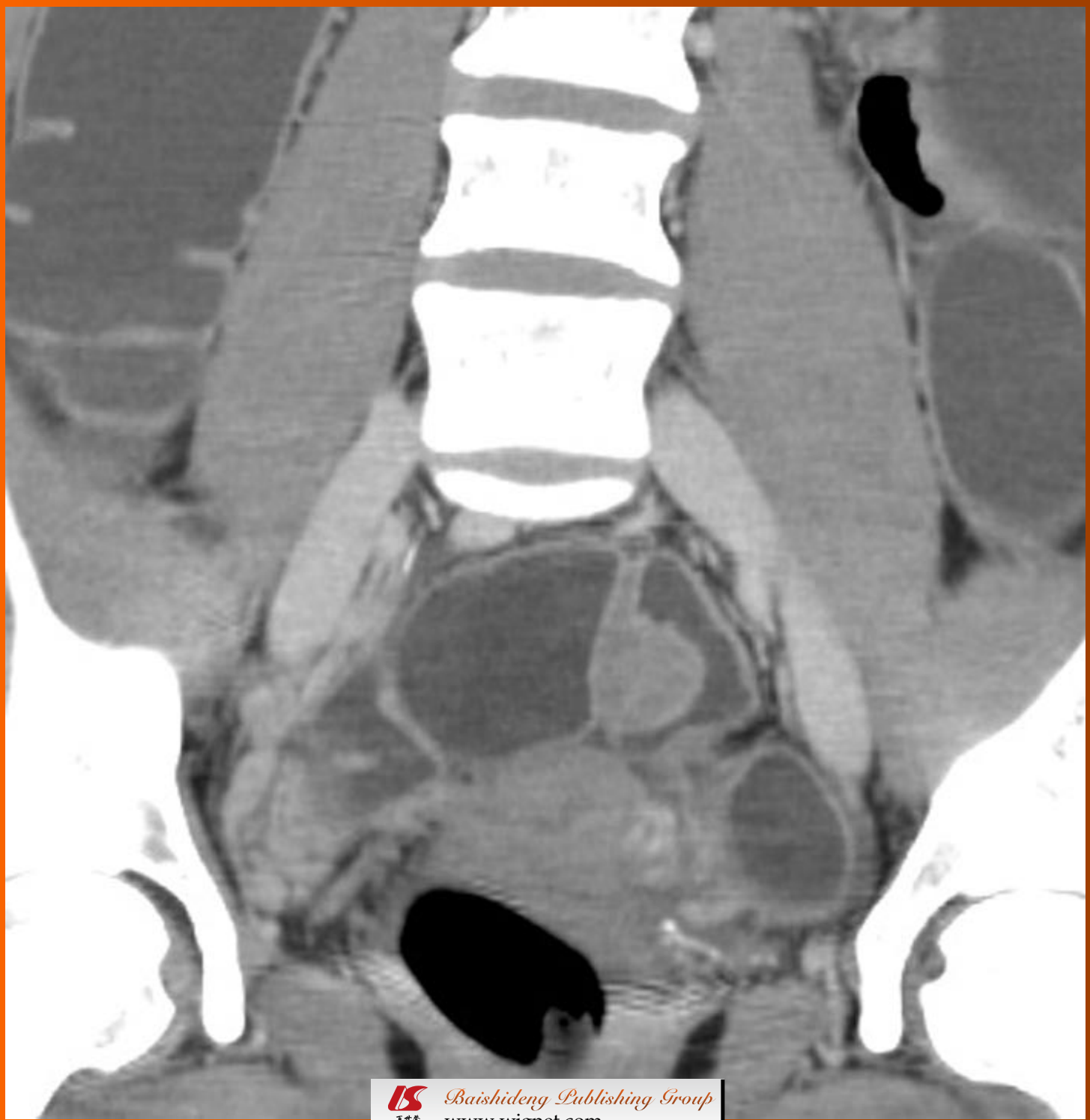
ISSN 1948-9366 (online)

World Journal of *Gastrointestinal Surgery*

World J Gastrointest Surg 2011 March 27; 3(3): 31-42



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World Journal of Gastrointestinal Surgery

A peer-reviewed, online, open-access journal of gastrointestinal surgery

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2009-2013

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World Journal of Gastrointestinal Surgery

Contents

Monthly Volume 3 Number 3 March 27, 2011

Contents

World Journal of Gastrointestinal Surgery
Volume 3 Number 3 March 27, 2011

NAME OF JOURNAL
World Journal of Gastrointestinal Surgery

LAUNCH DATE
November 30, 2009

SPONSOR
Beijing Baishideng BioMed Scientific Co., Ltd.,
Room 903, Building D, Ocean International Center,
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http://www.wjgnet.com

