

Synovial Sarcoma of the Upper Digestive Tract: A Report of Two Cases with Demonstration of the X;18 Translocation by Fluorescence *In Situ* Hybridization

Steven D. Billings, M.D., Lorraine F. Meisner, Ph.D., Oscar W. Cummings, M.D., Eduardo Tejada, M.D.
Department of Pathology and Laboratory Medicine, Indiana University School of Medicine (SDB, OWC), Indianapolis, Indiana; University of Wisconsin, Department of Pathology and Laboratory Medicine and the State Laboratory of Hygiene (LFM), Madison, Wisconsin; and Department of Pathology, Indiana University School of Medicine and the Laboratory Service, Richard L. Roudebush Veterans Affairs Medical Center (ET), Indianapolis, Indiana

Two cases of synovial sarcoma that arose in the upper digestive tract are reported. One case was a polypoid mass that arose at the gastroesophageal junction; the other was a large intramural mass that arose in the wall of the stomach. Both cases had a classic biphasic pattern. In the stomach tumor, the biphasic morphology was focal and there was an abrupt transition to poorly differentiated synovial sarcoma. The tumors had immunohistochemical features that were consistent with synovial sarcoma. Ultrastructural evaluation of the gastroesophageal tumor supported the diagnosis. The diagnostic X;18 translocation was demonstrated by fluorescence *in situ* hybridization on sections from paraffin-embedded tissue in 86% and 50% of interphase nuclei from the gastroesophageal and gastric tumor, respectively. The translocation was present in equal frequency in the epithelial and spindle cells in the biphasic areas and the poorly differentiated areas of the gastric tumor, indicating that the development of the more aggressive subclone was probably due to genetic mutations not encompassing the SYT-SSX gene fusion product. We are aware of only five reported cases of synovial sarcoma arising in the digestive tract, all in the proximal esophagus. These cases are the first reported arising in the gastroesophageal junction and stomach and the only cases of synovial sarcoma of the digestive tract in which the diagnostic translocation was demonstrated. Sarcomatoid carcinoma (carcinosarcoma) and gastrointestinal stromal tumor are the main

differential diagnoses for synovial sarcoma in this site. Synovial sarcoma of the digestive tract may be underdiagnosed, and its recognition may have important clinical implications. Fluorescence *in situ* hybridization is helpful in making this distinction.

KEY WORDS: Chromosomal translocation, Esophageal neoplasms, Fluorescence *in situ* hybridization, Immunohistochemistry, Stomach neoplasms, Synovial sarcoma.

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Synovial sarcomas are malignant mesenchymal tumors of uncertain histogenesis. They may be biphasic, monophasic, or poorly differentiated. They usually arise in the extremities intimately related to tendons and bursal structures of large joints but rarely in the synovium. Occasionally, they arise in the head and neck, most commonly as retropharyngeal or parapharyngeal masses (1).

We describe two cases of synovial sarcoma that arose in the upper digestive tract. One case originated at the gastroesophageal junction, and the other arose in the stomach. We are aware of five previously reported cases of primary synovial sarcoma of the cervical esophagus (2-6), but, to our knowledge, synovial sarcoma involving gastroesophageal junction or stomach has not been previously reported. Both tumors displayed histologic features of synovial sarcoma. Immunohistochemical staining profiles supported the diagnosis. Ultrastructural features of the gastroesophageal junction tumor were consistent with synovial sarcoma. However, because of the unusual locations, cytogenetic studies were performed to confirm the diagnoses. The characteristic X;18 translocation was demonstrated by using fluorescence *in situ* hybridization (FISH) studies on paraffin-embedded tissue.

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Address reprint requests to: Steven D. Billings, M.D., Department of Pathology and Laboratory Medicine, Indiana University Medical Center, University Hospital 3465, 550 North University Boulevard, Indianapolis, IN 46202-5280; fax: 317-274-5346.

MATERIALS AND METHODS

Formalin-fixed, paraffin-embedded tissue was sectioned at 4 μ m and stained with hematoxylin and eosin, and periodic-acid-Schiff with and without diastase predigestion according to standard methods.

For immunohistochemical examination, sections from both tumors were stained with monoclonal antibodies directed against epithelial membrane antigen (EMA; Ventana, Tucson, AZ), vimentin (Ventana), CD56 (Novocastra, Newcastle upon Tyne, UK), CD57 (Becton-Dickinson, San Jose, CA), and CD99 (O13; Signet, Dedham, MA). Monoclonal antibodies directed against cytokeratin AE1/3 (DAKO, Carpinteria, CA) and wide-spectrum antikeratin polyclonal antibodies (DAKO) were used on the gastroesophageal tumor. A cocktail of cytokeratin AE1/3 (DAKO) and CAM5.2 (DAKO) was used on the gastric tumor. Antibody dilutions were individually optimized (Table 1) for use with the Ventana automatic slide stainer (Ventana). Microwave pretreatment was used for antibodies directed against EMA, vimentin, CD56, CD57, CD99, and the AE1/3 and CAM 5.2 cocktail. Appropriate tissue served as positive controls. Negative controls were carried out with the Ventana Medical Systems negative control reagent, mouse monoclonal antibody Clone MOPC-21', which is not directed against a known epitope present in human tissue.

