated successfully by conservative measures and those cases for which permission for autopsy could not be obtained.

Comparing the cases at the Johannesburg General Hospital with those reported by Frank Forty,[19] we find that of 19 cases not operated on in Johannesburg, 17 died, while of 19 in Frank Forty's series who were not operated on, all died. Five cases in Johannesburg had bowel resection — of these 4 died; in Frank Forty's series, 6 cases had bowel resection and of these 4 died. Frank Forty reports a laparotomy only in 7 cases; of these 6 died.

At the Johannesburg General Hospital heparin was given to only 5 cases; of these 4 died and 1 survived.

Of the 5 cases subjected to operation, only 2 were diagnosed pre-operatively, and 1 of these 2 survived. Many cases were unsuitable for surgery but in several the diagnosis was missed until it was too late.

These cases of mesenteric vascular occlusion at the Johannesburg General Hospital present the same gloomy picture as those described elsewhere. The delay in diagnosis is partly responsible for the very poor prognosis and irrespective of other advances this is the main factor which must be improved before we can expect any fall in the high mortality.

SUMMARY

1. A brief review of the literature is given, with a review of the aetiology, clinical features, diagnosis and treatment of mesenteric vascular occlusion.

2. A plea is made for the use of more precise nomenclature in referring to this condition.

3. There were 24 cases of mesenteric vascular occlusion at the Johannesburg General Hospital between 1953 and 1958, of which 21 died. The delay in diagnosis is partly responsible for the very poor prognosis.

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