PUBLISHING
Baishideng Publishing Group Co., Limited,
Room 1701, 17/F, Henan Building,
No.90 Jaffe Road, Wanchai,
Hong Kong, China
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E-mail: baishideng@wjgnet.com
http://www.wjgnet.com

PUBLICATION DATE
March 27, 2011

SERIAL PUBLICATION NUMBER
ISSN 1948-9366 (online)

EDITORIAL OFFICE
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World Journal of Gastrointestinal Surgery
Room 903, Building D, Ocean International Center,
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World Journal of Gastrointestinal Surgery

Online Submissions: http://www.wjgnet.com/1948-9366office
wjgs@wjgnet.com
doi:10.4240/wjgs.v3.i3.31

World J Gastrointest Surg 2011 March 27; 3(3): 31-38
ISSN 1948-9366 (online)

EDITORIAL

Bowel endometriosis: Recent insights and unsolved problems

Simone Ferrero, Giovanni Camerini, Umberto Leone Roberti Maggiore, Pier L Venturini, Ennio Biscaldi, Valentino Remorgida

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Received: August 29, 2010 Revised: March 14, 2011
Accepted: March 21, 2011
Published online: March 27, 2011

Abstract

Bowel endometriosis affects between 3.8% and 37% of women with endometriosis. The evaluation of symptoms and clinical examination are inadequate for an accurate diagnosis of intestinal endometriosis. Transvaginal ultrasonography is the first line investigation in patients with suspected bowel endometriosis and allows accurate determination of the presence of the disease. Radiological techniques (such as magnetic resonance imaging and multidetector computerized tomography enteroclysis) are useful for estimating the extent of bowel endometriosis. Hormonal therapies (progestins, gonadotropin releasing hormone analogues and aromatase inhibitors) significantly improve pain and intestinal symptoms in patients with bowel stenosis less than 60% and who do not wish to conceive. However, hormonal therapies may not prevent the progression of bowel endometriosis and, therefore, patients receiving long-term treatment should be periodically monitored. Surgical excision of bowel endometriosis should be offered to symptomatic patients with bowel stenosis greater than 60%. Intestinal endometriotic nodules may be excised by nodulectomy or segmental resection. Both surgical procedures improve pain, intestinal symptoms and fertility. Nodulectomy may be associated with a lower rate of complications.

Key words: Bowel endometriosis; Diagnosis; Endometriosis; Gonadotropin releasing hormone analogue; Laparoscopy; Nodulectomy; Progestin; Colorectal resection

Peer reviewer: Imtiaz Ahmed Wani, PhD, Shodi Gali, Amira Kadal, Srinagar, India

Ferrero S, Camerini G, Leone Roberti Maggiore U, Venturini PL, Biscaldi E, Remorgida V. Bowel endometriosis: Recent insights and unsolved problems. *World J Gastrointest Surg* 2011; 3(3): 31-38 Available from: URL: http://www.wjgnet.com/1948-9366/full/v3/i3/31.htm DOI: http://dx.doi.org/10.4240/wjgs.v3.i3.31

INTRODUCTION

The term bowel endometriosis is used to indicate that endometrial-like gland and stroma infiltrate the intestinal wall reaching at least the subserous fat tissue and the subserous part of the enteric plexus[1] (Figure 1). Endometriotic foci located on the bowel serosa should be considered peritoneal endometriosis and not intestinal endometriosis. The exact prevalence of bowel endometriosis in the general population is unknown, although it is estimated that it affects between 3.8% and 37% of women with endometriosis[1]. The highest frequency of endometriotic nodules is on the sigmoid colon and the rectum, followed by the ileum, the appendix and the cecum[1].

SYMPTOMS

Intestinal endometriotic lesions may have variable size and depth of infiltration in the bowel wall and they may, therefore, cause a wide range of symptoms. In addition, bowel nodules are associated with the presence of other endometriotic lesions in the pelvis in over 99% of patients[2]. As a consequence, it may be difficult to determine whether intestinal endometriosis contributes to abdominal and pelvic pain. In general, small endometriotic lesions reaching only the subserosal fat tissue do not cause symptoms[3]. Larger nodules infiltrating the intestinal muscular layer cause a wide range of symptoms including dyschezia, constipation, diarrhea, abdominal bloating, painful bowel movements, passage of mucus in the stools and cyclical rectal bleeding[3,4]. The symptoms associated with intestinal endometriosis often mimic diarrhea-predominant or constipation-predominant irritable bowel syndrome[5,6] and this differential diagnosis may be particularly challenging[7]. It remains unclear how bowel endometriosis causes intestinal symptoms. Obviously, large endometriotic nodules may contain extensive fibrosis and thicken the bowel wall, resulting in a stenosis of the intestinal lumen (Figure 2) and perhaps mechanically hampering bowel transit. In addition, intestinal endometriotic lesions infiltrate and disrupt intestinal nervous plexus[3], damage interstitial Cajal cells[3], reduce the density of intestinal sympathetic nerve fibers[8] and thus, cause an alteration in bowel physiology.