For ultrastructural evaluation, tissue was fixed in 3% glutaraldehyde, postfixed in osmium tetroxide, and stained with uranyl acetate and lead citrate. The specimen was thin sectioned and examined under a transmission electron microscope.

Cytogenetic evaluation was performed using FISH with dual color DNA probes, a green probe for the X centromere and a red probe for a region in the middle of the 18 long-arm (18q21). Hybridization with probes DXZ1 (Oncor, Gaithersburg, MD) labeled with fluoroisothiocyanate for the X centromere and digoxigenin-labeled 18qD18541 detected with rhodamine (Oncor) were performed as previously described (7). Briefly, this involved deparaffinization, digestion with pronase for 30 min, dena-

turation of specimen DNA in 70% formamide at 70° C, separate denaturation of probe DNA in a microwave oven for 40 seconds, and hybridization of probe and tumor DNA overnight in a 37° C humidified chamber according to the manufacturer's instructions. Slides from the gastroesophageal synovial sarcoma and an adenocarcinoma of the lung were processed together, and 100 cells from each were scored blindly. Slides from the gastric tumor were processed in the same fashion with an adenocarcinoma of the stomach as a control. If the X;18 translocation is present, one would expect a high frequency of spatial association between the X and 18 signals that would otherwise be randomly distributed within the nucleus. Because we are unaware of any report of an X;18 translocation in lung or gastric cancer, this tissue was used to control for the frequency of X and 18 signals that might be closely associated by chance. A close association was defined as the distance between a red and green signal less than the width of three centromere signals. Random associations were defined as signals more than or equal to three centromere widths. Using this methodology, we have previously found that the frequency of close associations in synovial sarcoma generally exceeded 75% compared with 20% or less of tumor nuclei lacking an X;18 translocation (7).

CASE REPORTS

Case 1

A 47-year-old Hispanic man had a history of persistent peptic ulcer disease and pyloric stenosis associated with gastric outlet obstruction. He had undergone repeated esophagogastroduodenoscopies with pyloric dilatations and standard medical therapy without significant improvement. As a result of the refractory nature of his peptic ulcer disease and gastric outlet obstruction, he underwent vagotomy, antrectomy, and a Billroth II procedure.

During surgery, a pedunculated, polypoid mass was found arising at the gastroesophageal junction. The mass was excised and submitted as a separate specimen. Histologic examination of the polypoid mass revealed a biphasic tumor consistent with synovial sarcoma. The tumor was focally present at the surgical margin. The patient underwent a subsequent completion gastrectomy with partial esophagectomy. Residual synovial sarcoma was not identified.

No adjuvant therapy was given. Follow-up evaluation including roentgenograms, computerized tomographic (CT) scans, and frequent esophagojejunostomy endoscopic procedures revealed no evidence of other primary site, metastasis, or recur-

TABLE 1. Summary of Antibody Dilutions and Results

Antibody	Dilution	Case 1	Case 2
Vimentin	1 : 100	E- /S+	E- /S+
CD56	1 : 20	E- /S+	E- /S+
CD57	1 : 10	E- /S-	E+ /S+ (focal)
CD99	1 : 10	E- /S-	E- /S-
EMA	1 : 100	E+ /S-	E+ /S-
AE1 /3	1 : 200	E+ /S focally +	not performed
Polyclonal Cytokeratin	1 : 9600	E+ /S focally +	not performed
AE1 /3 + CAM5.2 cocktail	1 : 40 (AE1 /3) 1 : 2 (CAM5.2)	not performed	E+ /S focally +

EMA, epithelial membrane antigen; E, epithelial cells; S, spindle cells.

rence. The patient has remained disease-free for 21 months.

Case 2

A 55-year-old woman had a 3-year history of abdominal swelling. Approximately 4 weeks before presentation, she developed dull right-side abdominal pain, nausea, vomiting, and rectal bleeding. The patient's medical history was noncontributory. An endoscopic biopsy revealed a poorly differentiated neoplasm interpreted as consistent with a malignant gastrointestinal stromal tumor (GIST). An abdominal CT scan demonstrated a large mass in the right upper quadrant between the gastric antrum, left lobe of the liver, duodenum, and pancreas. A chest CT scan demonstrated emphysematous change and a small peripheral nodule in the upper lobe of the right lung, which was too small to biopsy. No serum elevations of carcinoembryonic antigen, CA 19-9, CA 125, or alpha-fetoprotein were noted.

