Gastrointestinal stromal tumors in western Saudi Arabia

Rana Y. Bokhary, MD, FRCPC, *Jaudah A. Al-Maghrabi,* MD, FRCPC.

ABSTRACT

الأهداف: تحديد نمط أورام الجهاز الهضمي النسيجية (GIST) في مرضى مركزين من المراكز الطبية الكبيرة في المنطقة الغربية من المملكة العربية السعودية.

الطريقة: استخدمت دراسة استعادية للحالات التي تم تشخيصها كأورام الجهاز الهضمي النسيجية GIST في مختبرات التشريح النسيجية في مستشفى جامعة الملك عبد العزيز خلال الفترة من يناير 2000م إلى أكتوبر 2009م و مستشفى الملك فيصل التخصصي ومركز الأبحاث خلال الفترة من يناير 2002م إلى ديسمبر 2008م. بالإضافة إلى الحالات التي تم تشخيصها في الجهاز الهضمي كأورام الخلايا المغزلية الحميدة والخبيثة، وأورام خلايا شوان، و أورام العضلات الملساء الحميدة والخبيثة في مستشفى جامعة الملك عبد العزيز قبل إدخال الصبغة المناعية CD117 في المختبر لتشخيص أورام الجهاز الهضمي النسيجية من الفترة يناير 1995م إلى ديسمبر 1999م. أدرجت الحالات الإيجابية فقط في الدراسة. تم تحليل عمر وجنس المرضى المصابين بهذه الأورام، و كذلك موقع حدوث الورم في الجهاز الهضمي، و قطر الورم الأكبر، و عدد الخلايا المنقسمة.

النتائج: وجدت 37 حالة من أورام الجهاز الهضمي النسيجية حيث متوسط العمر للحالات هو 55.6 عام. وكانت 19 حالة في المعدة (51.4%)، و 7 حالة في الأمعاء الدقيقة (18.9%)، و 2 حالة في القولون (5.4%)، و 4 حالة في المساريق المغلفة للأمعاء (10.8%)، و 5 حالة في البطن (13.5%). تقع 15 حالة (40.5%) في المجموعة المرتفعة المخاطر، و 13 حالة (35.2%) في مجموعة المخاطر المتوسطة، و 3 حالة (8.1%) في المجموعة المنخفضة المخاطر، و حالة واحدة (2.7%) في المجموعة المنخفضة المخاطر جدا للسلوك العدواني.

خاتمة: أن أورام الجهاز الهضمي النسيجية GIST منتشرة بكثرة في المملكة العربية السعودية مما يعتقد عموما. حدثت غالبية الحالات في الذكور البالغين في العمر أكثر من 40 عام. و تعد المعدة المكان الأكثر شيوعا للحدوث. تقع الغالبية العظمى من الأورام المشخصة في المجموعة المرتفعة المخاطر للسلوك العدواني.

Objectives: To determine the pattern of gastrointestinal stromal tumors (GIST) in the population of 2 large tertiary centers in the western province of the Kingdom of Saudi Arabia (KSA).

Methods: This retrospective study used cases diagnosed as GIST at the histopathology laboratories of King Abdul-Aziz University Hospital between January 2000 and October 2009, and King Faisal Specialist Hospital and Research Center, Jeddah, KSA, between January 2002 and December 2008. Additionally, cases diagnosed as gastrointestinal spindle cell tumors, sarcomas, schwannomas, leiomyomas, and leiomyosarcomas at King Abdul-Aziz University Hospital between January 1995 and December 1999 prior to the introduction of CD117 immunostain testing in the lab were tested for it. Positive cases were included in the study. Age and gender of the patients, as well as tumor location, maximum diameter, and mitotic count were analyzed.

Results: Thirty-seven cases were found in which the mean age was 55.6 years. Nineteen tumors were located in the stomach (51.4%), 7 in the small bowel (18.9%), 2 in the colorectum (5.4%), 4 in the mesentery (10.8%), and 5 in the abdomen (13.5%). Fifteen cases (40.5%) were high risk, 13 (35.2%) were intermediate risk, 3 (8.1%) were low risk, and one case (2.7%) was very low risk for aggressive behavior.