DIAGNOSIS

Both the evaluation of symptoms and clinical examination are inadequate for an accurate diagnosis of intestinal endometriosis[9,10]. Therefore, ultrasonographic or radiological techniques are required to confirm this diagnosis before surgery[1]. Although no gold standard is universally accepted for the diagnosis of bowel endometriosis, magnetic resonance imaging (MRI) is one of the techniques most commonly used (Figure 3). A study comprising 195 patients with suspected endometriosis demonstrated that MRI has a sensitivity of 88%, a specificity of 98%, a positive predictive value of 95%, a negative predictive value of 95%, and an accuracy of 95% in diagnosing intestinal endometriosis[11]. These findings were subsequently confirmed by several other investigations[9,10,12,13]. However, in some cases, the diagnosis of intestinal endometriosis by MRI may be difficult because nodules with small hemorrhagic content have a signal intensity very close to that of the surrounding muscular structures[14]. Therefore, the injection of ultrasonography jelly in the vagina and the rectum during MRI has been proposed to facilitate the identification of intestinal lesions (Figure 4)[15].

Several studies have demonstrated that transvaginal ultrasonography may not only accurately diagnose the presence of rectosigmoid endometriosis but it may also estimate the depth of infiltration of the nodules in the intestinal wall[16-20]. However, it is well known that the diagnostic accuracy of ultrasonography depends on the experience of the operator. Adding water contrast to the rectum during transvaginal ultrasonography may facilitate not only the identification of intestinal endometriotic lesions but also the evaluation of the characteristics of the nodules (size, number, depth of infiltration in the intestinal wall, degree of stenosis of the intestinal lumen)[21-23]

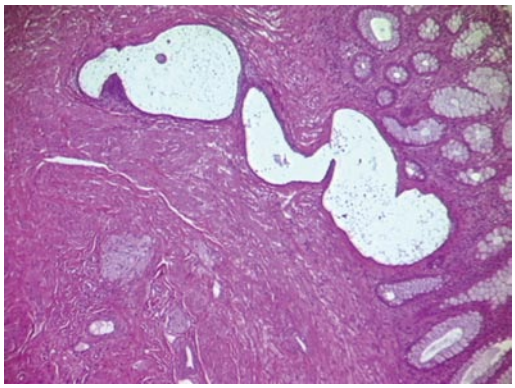

Figure 1 Section of a bowel endometriotic nodule, hemeatoxylin eosin staining demonstrates the nodule infiltrate the mucosa.

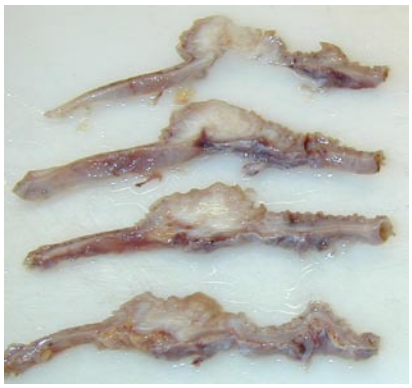

Figure 2 Sections of an intestinal endometriotic nodule demonstrating the thickening of the bowel wall caused by the endometriotic nodule.

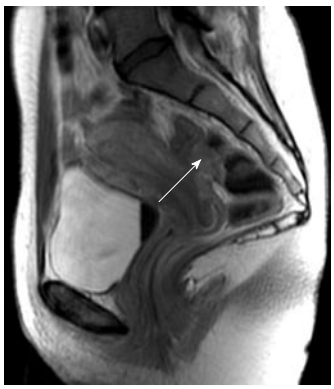

Figure 3 Magnetic resonance imaging, T2W sagittal image. A nodule infiltrating the rectum is well detectable (arrow). Enhancement of the nodule is observed after injection of iodinated contrast medium.

