TUMORS OF THE INTESTINES

DAVID A. WOOD, M.D.

ATLAS OF TUMOR PATHOLOGY

Section VI—Fascicle 22

TUMORS OF THE INTESTINES

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David A. Wood

TUMORS OF THE INTESTINES

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TUMORS OF THE INTESTINES

INTRODUCTION

The conventional anatomic divisions of the intestinal tract are as follows: (1) the small intestine that consists of the duodenum, with supra-ampullary, infra-ampullary, and periampullary portions, the jejunum, and the ileum; (2) the large intestine that consists of the cecum, the vermiform appendix, the ascending colon, the hepatic flexure, the transverse colon, the splenic flexure, the descending colon, the sigmoid colon, and the rectum; and (3) the anal canal. Generally, surgical descriptions refer to only three subdivisions of the large intestine: (1) the right colon, extending from the ileocecal junction to a point just beyond the hepatic flexure; (2) the midcolon, lying between the hepatic and splenic flexures; (3) the left colon, coursing the splenic flexure to the beginning of the rectum. Some surgeons designate only: right colon, left colon, and rectum.

Tumors involving the intestinal tract are chiefly epithelial in type. These tumors and the nonepithelial tumors, of which there is a small percentage, vary greatly in natural history, clinical behavior, etiology, and site of origin; they also vary in rapidity of growth, size, shape, cellular arrangement, spread, prognosis, and metastatic potential. The variations are determined by biologic factors, structural components, anatomic sites, boundaries, segmental zones, peritoneal relationships, venous blood return, lymphatic drainage, and by tissues contiguous to the tumors.

The nonepithelial tumors also vary in type; in general, nonepithelial intraluminal tumors such as lipoma and lymphangioma are benign, and most extraluminal forms of the tumors are malignant. Although few comprehensive descriptions of these tumors have been published, a plethora of reports of single cases and of a small series of cases is available; the paucity of adequate descriptions of the characteristics of most of these nonepithelial tumors is presumed to be due to their rarity. Most of these tumor types may be situated in any portion of the intestinal tract, but certain of the rarer neoplasms such as neurofibroma, angioma, and lymphosarcoma occur predominantly in the small intestine.

Although tumors of the small intestine constitute only 1 to 3 percent of all tumors of the intestinal tract (table I), I have devoted a limited, but seemingly disproportionately large, part of this fascicle to the rarer tumors of this segment.

Carcinoma, carcinoid tumor, leiomyosarcoma, and lymphosarcoma are the most common tumors of the small intestine. These tumors differ in their

sites of predilection, adenocarcinoma being the most prevalent in the duodenum, leiomyosarcoma in the jejunum, and lymphosarcoma in the ileum.

Some of the interesting and unexplained vagaries of intestinal neoplastic disease are provided by the rarity of occurrence of tumors from the pylorus to the ileocecal valve, the rare occurrence of tumors of mesodermal origin, and the infrequency with which gastric carcinoma involves the contiguous duodenal mucosa by continuity.

The high incidence of carcinomas in the stomach and rectum—the portals of entry and of exit of the gastrointestinal tract—is also of interest. Approximately three fourths of all malignant intestinal tumors are situated within view of the sigmoidoscope; one-half are within reach of the examining finger.

As with neoplasms elsewhere, biologic factors play a poorly understood role in the tumor-host relationship of neoplasms of the intestinal tract; these factors probably influence localization, growth, and metastasis. For example, a bulky tumor may remain localized, but a small lesion may infiltrate the area by extensive lymphatic permeation, regional lymph node involvement, intravasal (venous) invasion, and systemic metastasis. The regression of occasional epithelial tumors, such as adenoma and carcinoma, of the sigmoid colon and the rectum, after diversion of the fecal stream is also noteworthy (Dunphy et al.).