Conclusion: The GISTs are more prevalent in Saudi Arabia than is generally thought. Most cases occurred in male adults over 40 years of age. The stomach is the most frequent location of occurrence. Most tumors are of the high-risk group.

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From the Department of Pathology (Bokhary, Al-Maghrabi), Faculty of Medicine, King Abdul-Aziz University Hospital, and the Department of Pathology and Laboratory Medicine (Al-Maghrabi), King Faisal Specialist Hospital and Research Center, Jeddah, Kingdom of Saudi Arabia.

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Address correspondence and reprint request to: Dr. Rana Y. M. Bokhary, Assistant Professor and Consultant Pathologist, Department of Pathology, Faculty of Medicine, King Abdul-Aziz University Hospital, PO Box 80205, Jeddah 21589, Kingdom of Saudi Arabia. Tel. +966 (2) 6401000 Ext. 17084. Fax. +966 (2) 6408433. E-mail: bokharyr@hotmail.com

Gastrointestinal stromal tumors (GISTs) are the most common mesenchymal neoplasms of the human gastrointestinal (GI) tract. They account for approximately 0.1-3% of all GI tumors with an approximate annual incidence of 0.68-1.45 cases per 100,000.[1-5] Prior to the year 2000, these tumors were diagnosed as leiomyomas, leiomyosarcomas, leiomyoblastomas, or schwannomas, based predominantly on their histological features. Currently, GISTs are distinguished immunohistochemically from these morphologically similar neoplasms of the gastrointestinal tract. It has been shown that positive immunohistochemical staining for CD117 enables the identification of GISTs.[6] The GISTs occur predominantly in people over the age of 40, with a small male predominance. However, GIST cases have also been reported in children.[1-5,7-9] The stomach is the most common location of involvement, followed by the small intestine.[1-5] There are rare reported cases in the gallbladder, pancreas, liver, and urinary bladder. These tumors arising outside the GI tract are known as extra-gastrointestinal stromal tumors (EGISTs).[10-13] The biological behavior of GISTs is complex and unpredictable. Tumor size and mitotic counts are considered to be the most important parameters for predicting the clinical course and chance of recurrence.[14,15] A consensus conference held at the U.S. National Institutes of Health (NIH) in 2001 provided a practical scheme for the assessment of risk of this disease. Their risk categorization is based on the evaluation of the size (largest dimension in cm) and mitotic rate (number of mitotic figures per 50 high power fields) of the tumors.[16] More recently, a large number of clinicopathologic studies from the Armed Forces Institute of Pathology have added the dimension of assessing these tumors in a site-specific manner. Their criteria still rely on the assessment of tumor size and mitotic activity, but they differ between the different locations of the tumor.[17,18] The pattern of GISTs in Saudi Arabia has not been well studied and only 4 cases of gastric GISTs were mentioned in the 2005 cancer incidence report issued by the Saudi Cancer Registry of the Ministry of Health.[19] Three of these affected individuals were males while the fourth was a female. The GISTs in other locations were not mentioned specifically in the report, and these are most probably included in the "other" category of tumors in the colorectum. According to our experience, we suspect this figure to be low, which could be due to under-reporting of the newly diagnosed cases to the Ministry. We conducted this retrospective study to provide us with an idea of the features of GIST in the population of the Western Province of Saudi Arabia, and to compare our area with the rest of the world.