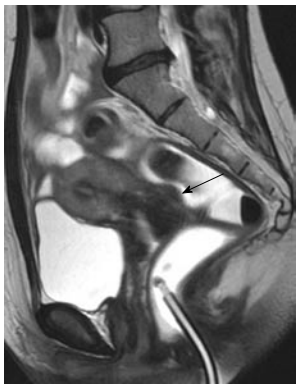

Figure 4 Magnetic resonance imaging enteroclysis. The rectosigmoid is distended by using 250-300 mL of ultrasonographic gel diluted with saline solution; the 20-Fr Foley catheter used for retrograde distension can be observed in the figure. The fluid solution has a biphasic behavior on MR sequences: hypointense in T1W images and hyperintense in T2W images. A small rectovaginal endometriotic nodule (larger diameter 12 mm) is observed (arrow).

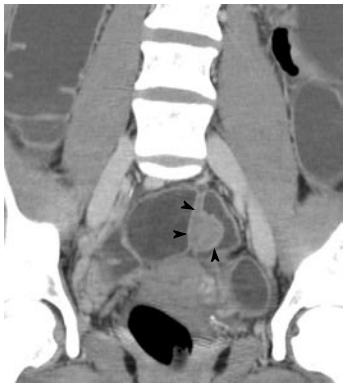

Figure 5 Multidetector computerized tomography enteroclysis, coronal reconstruction. Endometriotic nodule infiltrating the muscularis propria of the sigmoid (shown by the arrowheads); the mucosa is not infiltrated.

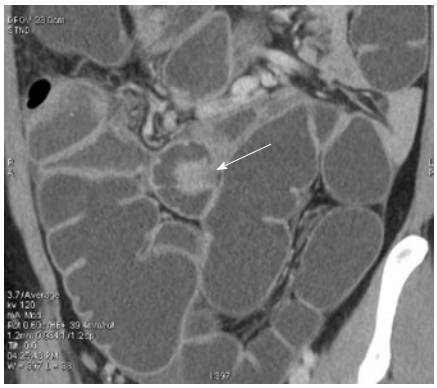

Figure 6 Multidetector computerized tomography enteroclysis, the arrow shows an endometriotic nodule infiltrating the ileum.

However, bowel preparation is required before adding water contrast to the rectum.

Double-contrast barium enema has been widely used in the past and remains an accurate technique for the diagnosis of bowel endometriosis[24-30]. Bowel nodules appear as extrinsic masses that are associated with mucosal fine crenulations[26]. The value of this exam is limited by the fact that the degree of infiltration of endometriosis in the intestinal wall cannot be determined.

Multidetector computerized tomography enteroclysis (MDCT-e) has recently been suggested for the diagnosis of bowel endometriosis[31,32]. After bowel preparation, retrograde colonic distension is performed on the computerized tomography bed by introducing about 2000 mL of water. During the enteroclysis, pharmacological inhibition of peristaltic waves is achieved by intravenous injection of hyoscine butylbromide. Patients are examined with a 16-row MDCT scanner in supine position; a volumetric acquisition is performed from the dome of the diaphragm to the pubic symphysis, in portal phase (40 s after the arterial peak) after the intravenous injection of iodinated contrast medium. MDCT-e allows estimation not only of the presence of intestinal endometriosis but also of the characteristics of the nodules, in particular, the depth of infiltration of endometriosis in the intestinal wall. The infiltration of the intestinal serosa is characterized by the presence of small nodules adjacent to the bowel loop resulting in an irregular intestinal profile in these nodules, the fat plane between endometriosis and the bowel wall is preserved. When the muscularis propria is infiltrated, MDCT-e allows observation of the multilayered stratification of the thickened bowel wall. Full thickness infiltration of the bowel wall is diagnosed when the solid nodules infiltrate the submucosa (which appears as a hypodense layer between the lumen and the muscularis propria) reaching the intestinal mucosa (Figure 5)[31,32]. A strength of MDCT-e is that it allows detection of endometriotic lesions in the cecum and lower ileal loops which might be undiagnosed by other exams (Figure 6). The major disadvantages of MDCT-e are the use of iodinated contrast medium and ionizing radiations.

Rectal endoscopic ultrasonography has been widely used for the diagnosis of intestinal endometriosis[12,33-34]. This exam permits to estimation of the precise depth of infiltration of endometriosis in the intestinal wall (in particular, the infiltration of the muscularis propria), the maximum diameter of the lesions and the distance of the lesions from the anus[1]. In the last few years, rectal endoscopic ultrasonography has been largely replaced by transvaginal ultrasonography which is better tolerated by the patients. In addition, the equipment for performing rectal endoscopic ultrasonography is often unavailable to gynecologists, the clinicians who are commonly involved in the diagnosis and management of endometriosis.