Contributory causal factors in the etiology of tumors of the intestinal tract may be local or general. Local factors are often associated with the development of epithelial growths: pre-existent benign overgrowths, such as hyperplasia, and adenoma; metaplasia; inflammatory disease, such as chronic ulcerative colitis; physiological differences between different segments of the tract, such as between the small intestine and the left colon; embryologic abnormalities, such as persistent vestigial structures, anatomic defects, and heterotopic tissue; and possible tissue-specific chardcteristics that are peculiar to a particular anatomic site, such as the appendix vermiformis. General factors such as genes, metabolism, endocrines, and development may play an important role; they may affect the entire tract in certain tumors, but more often the nonepithelial tumors: neurofibroma, lipoma, hemangioma, and lymphangioma.

Notable examples of the role of genetic factors in the development of intestinal tumors are familial adenomatosis and Peutz-Jeghers syndrome.

Although developmental and embryonal factors may contribute to the etiology of certain intestinal tumors, their role is vague, ill defined and controversial. Both local and general factors seem to participate in the formation of some of these tumors. Hypothetic carcinogens are also considered for completeness; however, the evidence concerning possible carcinogen-containing food additives and possible endogenous carcinogens that are the results of metabolic defects is still not convincing. Viral agents are also

suspected by some research investigators to be responsible for the development of polypoid adenomas and for the association of carcinomas with chronic ulcerative colitis, but this hypothesis has not been proved. Despite isolated case reports that suggest the possibility, amebiasis and bilharziasis play no known causal role.

Although the association between pre-existent benign epithelial overgrowths and the development of carcinoma is in many cases striking, a causal relationship has not been proved. It is thought, however, that localized mucosal hyperplasia may be a precursor of adenoma and, as such, be of causal significance. Squamous and osseous metaplasias also arouse considerable interest, but being rare, these changes probably make minimal, if any, contribution to causation. Squamous metaplasia is more prone to become malignant; foci have been found in supra-ampullary duodenal carcinomas (Stewart and Lieber), in the colon, and in association with mixed epithelial tumors such as adenoacanthoma and "collision tumors." Osseous metaplasia, however, is predominantly a passive and a benign component of the stroma of adenomas and carcinomas (Dukes, 1939, 1949); on rare occasions, evidence of this change has been seen in regional metastatic lesions (fig. 152).

The contribution of inflammatory diseases to the etiology of intestinal carcinoma is interesting but poorly understood, as illustrated by the contrasting entities of chronic ulcerative colitis and chronic duodenal ulcer. In the former, the incidence of carcinoma is undeniably higher (Bargen et al.), but there is absolutely no direct correlation between the incidence of the latter and of carcinoma of the duodenum (Hinton). Occasionally, diverticulitis coexists with carcinoma and resembles certain features of the segmental obstructive type of carcinoma; however, the association is wholly fortuitous.

Differences in function of certain segments of the intestinal tract may either maximize or minimize the tendencies toward tumor growth and development. The relative freedom from stasis with consequent rapid flow of intestinal contents, and the relative paucity of bacterial flora in the small intestine may in some way be responsible for the strikingly lower incidence of carcinoma in this segment of the intestine than in the sigmoid colon and the rectum.

Except in man, gastric carcinoma and spontaneous malignant tumors of the intestinal tract are rare in all mammals that have been observed; therefore, these tumors would seem to be excellent examples of species specificity. Malignant tumors of the intestinal tract are rarely found in the usual laboratory animals such as mice, rats, dogs, and hamsters. Intestinal carcinoma, when present in mice, is situated chiefly in the prolapsed rectum and more often is squamous than adenocarcinomatous. Intestinal adenocarcinoma in mice has been induced with carcinogens such as methylcholanthrene (Stewart and Lorenz, 1941, 1942). The few para-anal tumors that have been found

in older dogs have been well circumscribed, subepidermal, nonmetastasizing growths that originated from glands and consisted of rather characteristic columns and clumps of large polyhedral cells. Polypoid adenomas that also are termed polyps have been found in the large intestines of hamsters.