Methods. The cases of this retrospective study were drawn from the Pathology Laboratories' archives of King Abdul-Aziz University Hospital (KAUH), and King Faisal Specialist Hospital and Research Center (KFSHRC), Jeddah, Saudi Arabia, which are considered 2 of the largest tertiary centers in the Western Province of the country. As the first case diagnosed as GIST in KAUH Pathology Laboratory was performed in 2000, the digital archives of KAUH were searched for cases diagnosed as GI sarcomas, leiomyomas, leiomyosarcomas, leiomyoblastomas, smooth muscle tumors or spindle cell tumors prior to that date (January 1995 to December 1999). The archives were also searched for cases diagnosed as GIST from January 2000 to October 2009. The cases from KFSHRC comprise those diagnosed as GIST from January 2002 to December 2008. The CD117 "c-kit" immunostain (Dako, Glostrup, Denmark, c-kit Rabbit, antihuman polyclonal antibody, 1:50) was performed on the cases diagnosed in KAUH as GI sarcoma, leiomyoma, leiomyosarcoma, leiomyoblastoma, smooth muscle tumor, or spindle cell tumor. They were included in the study as GISTs only when CD117 immunostain positivity was obtained. For the cases originally diagnosed as GIST in both centers, the reports and slides were checked for the presence of positive CD117 immunostain. The reports of all the positive cases were reviewed for the age and gender of the patients as well as for the location and the maximum diameter (in centimeters) of the tumor and their mitotic count. The slides of the cases in which the mitotic count was not mentioned in the report were re-examined microscopically and mitosis was counted in 50 high power fields. The cases were subclassified according to the 2001 NIH Workshop criteria for risk assessment into 4 risk categories (very low, low, intermediate, and high risk for aggressive behavior).[7] Simple descriptive statistics were used to analyze the data. Ethical approval for this study was obtained from the Bioethical and Research Committee of the Faculty of Medicine of KAUH.

Results. A total of 37 GIST cases were found, 18 cases from KFSHRC, and 19 cases from KAUH. Among the KAUH cases, 14 cases were diagnosed as GIST (after 2000), while the other 5 cases were originally diagnosed as smooth muscle tumor of uncertain malignant potential (one case), leiomyoma (2 cases), and leiomyosarcoma (2 cases). Of the 37 cases, 12 cases occurred in females and 25 occurred in males with a ratio of 1:2.1. Age ranged between 31-78 years (mean 55.6 years). No cases diagnosed in children were found. **Table 1** summarizes GIST features, including location, size, and risk category. The risk for 5 cases (13.5%)

Table 1 - Features of gastrointestinal stromal tumors in the studied population (N=37).

Feature	Number of patients
Gender	
Males	25
Female	12
Age (years)	31-78
Location, n (%)	
Stomach	19 (51.4)
Small bowel	7 (18.9)
Colorectum	2 (5.4)
Mesentery	4 (10.8)
Abdomen	5 (13.5)
Size (cm)	1.2-30
Risk category, n (%)	
Very low	1 (2.7)
Low	3 (8.1)
Intermediate	13 (35.2)
High	15 (40.5)

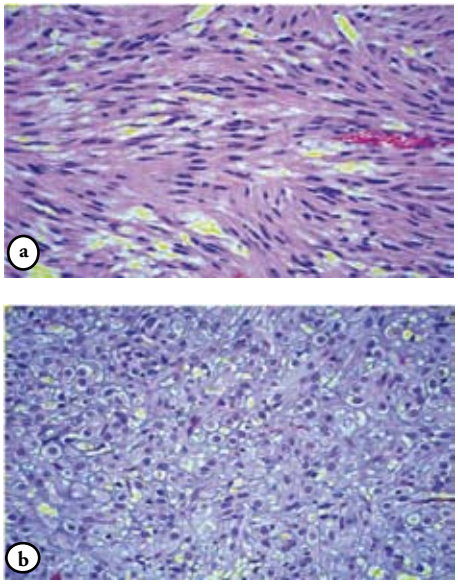

Figure 1 - Gastrointestinal stromal tumors are composed of either **a**) spindle cells arranged in fascicles (Hematoxylin & Eosin [H & E] stain, 40x), **b**) epithelioid cells (H & E stain, 40x), or a combination of both.