Colonoscopy has limited value in the diagnosis of intestinal endometriosis because the disease infiltrates the intestinal wall from the serosa toward the mucosa and, therefore, only large nodules infiltrating the mucosa and/or causing a severe stenosis of the intestinal lumen can be diagnosed during this type of exam[1]. In rare cases, colonoscopy may be used to exclude the presence of colorectal cancer.

MANAGEMENT OF BOWEL ENDOMETRIOSIS

The management strategy for bowel endometriosis depends on the size of the nodule, the degree of stenosis of the intestinal lumen and the symptoms experienced by the patients. In a small percentage of women, intestinal endometriotic lesions do not cause symptoms and, therefore, these patients may not receive treatment. However, in these patients, the thickening of the bowel lumen (leading to intestinal stenosis) should be evaluated in order to exclude the risk of bowel occlusion. Larger endometriotic nodules causing intestinal symptoms may be treated by hormonal therapy or by surgery.

MEDICAL TREATMENT OF BOWEL ENDOMETRIOSIS

Hormonal therapy cannot be offered to all women with intestinal endometriosis. Patients with intestinal nodules causing a bowel stenosis and those wishing to conceive are not good candidates for long-term endocrine therapy, which inhibits ovulation. The available ultrasonographic and radiological techniques allow accurate estimation of the presence, number and depth of infiltration of intestinal endometriotic lesions; hormonal therapy may be safely offered to women with estimated bowel stenosis of < 60%.

Hormonal therapies commonly used to treat pelvic endometriosis can generally be prescribed to women with bowel endometriosis. In the past, gonadotropin-releasing hormone analogues (GnRH-a) have occasionally been used to treat patients with bowel endometriosis[35]. However, most authors simply report the recurrence of pain symptoms when the therapy with GnRH-a was discontinued[36,37]. In one case report, the disappearance of an intestinal endometriotic polyp was reported after 3-mo treatment with GnRH-a[38]. More recently, a prospective study systematically investigated the effects of a 12-mo treatment with triptorelin and tibolone on pain and intestinal symptoms in 18 women with colorectal endometriotic nodules[39]. As expected, the treatment improved pain symptoms. In addition, it significantly reduced the severity of symptoms mimicking diarrhea-predominant irritable bowel syndrome, intestinal cramping, abdominal bloating and passage of mucus with stools. On the contrary, seven women with symptoms mimicking constipation- predominant irritable bowel syndrome reported only minor changes in their intestinal symptoms[39].

Progestins are among the most commonly used medical treatments for endometriosis. Several studies have proved that these agents effectively reduce the severity of symptoms caused by pelvic endometriosis and are in general well tolerated[40]. One prospective study examined the effects of progestins on the symptoms caused by intestinal endometriosis[41]. Forty symptomatic women with colorectal endometriosis were treated with norethisterone acetate (2.5 mg/d) for 12 mo. This reduced the severity of chronic pelvic pain, deep dyspareunia and dyschezia. As expected, the treatment resulted in the disappearance of symptoms related to the menstrual cycle such as dysmenorrhea, constipation during the menstrual cycle, diarrhea during the menstrual cycle and cyclical rectal bleeding. The severity of diarrhea, intestinal cramping and passage of mucus significantly improved during treatment. However, the administration of norethisterone acetate did not produce a significant effect on constipation, abdominal bloating and feeling of incomplete evacuation after bowel movements. More recently, aromatase inhibitors have been suggested for the treatment of endometriosis[42]. A prospective pilot study comprising six women with colorectal endometriosis demonstrated that pain and intestinal symptoms are improved by the combined administration of an aromatase inhibitor (letrozole, 2.5 mg/d) and norethisterone acetate (2.5 mg/d) continuously for 6 mo[43].

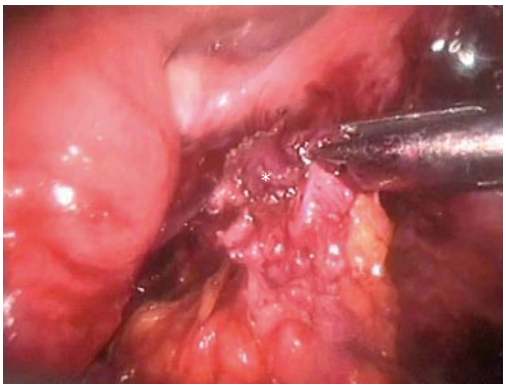

Figure 7 Nodulectomy, the endometriotic nodule is shown by the asterisk.