During laparotomy, a large mass arising from the distal stomach and adherent to the left lobe of the liver, gallbladder, and retroperitoneum overlying the pancreas was found. Multiple liver metastases were present. A hemigastrectomy with retrocolic Billroth II procedure was performed to debulk the tumor. No evidence of another primary site has been found. The patient died of disease 6 months after her resection.

RESULTS

Macroscopic Findings

The tumor at the gastroesophageal junction was a 5.2 × 2.2 × 1.3-cm, pedunculated, tan-brown, soft, smooth-surface mass. The gastric mass was a 16 × 15 × 7.5-cm spherical, intramural mass covered by a focally ulcerated gastric epithelium. On cut section, it had a fleshy, tan-pink appearance and large areas of necrosis.

Light Microscopic Findings

The gastroesophageal tumor showed the classic biphasic pattern of synovial sarcoma composed of epithelial cells in a sarcomatous spindle cell background (Fig. 1). The epithelial cells were arranged in discrete, simple to complex, gland-like structures and focally solid nests. The gastric tumor focally exhibited biphasic morphology (Fig. 2). The gland-like structures of both tumors contained eosinophilic, periodic-acid-Schiff-positive, diastase-resistant, amorphous material. The epithelial cells in both lesions had uniform, round to oval, vesicular nuclei with occasional, small nucleoli and relatively abundant eosinophilic cytoplasm. The spin-

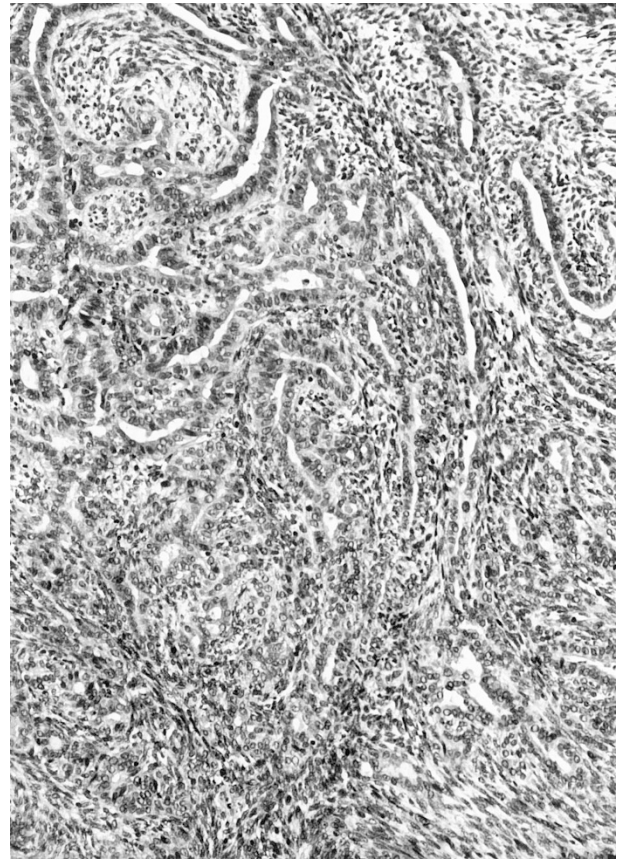


FIGURE 1. The gastroesophageal tumor was composed of well-demarcated, gland-like structures in a spindle cell background.

dle cell component of the biphasic region in both tumors varied from densely cellular to paucicellular to myxoid. The spindle cells had plump, fusiform nuclei with fine, granular chromatin, inconspicuous nucleoli, and lightly basophilic to eosinophilic cytoplasm. Scattered mast cells and neutrophils were present in the background. The majority of the gastric tumor was poorly differentiated, appearing as a high-grade sarcoma, with an abrupt transition from the biphasic to poorly differentiated areas in the tumor (Fig. 2). The poorly differentiated areas were predominantly composed of sheets of round to oval, hyperchromatic tumor cells admixed with some loose, myxoid areas. In the gastroesophageal tumor, the mitotic rate was low with an average of one mitotic figure per 10 high power fields. In the gastric tumor, the mitotic rate was variable, with an average of 9 mitotic figures per 10 high power fields in the biphasic portion of the tumor but greater than 50 mitotic figures per 10 high power fields in the poorly differentiated areas, including many atypical mitotic figures.