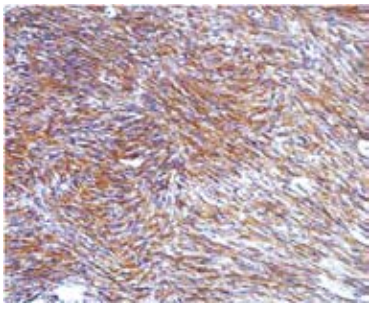

Figure 2 - The CD117 (c-kit) immunostain is a sensitive maker for gastrointestinal stromal tumors (20x).

could not be evaluated because we only reviewed biopsies from these patients and no resection specimens for these cases could be found in both institutes. We found no correlation between the ages of the patient and the aggressive behavior potential of the tumors as the mean age of the patients in the high-risk group was 54.7, 57.9 in the intermediate risk group, 47 in the low risk group, and 51 in the very low risk group.

Discussion. Although GISTs are the most common GI mesenchymal neoplasms, detailed studies from Saudi Arabia were not found on literature review. The importance of studying this tumor lies in its unique line of targeted therapy, which depends on its pathogenesis. Currently, GISTs are defined as pleomorphic mesenchymal tumors of the GI tract that express the KIT protein (CD117- protooncogene that encodes the transmembrane tyrosine kinase receptor CD117) and often also CD34 on immunohistochemistry.[16] It is known that many GIST tumors have an activating mutation in either KIT or PDGFRα (Platelet-Derived Growth Factor Receptor Alpha) tyrosine kinase receptor.[20] This has led to the introduction of Imatinib mesylate, a tyrosine kinase inhibitor for KIT, for treatment of affected patients.

The mean age of patients in our study and the male predominance were similar to the figures published in the literature. Most of our cases occurred also in the stomach, and we found no extra-gastrointestinal cases. Most of the cases are high-risk for aggressive behavior according to the applied 2001 NIH Workshop criteria for risk assessment.

The GISTs vary greatly in size from a few millimeters to >30 cm, with a median size between 5-8 cm. Macroscopically, they arise from the gut wall (intramural) and may protrude from the serosal surface or intraluminally. Large tumors may ulcerate the overlying mucosa (in 20-30% of cases). Other tumors that protrude extensively from the serosal surface may extend directly into the pancreas or liver, obscuring its gastrointestinal origin. Their cut surface is typically tan and variably fibrous to fleshy. They are generally well circumscribed, usually with a pseudo-capsule. Large tumors may undergo significant necrosis and cystic degeneration at their centers, with only a residual rim of viable tissue at the periphery. Complex cystic masses and multinodular peritoneal seeding are characteristics of malignant GISTs.[21] The GISTs have many different histological features that may differ with location (**Figure 1**). Gastric GISTs are composed of spindle cells in 50% of cases, epithelioid cells in 30%, and are mixed in 20%. The mixed pattern may show sharp demarcation between spindled and epithelioid areas.[16,17,22] Most small intestinal GISTs are composed of spindle cells.[16] Colonic

and anorectal GISTs are found to be morphologically similar to small intestinal GISTs, while esophageal tumors and EGISTS resemble gastric ones.[23]

The CD117 (c-kit) is the most sensitive marker of GISTs from all sites, as it is expressed in more than 95% of cases.[24] The pattern of staining is usually strong, diffuse, and pancytoplasmic, in 90% of tumor cells (**Figure 2**). Immunoreactivity may be patchy sometimes; therefore, false-negative staining can be seen in small biopsy specimens. Up to 5% of cases are CD117 negative. Many of these CD117-negative tumors have PDGFRA mutations.[25] In addition to CD117, around 70% of GISTs diffusely express CD34. Approximately 30% of GISTs express muscle markers, including smooth muscle actin, calponin, and h-caldesmon. However, desmin is only very rarely expressed. Focal nuclear and cytoplasmic positivity for S100 protein occurs in 5-10%.[16] The Wilm's tumor gene protein (WT)-1 and calretinin appear to be sensitive for GISTs, with the added advantage of being positive in many KIT-negative examples.[26] Other immunostains that have been studied in CD117-negative GISTs include Nestin, PDGFRA, protein kinase theta and DOG1, but none of these are used routinely in workup of GIST cases.[27-29]