Although hormonal therapies may improve the symptoms caused by intestinal endometriosis, the natural history of bowel nodules is not established. It is well known that patients undergoing surgery because of extensive intestinal endometriotic lesions have often used hormonal therapies for years. Therefore, it is possible that hormonal therapies, despite improving symptoms, do not prevent the development or progression of intestinal endometriosis. A recent report described the case of a woman with a diagnosis of an endometriotic serosal sigmoid nodule at MDCT-e which enlarged after 41 mo of continuous oral contraceptive pill, infiltrating the bowel submucosa and requiring segmental bowel resection[44]. Therefore, patients using long-term hormonal therapies to treat intestinal endometriosis should be carefully monitored by using transvaginal ultrasonography or MRI in order to identify the potential progression of intestinal nodules. In addition, these patients should be informed that the progression of the disease might occur despite the improvement in pain symptoms.

SURGICAL TREATMENT OF BOWEL ENDOMETRIOSIS

Small endometriotic lesions infiltrating only the intestinal

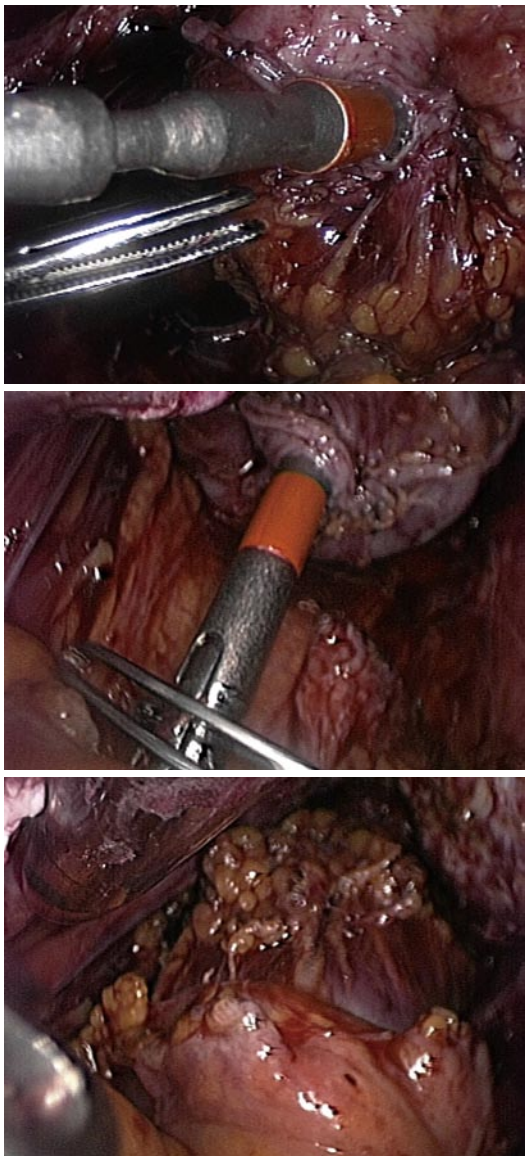

Figure 8 A mechanic circular stapler inserted transrectally is used to perform an end-to-end anastomosis.

serosa or the adventitia are unlikely to cause symptoms and may, therefore, not be excised. However, they can be easily removed by cutting the normal bowel wall adjacent to the lesion with scissors or CO_2 laser. The defect of the intestinal wall can then be repaired by interrupted suture.

Larger colorectal endometriotic nodules infiltrating the intestinal muscularis propria can be excised by either nodulectomy (partial or full thickness) (Figure 7) or segmental bowel resection. The choice of the surgical technique is based partly on the characteristics of the intestinal lesions such as, number of nodules, size of the nodules, depth of infiltration of the intestinal wall and percentage of the intestinal circumference infiltrated by endometriosis. However, surgeon's experience and preferences also influence the decision to perform either bowel segmental resection or nodulectomy.