Tumors described in this fascicle originate in any site in the intestinal tract; however, tumors arising from the visceral peritoneum are not included (see Fascicles 23 and 24, "Tumors of the Retroperitoneum, Mesentery, and Peritoneum"). To aid in differential diagnosis, certain entities that are productive of benign masses or of pseudotumors are discussed. These include non-neoplastic masses that arise through faulty embryogenesis, localized diverticulitis, stenosing endometriosis of the colon, mucocele of the appendix vermiformis, eleoma (paraffinoma), amebic granuloma, cicatrizing segmental colitis, and "eosinophilic" granuloma of obscure origin. Tumors in persistent vestigial structures vary slightly, if at all, in cell type from similar tumors elsewhere.

Many physiologic and pathologic features contribute to marked differences in the gross appearance of benign and malignant intestinal tumors. Fluidity and flow of the intestinal stream influence the direction of tumor growth, that is, whether it shall be intraluminal or extraluminal. Most intestinal tumors of epithelial origin are intraluminal, since a common characteristic of intestinal tumors is a proneness to grow toward the area of least resistance. Since the passing intestinal stream usually pulls on the intraluminal tumor sufficiently to form a pedicle, these tumors may exhibit different degrees of pedunculation. Many of the larger intraluminal tumors are benign.

The extraluminal tumors are rare, but when they occur, these exophytic growths usually attain greater size than intraluminal tumors; they are also the more commonly malignant of the two tumor types. Symptoms tend to be minimal or absent until late in the course of extraluminal tumors; therefore, prognosis is poor. The usual range in tumor size is from barely visible masses that are a few millimeters in size to 10 to 15 cm. or more in diameter.

In the small intestine and in the ascending colon, the fluidity of the intestinal contents is greater than in the sigmoid colon; therefore, obstruction does not develop as early in the growth of the tumor. In the small intestine particularly, malignant disease, whether extraluminal or intraluminal, is rarely diagnosed before extension or metastasis has rendered it incurable.

The form and the size of intestinal tumors are related to such factors as the anatomic site of origin and the predominant direction of tumor growth, that is intraluminal, extramural and mural. An hourglass shape may be encountered in a few tumors, such as smooth muscle tumors that may exhibit both an extraluminal and an intraluminal growth.

Characteristically, a constrictive growth encircles the intestinal lumen and reduces it in size until only a narrow, distorted passage remains;

occasionally, the lumen may even be almost obliterated. The growth may also be either segmental or of "napkin-ring" configuration. In the small intestine, a segmental constrictive growth is usually associated with malignant lymphoma; in the descending colon, it is more characteristic of carcinoma. In the large intestine, it is more common in the descending colon and the sigmoid than in the ascending colon and the cecum. In some instances, segmental constrictive growths eventually ulcerate deeply; excavation may proceed until only a hollow, irregular sphere remains through which the "lumen" passes and becomes constricted at both its entrance and its exit.

Infiltrative growths that are ulcerative in type quite readily extend through the muscularis propria and invade the surrounding peritoneal and fatty tissues of the mesocolon and of the mesentery. Such tumors, especially carcinomas, are prone to be small, to ulcerate, to metastasize early, and partially to encircle the lumen.

Polypoid tumors often attain relatively large bulk even though they may only slightly infiltrate the intestinal wall. Those which are intraluminal rarely enlarge to more than 6 cm. in width before symptoms of obstruction reveal their presence. Since many of the polypoid carcinomas are well differentiated histologically, they may be slower to metastasize. Eventually, however, many of these tumors ulcerate; occasionally, only their broad, irregular ulcer base remains.

In the intestinal tract, diffuse growth is the rarest of the various forms of tumor growth. It is most common in highly anaplastic carcinomas and spreads well beyond its gross boundaries by continuous permeation of submucosal lymphatics. The margins are poorly defined and the surface is "pebbled" rather than ulcerated. When carcinomatous and when accompanied by a desmoplastic reaction, this form of growth is occasionally referred to as linitis plastica.