Immunohistochemistry

The immunohistochemical results are summarized in Table 1. The epithelial cells were positive for cyto-

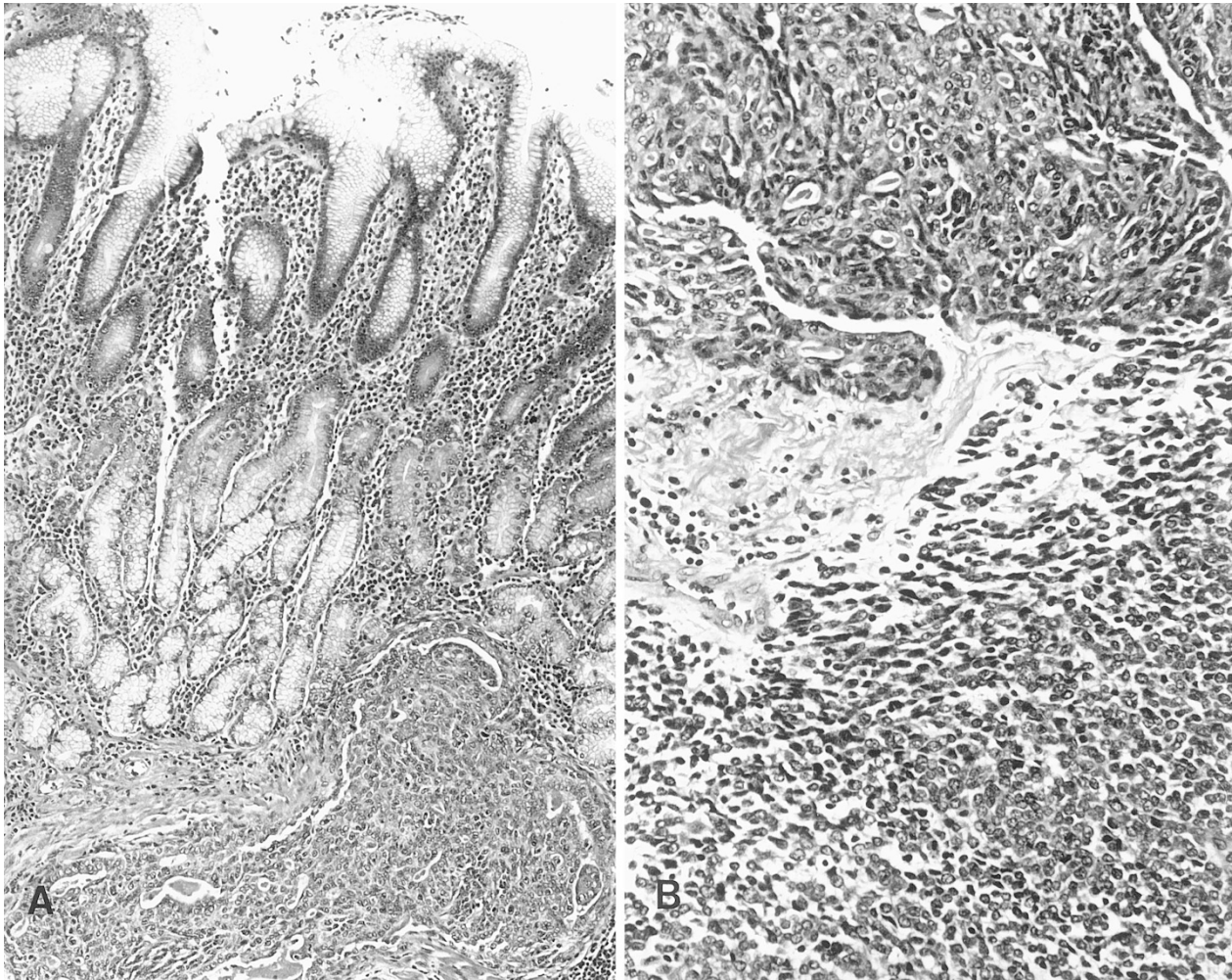


FIGURE 2. A, low power view of the gastric tumor demonstrating extension of the tumor into the superficial subepithelial stroma. B, there was an abrupt transition from the focal, biphasic area (*top*) to the predominant, poorly differentiated areas of the gastric tumor (*bottom*).

keratin (AE1/3, wide-spectrum polyclonal cytokeratin, or AE1/3 and CAM 5.2 cytokeratin cocktail), whereas the spindle cells showed only rare, focal staining. The glands exhibited luminal positivity for EMA. The spindle cells in both tumors were positive for vimentin and the neural crest adhesion molecule CD56, whereas the epithelial cells were negative; in the stomach lesion, the staining for vimentin was somewhat patchy. The gastric tumor was focally positive for CD57, whereas the gastroesophageal tumor was negative. Both tumors were negative for CD99 (O13). The poorly differentiated areas of the gastric tumor had a similar immunohistochemical staining profile as the spindle cell component of the biphasic area. The poorly differentiated component exhibited the same staining pattern for the mesenchymal and neural antigens but failed to exhibit the focal staining for cytokeratin seen in the spindle cell component of the biphasic portion of the tumor.