Colorectal resection which is a standardized surgical procedure that has been used for decades for the treatment of sigmoid or rectal cancer is similarly performed (most often by laparoscopy) in women with endometriosis. The endometriotic nodule is separated from the adherent tissues, but no attempt is made to separate the nodule from the bowel. Bowel preparation requires opening the pararectal spaces in order to mobilize the bowel. As endometriosis is not a malignant disease, the separation of the fibrofatty tissue attached to the bowel is best performed immediately adjacent to the bowel wall because the vessels are smaller and easier to coagulate. The mesentery is dissected no more than 2 cm past the endometriotic lesions in order to maintain adequate blood supply to the edges of the anastomosis[1]. After the bowel is resected caudal to the endometriotic lesion by using Endo GIA, the proximal portion of the bowel is extracted through a small sovrapubic incision. After accurate inspection and palpation, the bowel segment infiltrated by endometriosis is resected. The anvil of a transanal circular stapler is inserted into the proximal bowel stump and fixed by a purse-string suture. An end-to-end anastomosis is performed transrectally using a mechanic circular stapler (Figure 8). After completing the anastomosis, the possible presence of leaks must always be checked. In patients with endometriosis, the most important argument in favor of segmental bowel resection is the fact that this technique allows a more radical removal of intestinal endometriotic foci, thus minimizing the risk of recurrences. Over the last 10 years, several studies have showed that, in women with colorectal endometriosis, segmental bowel resection improves pain, intestinal symptoms and quality of life[12,45-48]. However, segmental resection may be associated with several complications including urinary retention, inadvertent ureteral lesions, anastomosis leakage and fecal peritonitis, rectovaginal fistulas, anastomotic stenosis, pelvic abscesses and postoperative constipation[1,30,46]. Segmental bowel resection can be performed either by laparoscopy or by laparotomy. During segmental colorectal resection because of bowel endometriosis, the reported conversion rates from laparoscopy to laparotomy range from 0% to 20%[49,50]. A recent randomized trial comparing laparoscopic and open colorectal resection for endometriosis demonstrated that the two surgical techniques result in a similar improvement in symptoms and quality of life[49]. However, blood loss, analgesic consumption and complication rate were higher in patients undergoing open surgery than in those undergoing laparoscopy[49].

It is currently debated whether bowel endometriosis should be removed by segmental resection or by nodulectomy. A prospective surgical and histological study evaluated the completeness of full thickness disk resection[46]. In 16 women requiring segmental bowel resection, nodulectomy was performed before segmental resection. In 7 out of 16 cases (43.8%), endometriosis was still present in the muscularis propria adjacent to the site of nodulectomy. A similar rate of persistent disease was observed after laparoscopic and laparotomic full thickness disc resection[51]. However, the clinical implications of these observations remain unclear because it seems unlikely that

small endometriotic lesions will cause symptoms or progress to large intestinal nodules. In addition, in segmental resection, persistence of endometriosis may also be observed at the margin of the resected intestinal segment[51]. A recent study of 500 women with deep endometriosis and rectal muscularis involvement demonstrated that, after laparoscopic nodulectomy, symptoms recurred only in 8% of the patients after a median follow-up of 3.1 years[30]. In addition, the recurrence of symptoms was significantly lower in patients who conceived after surgery than in those who did not conceive[30]. When compared with segmental bowel resection, nodulectomy may have the advantage of a lower incidence of postoperative unpleasant functional digestive outcomes (such as increase in the daily number of stools, severe postoperative constipation and urinary dysfunction)[52]. In fact, during nodulectomy, nerves and vascular blood supply are preserved because it is not necessary to perform the deep lateral dissection that is required during segmental resection[30,52]. Similarly, after segmental bowel resection, the full recovery of gastrointestinal function can usually be observed several months after surgery[53].

FERTILITY AFTER SURGERY FOR BOWEL ENDOMETRIOSIS

Several studies have showed that good pregnancy rates are achieved after surgical excision of intestinal endometriosis. A retrospective study of 22 women wishing to conceive reported a 45.5% pregnancy rate after a mean follow-up of 24 mo from laparoscopic colorectal resection[54]. Subsequently, a prospective cohort study of 46 symptomatic women undergoing colorectal resection because of endometriosis showed that the pregnancy rate was significantly higher after laparoscopic (57.6%) than after laparotomic procedures (23.1%)[55]. More recently, a study of 288 women wishing to conceive demonstrated that nodulectomy is associated with an 84% pregnancy rate (obtained either naturally or after assisted reproductive technologies) after a median follow-up of 3.1 years[30].

CONCLUSION

The symptoms of bowel endometriosis are not specific, therefore its presence should be suspected in all women with endometriosis who complain of intestinal symptoms. Transvaginal ultrasonography is the first line investigation for patients with suspected bowel endometriosis as it allows to accurate determination of the presence of the disease[19,20,22]. Additional radiological techniques (such as MRI and MDCT-e) are useful to determine the extent of intestinal endometriotic lesions[11,31].

The management of bowel endometriosis depends on the severity of symptoms, the extent of the disease, the desire of the patient to conceive and the tolerability of hormonal therapies. Progestins and gonadotropin releasing hormone analogues may be offered to women with stenosis of the bowel lumen < 60% who wish to avoid surgery and do not desire to conceive[39,41]. However, hormonal therapies may not prevent the progression of bowel endometriosis[44] and, therefore, patients receiving long-term treatment should be periodically monitored.