Although tumors that have divergent histogenesis, such as nerve sheath tumors, melanomas, carcinomas, carcinoids, and sarcomas, may share characteristics of the modes of extension and spread and metastatic phenomena, they do differ; the differences, however, are not always distinctive. Carcinoma spreads in a generally characteristic pattern, except as modified by the degree of cellular differentiation and the histologic type, such as is found in mucoid adenocarcinoma. Sarcomas as a group, with the possible exception of the malignant lymphomas, tend to be external; they grow extramurally and spread into the subserosa. In some instances, they extend into the mesentery or the mesocolon. In general, sarcomas and neurogenic intestinal tumors do not metastasize as readily as do carcinomas. Even though anal melanomas and carcinoid tumors may show a more variable and unpredictable tendency to invade veins earlier than carcinomas, their predominant initial route of spread is through the lymphatic system.

A summary of the modes of direct extension and of spread of intestinal tumors is as follows:

I. Direct extension
    - Intraluminal
    - Intramural
    - Extramural to contiguous structures such as the vagina, the uterus, and the uterine appendages

II. Spread (continuity and discontinuity)
    - Vascular system
        - Venous system
        - Portal circulation
        - Systemic circulation
    - Lymphatic system
        - Permeation
        - Embolism
    - Peritoneum
        - Local
        - Gravitational
        - Ovarian (Krükenberg)

III. Implantation in the line of bowel resection, the peritoneum, and the abdominal wall

Initially, the spread of all malignant tumors is by direct continuity. The extent of this spread depends upon their histogenesis and their sites of origin. Although characterized by eventual invasion of lymphatics and venules, the initial spread of carcinomas is mainly by direct intramural and extramural continuity. However, nonepithelial tumors may penetrate cleavage planes and ultimately may adhere to contiguous structures, with ensuing secondary extension.

Venous invasion by tumor cells, either with or without thrombosis and embolism, is more prominent earlier in the distant spread of the relatively small group of nonepithelial neoplasms, carcinoids, and melanoblastic tumors than in carcinomas. Except for malignant tumors that originate in the anus and, occasionally, in the distal rectum, tumor emboli are carried by tributaries of the portal vein to the sinusoidal network of the liver; they may lodge there sufficiently long to establish new lesions and then usually enter the systemic circulation and metastasize elsewhere. Tumor emboli from distally situated carcinomas and from the anus may originate from involvement of the anastomosing plexus surrounding the anus and may enter either the portal circulation (by way of the superior hemorrhoidal vein) or the systemic circulation through the middle and inferior hemorrhoidal veins. (Knowledge is incomplete concerning the number of cells in a tumor embolus—so-called critical cell mass that is necessary for the successful establishment of a metastasis.)

Involvement of the lymphatic system is of importance chiefly in the spread of carcinomas, carcinoids, and melanoblastic neoplasms and may occur by permeation, embolism, or both. The characteristics of the particular neoplasm influence the occurrence, incidence, and extent of lymphatic involvement. Neoplasms, such as leiomyosarcomas, are less likely to spread via the lymphatic system than are other tumors such as carcinoma and lymphosarcoma. Of the tumors that do spread via the lymphatic system, lymphosarcoma involves this system earlier than carcinoma; lymphosarcomas also involve the lymphatic system more commonly than carcinoma prior to complete intramural extension by direct continuous spread from the primary tumor. Although carcinomas almost invariably invade lymph channels eventually, with few exceptions, the degree of involvement depends on the completeness of the initial intramural extension. In carcinoids and melanoblastic tumors, lymphatic involvement is more variable and consequently less predictable, than in carcinoma. In both carcinoids and melanoblastic tumors, the incidence of early venous invasion is somewhat higher than in carcinoma.