Because of their highly variable clinical behavior, all GISTs should be considered to be of at least low malignant potential even when they appear histologically benign. Many potential prognostic indicators have been studied for these tumors, including site, size, histomorphology, immunohistochemistry, and molecular genetics.[14,15] The NIH held a GIST workshop in April of 2001, with the goal of developing a consensus approach to diagnosis and prognostication on the basis of morphologic features.[16] They classified these tumors as having very low risk, low risk, intermediate risk, or high risk for aggressive behavior, based on the tumor size (single largest dimension in cm) and mitotic count (number of mitotic figures per 50 HPF). This approach turned out to be reasonably reproducible because it relies on 2 relatively simple objective parameters. However, it did not account for the differences among GISTs in different locations. Therefore, recently, the Armed Forces Institute of Pathology developed criteria that still rely on the assessment of tumor size and mitotic activity, but these differ between the different locations of the tumor.[17,18]

The treatment of choice for primary GISTs remains complete surgical resection with negative margins. As chemotherapy and radiotherapy are useless in their treatment, targeted therapy with Imatinib mesylate is used. It is a non-selective tyrosine kinase inhibitor that has been approved by the U.S. Food and Drug Administration for the treatment of unresectable and metastatic GISTs.[30] Recent studies suggest that even tumors with PDGFRA exon 12 and exon 18 mutations and no KIT mutation are also sensitive to Imatinib.[31] Newer drugs such as SU11248 (Sunitinib malate) are developed and used as second-line therapy after Imatinib failure or in case of Imatinib intolerance.[32-34]

As our study is limited only to 2 institutions, it does not reflect the real pattern of GIST in the whole country, and more studies from other areas of the Kingdom are required.

The pattern of GISTs in Saudi Arabia shows epidemiological results similar to the universal data. More effective reporting of cases to the Cancer Registry of the Ministry of Health should be encouraged by all hospitals in the Kingdom in order for their incidence report to be more reflective of the incidence and prevalence of the disease. Other studies, preferably prospective, are required to show us the effectiveness of treatment and prognosis of GISTs in our population.

References

1. Kim KM, Kang DW, Moon WS, Park JB, Park CK, Sohn JH, et al. Gastrointestinal stromal tumors in Koreans: it's incidence and the clinical, pathologic and immunohistochemical findings. ***J Korean Med Sci*** 2005; 20: 977-984.
2. Tryggvason G, Gíslason HG, Magnússon MK, Jónasson JG. Gastrointestinal stromal tumors in Iceland, 1990-2003: the icelandic GIST study, a population-based incidence and pathologic risk stratification study. ***Int J Cancer*** 2005; 117: 289-293.
3. Goettsch WG, Bos SD, Breekveldt-Postma N, Casparie M, Herings RM, Hogendoorn PC. Incidence of gastrointestinal stromal tumours is underestimated: results of a nation-wide study. ***Eur J Cancer*** 2005; 41: 2868-2872.
4. Nilsson B, Bümming P, Meis-Kindblom JM, Odén A, Dortok A, Gustavsson B, et al. Gastrointestinal stromal tumors: the incidence, prevalence, clinical course, and prognostication in the preimatinib mesylate era--a population-based study in western Sweden. ***Cancer*** 2005; 103: 821-829.
5. Monges G, Bisot-Locard S, Blay JY, Bouvier AM, Urbieta M, Coindre JM, et al. The estimated incidence of gastrointestinal stromal tumors in France. Results of PROGIST study conducted among pathologists. ***Bull Cancer*** 2010. [Epub ahead of print].
6. Miettinen M, Lasota J. Gastrointestinal stromal tumors: review on morphology, molecular pathology, prognosis, and differential diagnosis. ***Arch Pathol Lab Med*** 2006; 130: 1466-1478.
7. Zhuge Y, Cheung MC, Yang R, Eldick D, Koniaris LG, Sola JE. Pediatric intestinal foregut and small bowel solid tumors: a review of 105 cases. ***J Surg Res*** 2009; 156: 95-102.
8. Alvarado-Cabrero I, García-Robles B, Medrano-Guzmán R, Hernández-Hoyos S, Alderete-Vázquez G. Gastrointestinal stromal tumors in the pediatric population. Report of two cases and a review of the literature. ***Cir Cir*** 2009; 77: 135-140.
9. Pappo AS, Janeway KA. Pediatric gastrointestinal stromal tumors. ***Hematol Oncol Clin North Am*** 2009; 23: 15-34, vii.
10. Bussolati G. Of GISTs and EGISTs, ICCs and ICs. ***Virchows Arch*** 2005; 447: 907-908.