Ultrastructural Examination

Ultrastructural examination was performed on the gastroesophageal tumor. Electron micrographs

showed a distinct, biphasic cell population with epithelial-like cells forming glandular, tubular, or solid structures and elongated stromal cells (Fig. 3). The epithelial-stromal junction was composed of a discrete electron-dense basal lamina. The nuclei in the epithelial cells tended to be round to oval, and the nuclei in the stromal cells tended to be more spindle shaped. Both were irregularly shaped with coarse, clumped, marginated chromatin. The cytoplasm of the epithelial cells had tubular mitochondria, Golgi apparatus, lysosomal bodies, and lipid vacuoles. Junctional complexes were present between adjoining cells. The luminal surface had microvilli and focal cytoplasmic filaments. No basal lamina or cellular attachment was noted between stromal cells, which appeared surrounded by fibrillar ground substance as well as mature collagen.

Cytogenetic Results with FISH

Approximately two thirds of the cells could not be scored because of overlapping, truncation, or unclear signals. In the scorable cells, fluorescent sig-

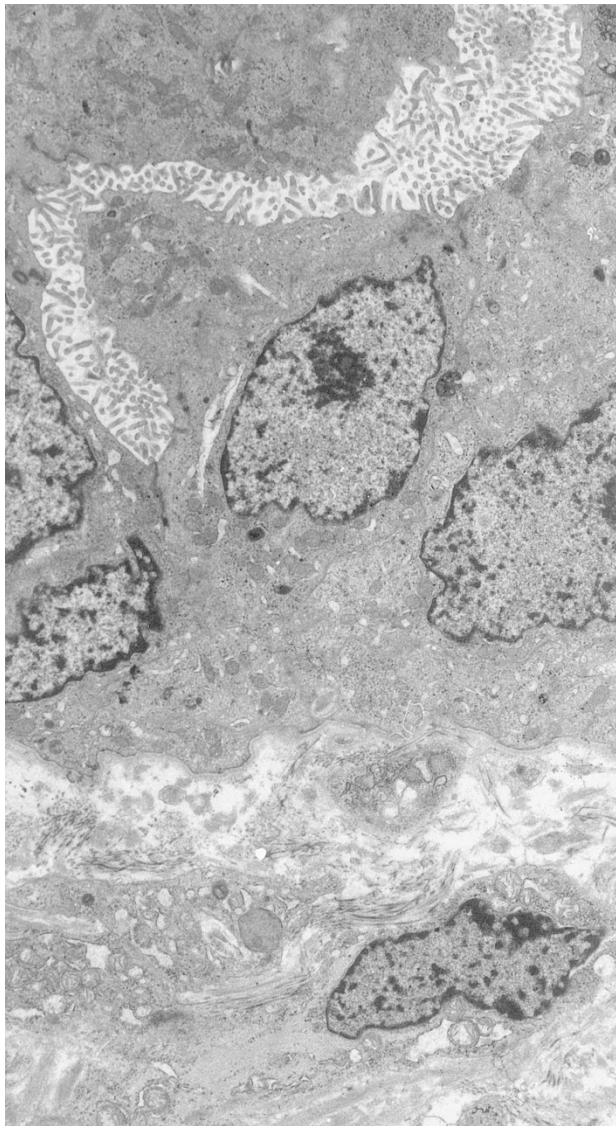


FIGURE 3. Ultrastructural examination of the gastroesophageal tumor demonstrated a distinct biphasic cell population separated by a discrete electron dense basal lamina. The epithelial cells have numerous microvilli projecting into a luminal space (magnification, 4950 \times).

nals for the X centromere and the 18q21 region were found to be closely associated in 86% and 50% of the interphase nuclei in the gastroesophageal and gastric tumor, respectively, indicating the presence of the X;18 translocation (Fig. 4). In the gastroesophageal tumor, the epithelial and spindle cells demonstrated the translocation with equal frequency. Similarly, the translocation was present with equal frequency in both components of the biphasic portion of the gastric tumor as well as in the poorly differentiated area. The control slide from the adenocarcinoma of the lung demonstrated a close association in 22% of tumor nuclei. This is somewhat higher than expected. However, the lung tumor had four X chromosomes, thereby increasing the chance of random close associations

of chromosomes X and 18. The control slide from the adenocarcinoma of the stomach demonstrated close associations of the X and 18 chromosomes in 2% of the cells.