Aberrations in the site of lymph node metastasis are related to such factors as anomalous pathways and retrograde lymph flow. In some instances, especially in areas of the hepatic flexure and of the sigmoid colon, the lymph flow by-passes the epicolic and the paracolic lymph nodes; it then proceeds directly to the intermediate or even to the principal nodes (Grinnell). Blockage of the main lymph channels by tumor or by antecedent inflammatory disease may divert the lymph flow in a retrograde route through paracolic nodes that are located distal to the primary neoplasm. In carcinoma involving the rectum and the anus especially, the possibility of retrograde lymph flow should be considered in determining the necessary extent of surgical procedures for eradication of the tumor. In my studies of rectal surgical specimens, retrograde lymphatic spread of carcinoma has been found in at least 5 percent of the cases, with caudal lymph node lesions sometimes several centimeters from the site of the tumor. For this reason, sphincter-preserving operations offer less than do more radical operations.

Usually, the lymph drainage follows normal anatomic pathways that parallel the arterial blood supply. Except for the efferent channels from the distal rectum and the anus, the lymph flow from both the small and the large intestines ultimately passes through the superior and the mesenteric lymph nodes prior to reaching the cysterna chyli and the thoracic duct.

Major subdivisions of the rectal lymphatic system are the extramural, the intramural, and the intermediate systems. The extramural lymphatic system has superior, lateral, and inferior collecting routes, that correspond to the superior, the middle, and the inferior hemorrhoidal vessels. The intramural lymphatic system consists of submucosal and intramuscular networks that freely communicate. The intermediate lymphatic system consists of a

subserous network of channels in the portion of the rectum that is covered by peritoneum; it also has a lymph sinus that is situated between the external layer of the muscularis propia and the perirectal fat in the portion of the rectum that is beneath the peritoneal reflexion. Efferent channels from the lymph sinuses and subserous lymph nodes communicate with the extramural lymphatics via the anorectal nodes of Gerota that are located in the hollow of the sacrum. Rectal carcinoma spreads into the intermuscular network (via radial channels) and through the intermediary lymphatic system more often than through the submucosal network (Coller et al., 1940). Both the intramural and the intermediate lymphatic systems drain into the extramural lymphatic system (Miles).

The lymphatic drainage of the anus differs from that of the remainder of the intestinal tract; this accounts for differences in the sites of metastasis, that is, in the inguinal lymph nodes rather than in the hypogastric, the iliac, and the preaortic nodes. In general, the anal lymph flow follows two main courses: (1) from the lower portion of the anal canal, it passes downward and forward across the perineum, then courses next to the vulva or the scrotum and the inner margins of the thighs to the superficial inguinal lymph nodes; (2) from the upper portion of the anal canal and the lower rectum, it follows the course of the inferior hemorrhoidal artery across the ischiorectal fossa and passes upward to the hypogastric, the common iliac, and the preaortic or lumbar nodes. The lymph reaches its destination by different routes that correspond chiefly to the distribution of the associated artery. Blockage of any portion of this lymphatic system may reverse the lymph flow and account for the variable patterns of lymph node metastasis.

Peritoneal dissemination is also a potential complication of malignant intestinal tumors whenever they penetrate the serosal coat of the intestinal wall or the visceral peritoneum that is contiguous to metastatic lesions in such structures as the mesentery, the mesocolon and the liver. As a mode of spread, peritoneal dissemination occurs often and is important in carcinoma of this area; it is found in about 20 percent of fatal cases and occasionally is accompanied by ascites.

Tumor spread that is secondary to implantation more often follows removal of a colonic carcinoma than of other malignant tumors of the intestines. It complicates about 15 percent of the surgical cases. In about two thirds of such cases, implantation occurs at the anastomotic suture line (Southwick et al.). Malignant cells have occasionally been found in the luminal contents as far from the primary growth as 35 cm. (McGrew et al., 1954, 1st and 2d references). Therefore, it is probable that the cells may have been implanted by the contamination of sutures or raw surfaces during operation. This local recurrence at the anastomotic site has been referred to as implantation metastasis (Vink).