Chapter 1
History of Mesenteric Vascular Disease

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Although it is generally stated in any short historical introduction to an article concerning chronic mesenteric ischemia that Dunphy first correlated chronic abdominal pain to subsequent mesenteric artery occlusion and gut infarction in 1936, his paper did not arise suddenly from a barren field [1]. The problem with "mesenteric occlusion" and death from ischemic necrotic bowel had interested physicians for years previously. The problem was a complex one, but the impediments to understanding mesenteric ischemia and treating it were dishearteningly simple: neither diagnostic angiography nor vascular intervention was extant. Diagnosis was made during exploratory laparotomy for acute abdominal crises or at autopsy. Bowel resection was the sole surgical option.

Tiedemann had described mesenteric occlusion and bowel infarction in a patient in 1843 [2]. Seven years later, Virchow added two further such patients to the literature [3]. Welch, in 1887, had posited an 80 % stenosis of the SMA was necessary for ischemic bowel changes [4]. In 1904, Jackson, Parker, and Quinby described both arterial and venous occlusions of the mesenteric circulation [5]. Trotter, in 1913, reviewed 359 cases of infarcted bowel [5]. He proposed a relationship between heart disease and embolus to the superior mesenteric artery and a relationship between arteriosclerosis of the aorta and mesenteric vessels and local thrombosis of the visceral vessels. Klein pointed out in 1921 in his thesis on embolism and thrombosis of the superior mesenteric artery a relationship between superior mesenteric artery stenosis and episodic abdominal pain [6].

Cokkinis, a registrar at the London Lock Hospital, wrote a thesis in 1926, which is remarkable for several observations [7]. He reported 76 cases of "mesenteric occlusion" mostly from the London Hospital. He felt primary thrombosis of the mesenteric vessels rare, but reported one case with gangrene of the intestines and

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both lower extremities, and felt that atheromas of the aorta and mesenteric arteries themselves were causative, leading directly to thrombosis. He also described aortic-origin emboli.

Ten years before Dunphy's postmortem study could confirm a history of postprandial pain and subsequent gut infarction, Cokkinis wrote: "The patient complains of abdominal symptoms extending over a period of weeks or months. Among the commonest of these are: colicky abdominal pain, which may have some relation to food…The symptoms are colicky abdominal pain, 1½ to 2 h after meals, nausea and vomiting…they may last for years and then arterial thrombosis supervenes and leads to infarction…The pathological lesion is one of arteriosclerosis of the mesenteric arteries, interfering with the flow of blood to the intestines during digestion."

Given the lack of diagnostic modalities of the day, this is the most remarkable and accurate description of chronic mesenteric ischemia. All that is lacking for completeness sake is weight loss and fear of eating.

In 1936 in his famous report, Dunphy described 12 patients dying of mesenteric infarction studied at autopsy. Seven (58 %) had a history of recurrent abdominal pain proceeding the terminal event, a period of time ranging from weeks to years. The imperative for early treatment was thus identified, even if the means were not yet available.

Surgical Revascularization

In 1951, Klass performed direct embolectomy of the superior mesenteric artery in two patients [8]. One must remember at this point that the Fogarty catheter had not yet been invented. Both patients died, but the mesenteric circulation was free of thrombosis at the postmortem. Stewart, that same year, performed an SMA embolectomy [9]. Five years later Van Weel reported a successful thrombectomy, although the patient required subsequent resection of the distal ileum and cecum [10]. This would count as a success today. In 1957 Mikkelsen described the arteriographic findings of ostial mesenteric lesions [11].

The first embolectomy of the SMA to be successful and not to require subsequent bowel resection was performed in 1957 and reported by Shaw and Maynard [12]. Shaw with Rutledge in 1958 [13] reported endarterectomy of the SMA and paramesenteric aorta as treatment of chronic mesenteric ischemia. The remarkable Houston surgeons – Morris, Crawford, Cooley, and DeBakey – in 1962 reported retrograde reconstruction of the celiac and superior mesenteric arteries. It was associated with tortuosity and kinking of those grafts in some patients [14].