11. Hu X, Forster J, Damjanov I. Primary malignant gastrointestinal stromal tumor of the liver. ***Arch Pathol Lab Med*** 2003; 127: 1606-1608.
12. Lasota J, Carlson JA, Miettinen M. Spindle cell tumor of urinary bladder serosa with phenotypic and genotypic features of gastrointestinal stromal tumor. ***Arch Pathol Lab Med*** 2000; 124: 894-897.
13. Mendoza-Marin M, Hoang MP, Albores-Saavedra J. Malignant stromal tumor of the gallbladder with interstitial cells of Cajal phenotype. ***Arch Pathol Lab Med*** 2002; 126: 481-483.
14. Miettinen M, El-Rifai W, H L Sobin L, Lasota J. Evaluation of malignancy and prognosis of gastrointestinal stromal tumors: a review. ***Hum Pathol*** 2002; 33: 478-483.
15 Mushtaq S, Mamoon N, Hassan U, Iqbal M, Khadim MT, Sarfraz T. Gastrointestinal stromal tumors-a morphological and immunohistochemical study. ***J Gastrointest Cancer*** 2009; 40: 109-114.
16. Fletcher CD, Berman JJ, Corless C, Gorstein F, Lasota J, Longley BJ, et al. Diagnosis of gastrointestinal stromal tumors: A consensus approach. ***Hum Pathol*** 2002; 33: 459-465.
17. Miettinen M, Sobin LH, Lasota J. Gastrointestinal stromal tumors of the stomach: a clinicopathologic, immunohistochemical, and molecular genetic study of 1765 cases with long-term follow-up. ***Am J Surg Pathol*** 2005; 29: 52-68.
18. Miettinen M, Makhlouf H, Sobin LH, Lasota J. Gastrointestinal stromal tumors of the jejunum and ileum: a clinicopathologic, immunohistochemical, and molecular genetic study of 906 cases before imatinib with long-term follow-up. ***Am J Surg Pathol*** 2006; 30: 477-489.
19. Al-Eid HS, Arteh SO. Cancer incidence report. Kingdom of Saudi Arabia: Ministry of Health Saudi Cancer Registry; 2005. [cited 2010 January 1] Available from URL: http://www.scr.org.sa
20. Miettinen M, Lasota J. Gastrointestinal stromal tumors--definition, clinical, histological, immunohistochemical, and molecular genetic features and differential diagnosis. ***Virchows Arch*** 2001; 438: 1-12.
21. Miettinen M, Blay JY, Sobin LH. Mesenchymal tumors of the stomach and small intestine. In: Hamilton SR, Lauri A, editors. World Health Organization classification of tumours: pathology and genetics of tumours of the digestive system. Lyon (FR): IARC Press; 2000. p. 62-65.
22. Trupiano JK, Stewart RE, Misick C, Appelman HD, Goldblum JR. Gastric stromal tumors: a clinicopathologic study of 77 cases with correlation of features with nonaggressive and aggressive clinical behaviors. ***Am J Surg Pathol*** 2002; 26: 705-714.
23. Greenson JK. Gastrointestinal stromal tumors and other mesenchymal lesions of the gut. ***Mod Pathol*** 2003; 16: 366-375.
24. Miettinen M, Lasota J. Gastrointestinal stromal tumors: pathology and prognosis at different sites. ***Semin Diagn Pathol*** 2006; 23: 70-83.