DISCUSSION

Primary sarcomas of the esophagus and stomach are rare. Fewer than 2% of primary esophageal tumors are mesenchymal in origin (8); the majority of esophageal sarcomas are leiomyosarcoma (9). In the stomach, the majority of malignant mesenchymal tumors include malignant gastric stromal tumors and leiomyosarcoma. To our knowledge, there have been only five previous case reports of synovial sarcomas arising in the digestive tract, all of which involved the cervical or proximal esophagus (Table 2). Similar to our gastroesophageal junction case, these lesions all were exophytic masses exhibiting classic biphasic histology. We are unaware of any previously reported case of synovial sarcoma arising in the stomach. Our cases are the first involving the digestive tract in which the characteristic X;18 translocation was identified.

The primary differential diagnosis of biphasic synovial sarcoma in the digestive tract is sarcomatoid carcinoma (carcinosarcoma). The age ranges for both tumors overlap; however, sarcomatoid carcinoma tends to present at an older age (10, 11) than synovial sarcoma (1). Sarcomatoid carcinomas of the esophagus (11–16) and stomach (12, 17–19) usually present as polypoid masses with a similar gross appearance as our tumor from the gastroesophageal junction. Gastric sarcomatoid carcinomas also frequently present as intramural masses (12), grossly indistinguishable from our case arising in the stomach. Carcinosarcomas of the esophagus are most common in the middle third of the esophagus (11), whereas gastric sarcomatoid carcinomas have a predilection for the pyloric stomach (12), although they have been reported in a variety of sites within the respective organs. Previously reported cases of esophageal synovial sarcomas have been restricted to the proximal esophagus (2–6).

The sarcomatous elements in esophageal or gastric sarcomatoid carcinoma may have a variety of histologic features. Typically, they have been undifferentiated spindle cells similar in appearance to synovial sarcoma, but overt mesenchymal differentiation showing smooth muscle, cartilage, or bone formation has been described (12, 17, 19, 20). Mesenchymal differentiation can occur in synovial sarcoma as well.

Most esophageal sarcomatoid carcinomas have a squamous epithelial component (6, 11, 12, 14, 20–22), although in rare cases adenocarcinoma or adenoid cystic carcinoma occurs (20). The lack of