Wylie, Stoney, and Ehrenfield, in the 1970s, described both transaortic visceral endarterectomy and antegrade supraceliac bypass to the visceral vessels [15]. Initially, when performing endarterectomy, they employed a thoraco-retroperitoneal approach but modified this to medial visceral rotation in later years for appropriate patients.

Mayo Clinic Legacy

Hollier et al proposed in 1981 that complete revascularization of all three mesenteric vessels was the ideal [16]. He found that recurrence with one-vessel reconstruction was 26 % and that with 3-vessel reconstructions was less than 10 %. However, that Mayo Clinic cohort included no antegrade reconstructions. Further experience from the Mayo Clinic showed that obsession with three-vessel reconstruction increased mortality from the current 8–10 % in that day to 15–20 % [17]. The mortality in the 1980s was right around 8–10 %, second only to repair or thoracoabdominal aortic aneurysms in terms of risk to the patient. In the succeeding decades, with improved anesthetic techniques, refinement of operative approaches and appropriate patient selection, mortality has steadily decreased and is currently in the 2–5 % range.

Antegrade reconstruction was felt to be the gold standard of repair, but clamping of the paravisceral aorta was not without risk, especially in elderly patients with coronary artery disease, associated renal artery disease, and aortoiliac occlusive disease.

The group from Oregon modified infrarenal or retrograde bypass to bring the distal end of the graft in a curving manner such that the visceral artery anastomosis was constructed in an antegrade manner, to decrease turbulence of flow. Initially, they reconstructed both the celiac and superior mesenteric arteries [18, 19]. Later experience revealed that superior mesenteric artery reconstruction alone was satisfactory.

The Mayo group subsequently reported 91 cases [20]. That study was postulated on the premise that antegrade reconstruction would prove, albeit in a retrospective study, superior to infrarenal repair. That hypothesis was proved false. In properly selected patients, isolated retrograde reconstruction of the superior mesenteric artery was statistically no different than antegrade reconstruction. Further, reconstruction originating from a common or external iliac artery was felt to be superior to that originating from the infrarenal aorta, because the long axis of the graft is parallel to that of the aorta, as opposed to the perpendicular orientation seen with grafts originating from the infrarenal aorta, thereby eliminating the kinking seen in the latter group of grafts when the viscera are returned to their normal position. In addition, there is a subset of patients with densely calcific aortas whose iliac arteries are spared and thus provide superior donor sites.

Endovascular Therapy

Endovascular treatment of mesenteric disease was introduced in 1980. It is used in the majority of patients today, as its mortality is less in the hands of most practitioners than open repair. Further studies from the Mayo Clinic by Oderich et al detailing 229 patients, on the other hand, have shown that the mortality from open repair is less than 3 % and is in essence equal to that of endovascular repair. Nonetheless, these are retrospective studies from a high-volume institution with a long-standing interest in the problem, and the patients are carefully selected [21].

Those patients with flush occlusions of the celiac and superior mesenteric arteries and those with long calcific occlusions of the superior mesenteric artery are probably better treated by open repair. Multiple studies have shown less re-intervention after open repair (reflecting the same experience seen in most vascular beds when open and endovascular reconstructions are contrasted). Fortunately, open repair may be safely tailored to the patient's anatomy and physiology in this day and age, ranging from antegrade supraceliac reconstruction of the both the celiac and SMA, usually reserved for young relatively healthy patients whose life expectancy is long, to grafts originating from the iliac arteries and carried to the superior mesenteric artery in more elderly fragile patients. Whereas prosthetic grafts have historically outperformed saphenous vein for mesenteric reconstructions, retrograde saphenous mesenteric bypasses performed in the face of infection appear to have very acceptable patency rates.

The history of reconstructions for chronic mesenteric ischemia has been a progression. Currently, patients may be reconstructed via transaortic endarterectomy, antegrade graft reconstruction, or retrograde repair. The choice is usually made dependent on anatomy, physiology, and the applicability of endovascular techniques to these patients.

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