25. Medeiros F, Corless CL, Duensing A, Hornick JL, Oliveira AM, Heinrich MC, et al. KIT-negative gastrointestinal stromal tumors: proof of concept and therapeutic implications. ***Am J Surg Pathol*** 2004; 28: 889-894.
26. Tsuzuki T, Hara K, Takahashi E, Maeda N. Wilm's tumor gene protein (WT-1) and Calretinin (Cal) immunoreactivity in gastrointestinal stromal tumor (GIST). ***Mod Pathol*** 2005; 18 (Suppl 1): 121A. Available from URL: http://www.nature.com/modpathol/journal/v18/n1s/pdf/3800911a.pdf
27. Sarlomo-Rikala M, Tsujimura T, Lendahl U, Miettinen M. Patterns of nestin and other intermediate filament expression distinguish between gastrointestinal stromal tumors, leiomyomas and schwannomas. ***APMIS*** 2002; 110: 499-507.
28. Espinosa I, Lee CH, Kim MK, Rouse BT, Subramanian S, Montgomery K, et al. A novel monoclonal antibody against DOG1 is a sensitive and specific marker for gastrointestinal stromal tumors. ***Am J Surg Pathol*** 2008; 32: 210-218.
29. Lee HE, Kim MA, Lee HS, Lee BL, Kim WH. Characteristics of KIT-negative gastrointestinal stromal tumours and diagnostic utility of protein kinase C theta immunostaining. ***J Clin Pathol*** 2008; 61: 722-729.
30. Demetri GD, von Mehren M, Blanke CD, Van den Abbeele AD, Eisenberg B, Roberts PJ, et al. Efficacy and safety of imatinib mesylate in advanced gastrointestinal stromal tumors. ***N Engl J Med*** 2002; 347: 472-480.
31. Corless CL, Schroeder A, Griffith D, Town A, McGreevey L, Harrell P, et al. PDGFRA mutations in gastrointestinal stromal tumors: frequency, spectrum and in vitro sensitivity to imatinib. ***J Clin Oncol*** 2005; 23: 5357-5364.
32. Demetri GD, van Oosterom AT, Garrett CR, Blackstein ME, Shah MH, Verweij J, et al. Efficacy and safety of sunitinib in patients with advanced gastrointestinal stromal tumour after failure of imatinib: a randomised controlled trial. ***Lancet*** 2006; 368: 1329-1338.
33. Seandel M, Shia J, Linkov I, Maki RG, Antonescu CR, Dupont J. The activity of sunitinib against gastrointestinal stromal tumor seems to be distinct from its antiangiogenic effects. ***Clin Cancer Res*** 2006; 12: 6203-6204.
34. Gupta P, Tewari M, Shukla HS. Gastrointestinal stromal tumor. ***Surg Oncol*** 2008; 17: 129-138.

Related topics

Cao DX, Wang QG, Ji Y, Tan YS, Yang ZA, Zhang B, et al. Diagnostic methods for initial evaluation of primary gastrointestinal stromal tumors. ***Saudi Med J*** 2010; 31: 262-269.

Elshenawy YM, Ganote CE, Al-Abbadi MA. Fatal abdominal sarcomatosis secondary to gastrointestinal stromal tumor with bland histology. ***Saudi Med J*** 2009; 30: 1469-1472.

Hussain D, Baliga SK, Al-Madhi S, Hartung RU. Malignant gastrointestinal stromal tumor of the rectum with prolapse. ***Saudi Med J*** 2007; 28: 1291-1292.