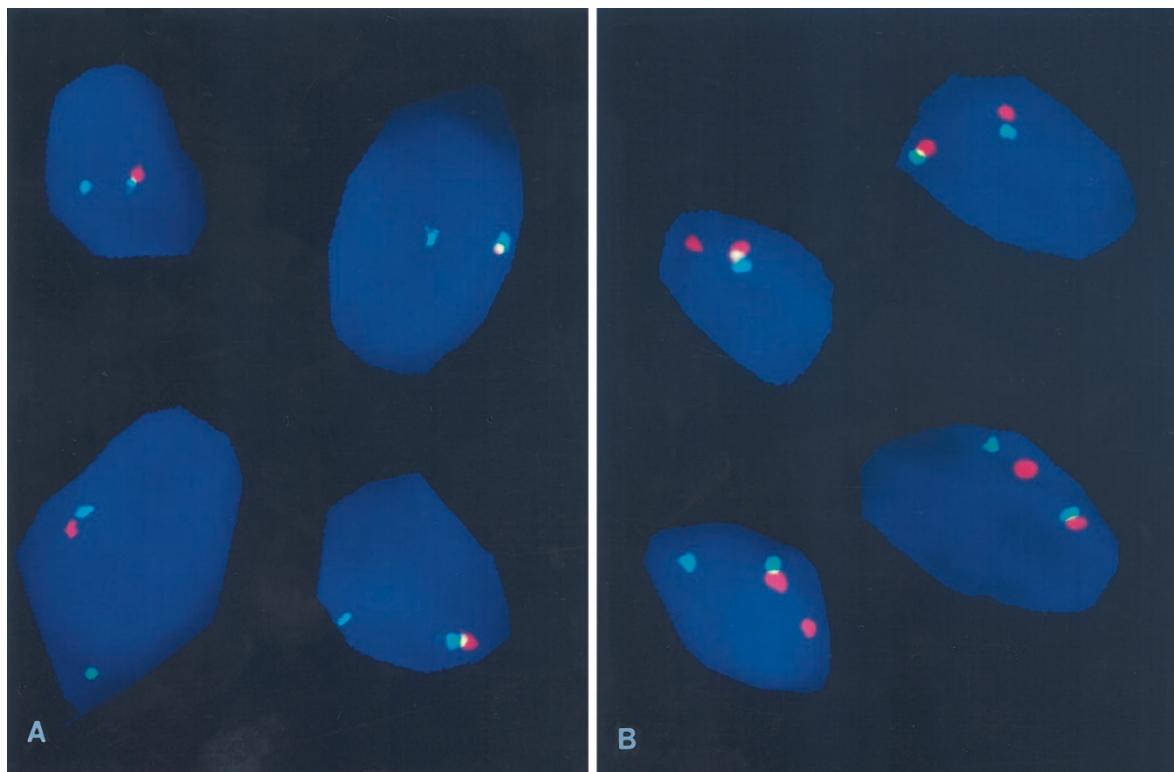


FIGURE 4. Typical appearance of the red X and green 18 probes characterizing the diagnostic t(X;18) in four separate synovial sarcoma nuclei from the gastroesophageal tumor (A) and the gastric tumor (B). The translocation is characterized by the red and green signals touching or lying less than three centromere lengths to each other (*lower left corner of A*) in the interphase nuclei. The second green 18 signal is randomly located in the nuclei. The second red X signal is randomly located in the gastric tumor, but there is no second red X signal in the gastroesophageal tumor because the patient is male. This pattern is consistent with the presence of the X;18 translocation.

TABLE 2. Summary of Reported Cases of Digestive Tract Synovial Sarcoma

Author (reference)	Year	Age	Gender	Location	Size (cm)	Gross features	Microscopic features	Follow-up
Palmer et al. (6)	1983	75	Female	Upper 1/3 esophagus	2.5	Papillary	Biphasic	DOD, 2 yr
Amr et al.(2)	1984	25	Male	Upper 1/3 esophagus	5 × 3 × 1.5	Polypoid	Biphasic	AWOD, 36 mo
Bloch et al. (4)	1987	15	Male	Cervical esophagus	10 × 6 × 3	Polypoid	Biphasic	AWOD, 36 mo
Antón-Pacheco et al. (3)	1996	14	Female	Cervical esophagus	7 × 6 × 6	Polypoid	Biphasic	AWOD, 30 mo
Habu et al. (5)	1998	20	Male	Upper esophagus	8.0 × 3.0	Polypoid	Biphasic	AWOD, 20 mo
Current Case #1	1999	47	Male	Gastroesophageal junction	5.2 × 2.2 × 1.3	Polypoid	Biphasic	AWOD, 21 mo
Current Case #2	1999	55	Female	Stomach	16 × 15 × 7.5	Spherical, intramural	Biphasic and poorly differentiated	DOD, 6 mo

DOD, died of disease; AWOD, alive without evidence of disease; AWD, alive with residual disease

squamous carcinoma in our case makes the diagnosis of esophageal sarcomatoid carcinoma less likely. In gastric sarcomatoid carcinoma, adenocarcinoma is the predominant epithelial component (9, 12, 17–19). The epithelial component can vary from well differentiated to poorly differentiated, including signet ring morphology (9, 12), but well differentiated adenocarcinoma is more typical (10). The neoplastic glands can blend with the stromal component or be well demarcated from it; similar patterns are seen in biphasic synovial sarcoma. Differentiating between synovial sarcoma and gastric

sarcomatoid carcinoma is therefore more problematic. In our opinion, the epithelial cells of our cases were somewhat more bland with more well-demarcated and complex glandular structures than in typical gastric sarcomatoid carcinoma. However, the histologic features of sarcomatoid carcinoma, particularly gastric tumors, can be strikingly similar.

The differential diagnosis of monophasic fibrous or poorly differentiated synovial sarcomas of the digestive tract includes benign and malignant GIST and leiomyosarcoma. In fact, the stomach lesion

was originally interpreted as consistent with a malignant GIST on the basis of a biopsy that showed only the poorly differentiated areas of the tumor and none of the biphasic component. The presence of the biphasic area caused us to question the original diagnosis.

Though synovial sarcoma can have a distinctive appearance, establishing a definitive diagnosis may require ancillary studies, especially in tumors that arise in unusual locations. By immunohistochemistry, the epithelial and mesenchymal elements of sarcomatoid carcinoma and synovial sarcoma can have essentially the same pattern of reactivity with antibodies directed against epithelial markers and vimentin. However, synovial sarcoma often expresses neuroectodermal antigens (23–25). CD99 expression has been demonstrated in 46 to 100% of synovial sarcomas (23). Folpe *et al.* (24) described immunoreactivity for CD56, the neural crest adhesion molecule, and CD57, a marker of nerve sheath differentiation, in 8 of 8 and 9 of 10 poorly differentiated synovial sarcomas, respectively. Immunostains for CD56 were positive in the poorly differentiated part of the gastric tumor and selectively marked the spindle cell component in the biphasic components of both tumors, as previously reported in biphasic synovial sarcoma (25). The gastric tumor showed focal positivity for CD57, whereas the gastroesophageal junction tumor was negative. Both were negative for CD99. Theoretically, a sarcomatoid carcinoma with neural differentiation could display a similar immunohistochemical profile, although we are not aware of a case of gastric or esophageal sarcomatoid carcinoma that expressed these neural antigens. Given the similar immunostaining profile of the spindle cell and poorly differentiated component of the gastric tumor, we interpreted the poorly differentiated area as evolving from the less prominent biphasic component. In more equivocal cases in which GIST would still be a consideration in the differential diagnosis, immunohistochemistry may be of use as GISTs are usually positive for CD34, unlike synovial sarcoma (26). The stomach tumor was negative for smooth muscle actin and desmin (data not shown).

The ultrastructural features support the diagnosis of synovial sarcoma but are not specific. Ultrastructural examination of esophageal (14) and gastric sarcomatoid carcinoma (9, 17, 18) has shown similar features as found in synovial sarcoma (1).

Because of the unusual locations of the tumors, we decided to pursue cytogenetic confirmation. FISH conclusively established the presence of the X;18 translocation, a sensitive marker demonstrated in 70 to 90% of synovial sarcomas (27–29). The specific t(X;18)(p11.2;q11.2) results in the fusion gene product *SYT-SSX* (30). Only rare nonsynovial sarcomas have exhibited an X;18 translocati-

tion, including a single case of fibrosarcoma (31), a case of malignant fibrohistiocytoma (32), a case of poorly differentiated sarcoma not otherwise specified (32), and four cases of malignant peripheral nerve sheath tumor (33). This translocation has not, to our knowledge, been described in carcinosarcomas of the esophagus or stomach. Loss of heterozygosity at different loci has been described in esophageal carcinosarcoma, but there was no evidence of an X;18 translocation (13). Because of its relative specificity, the X;18 translocation may prove useful in the diagnosis of synovial sarcoma in unusual locations. In the gastric synovial sarcoma, the percentage of cells with the translocation was slightly less than expected (50%) but still significantly more than would be expected by chance. The poorly differentiated portion of the gastric tumor demonstrated the translocation in the same proportion as the biphasic areas. Therefore, the development of the more aggressive subclone is likely due to additional genetic mutations not encompassing the gene fusion product of the translocation. It would be interesting to investigate whether this finding holds true for other cases of synovial sarcoma that exhibit poorly differentiated areas admixed with more typical biphasic and monophasic morphology.

The clinicopathologic characteristics of our cases and the five previously reported cases of synovial sarcoma arising in the esophagus are summarized in Table 2. All of the tumors involving the esophagus had an exophytic appearance variably described as papillary or polypoid. Our case arising in the stomach was intramural and spherical. Reported cases of digestive tract synovial sarcoma have a broad age range (14 to 75 years); the majority of the tumors occurred in younger individuals. Of the five patients with esophageal tumors, four were alive without disease after resection, with follow-up ranging from 20 to 48 months; one patient died of disease 2 years after diagnosis. The patient with the gastric synovial sarcoma died of disease 6 months after resection. The follow-up from previous reports and our case indicates that esophageal or gastroesophageal synovial sarcomas may have a better prognosis than synovial sarcoma that arises elsewhere. Symptoms from luminal obstruction could result in earlier detection, or other unknown factors may play a role. Theoretically, the opposite may be true for tumors that arise in the stomach because they could grow to large size before obstructive or other mass-related symptoms occur. Clinical follow-up has been limited, and synovial sarcoma is widely known to recur or metastasize after long disease-free intervals. Illustrative of this point is the 5- and 10-year overall survival of 60% and 34%, respectively, for synovial sarcoma (34). Singer *et al.* (34) found that synovial sarcomas smaller than 5

cm had 100% 10-year survival. It is interesting that the only reported death as a result of esophageal synovial sarcoma occurred with a tumor 2.5 cm in greatest dimension (6). This suggests that synovial sarcoma may behave differently in different sites. The 5-year survival rate of esophageal sarcomatoid carcinoma ranges from approximately 27 to 50% (11, 12), which is significantly worse than synovial sarcoma. Gastric sarcomatoid carcinoma has a much worse prognosis, approaching a mortality of 100% at 5 years (12). Distinguishing synovial sarcoma from sarcomatoid carcinoma at the gastroesophageal junction or stomach may therefore have important clinical implications in terms of patient prognosis. A retrospective cytogenetic examination of previously diagnosed sarcomatoid carcinomas to identify unrecognized synovial sarcomas might help clarify the prognostic implications. The distinction has therapeutic implications as well. Synovial sarcoma may respond to adjuvant chemotherapy with regimens including high-dose ifosfamide (35, 36).

Synovial sarcoma of the digestive tract is possibly an underdiagnosed condition. Only a handful of cases have been reported, yet we have seen two cases in the past 2 years. Some cases may have been previously diagnosed as sarcomatoid carcinoma or GIST. As cytogenetic and molecular techniques become more accessible, the accurate diagnosis of synovial sarcoma that arises in the digestive tract may increase.

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