The decision to carry out surgery should be undertaken in selected patients, such as those who are highly symptomatic, have a severe stenosis of the intestinal lumen or cannot use hormonal therapies because of the desire to conceive. Intestinal endometriotic lesions can be excised by either segmental resection or nodulectomy; it remains to be established whether one surgical technique is superior to the other. However, before surgery patients should be fully informed of potential complications including the time required to obtain a full recovery of the digestive function.

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S- Editor Wang JL **L- Editor** Hughes D **E- Editor** Zheng XM

World Journal of Gastrointestinal Surgery

Online Submissions: http://www.wjgnet.com/1948-9366office
wjgs@wjgnet.com
doi:10.4240/wjgs.v3.i3.39

World J Gastrointest Surg 2011 March 27; 3(3): 39-42
ISSN 1948-9366 (online)

CASE REPORT

A case report of extrahepatic portal vein aneurysm with thrombosis

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Received: February 21, 2010 Revised: November 29, 2010
Accepted: December 6, 2010
Published online: March 27, 2011

Abstract

Extrahepatic portal vein aneurysm (PVA) is very rare with only 17 previously reported cases. Methods of treatment include resection, thrombectomy, and portal venous decompression. We report herein the first case of large PVA with thrombosis which has been managed without surgical treatment over a long period. A PVA was detected in a 78-year-old woman by abdominal ultrasonography. Computed tomography revealed an aneurysm of 6 cm in a diameter in the porta hepatis. Portal venography showed obstruction of the portal vein and developed collateral vessels around the aneurysm. Since the patient had no symptoms of portal hypertension, we decided to carefully manage her clinical course without surgical treatment. At present, this patient is healthy and has developed no complications over the 5 years since leaving our hospital. This case suggests that surgical treatment is not required for PVA without portal hypertension.

Key words: Portal vein aneurysm; Thrombosis; Surgical treatment

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Ishimura K, Otani T, Wakabayashi H, Okano K, Goda F, Suzuki Y. A case report of extrahepatic portal vein aneurysm with thrombosis. *World J Gastrointest Surg* 2011; 3(3): 39-42 Available from: URL: http://www.wjgnet.com/1948-9366/full/v3/i3/39.htm DOI: http://dx.doi.org/10.4240/wjgs.v3.i3.39

INTRODUCTION

Portal vein aneurysm (PVA) is a very rare venous malformation. Barzilai *et al*[1] reported the first case of PVA in 1956. To our knowledge, only 17 proven cases of extrahepatic PVA have previously been reported in the English language, worldwide[1-16]. The etiology of PVA is unknown due to its rarity. PVA is thought to be either of congenital origin, caused by hypoplasia or atresia of the portal vein, or acquired as a result of portal hypertension or trauma. Methods of PVA management include observation, resection, thrombectomy, and portal venous decompression. We report herein a case of large PVA with thrombosis that has been managed without surgical treatment in a long-term.

CASE REPORT

A 78-year-old woman was admitted to our surgical department with a PVA. Her chief complaint was abdominal discomfort. The patient had no history of abdominal surgery, liver biopsy, trauma, or hepatitis. She had already been diagnosed 6 years previously at another medical institution with an aneurysm of 3 cm in a diameter located in the main portal trunk. However, she had not under-

Table 1 Reported extrahepatic portal vein aneurysms

Author (year)	Age (yr)	Sex	Size(cm)	Liver disease	Portal hypertension	Treatment
Barzilai *et al*[1] (1956)	21	F	2	Liver cirrhosis	+	Splenectomy
Leonsins *et al*[2] (1960)	52	M	8	Liver cirrhosis	+	Splenectomy
Sedgwick[3] (1960)	25	F	5	Liver cirrhosis	+	Cholecystojejunostomy
Hermann *et al*[4] (1965)	26	F	6	Portal fibrosis	+	Portocaval shunt
Liebowitz *et al*[5] (1967)	55	F	8	-	-	Splenectomy
Thomas[6] (1967)	18	M	8	Obstructive jaundice	+	Died
Thomas *et al*[6] (1967)	13	F	3	-	+	Portocaval shunt
Vine *et al*[7] (1979)	50	F	3	Hepatic parenchymal abnormality	-	Observation
Boyez *et al*[8] (1986)	57	F	4	-	-	Observation
Thompson *et al*[9] (1986)	21	F	6	-	-	Cholecystectomy
Andoh *et al*[10] (1988)	57	F	8	-	-	Partial resection PVA Splenectomy
Lee *et al*[11] (1989)	5	M	1.9	-	-	Observation
Baker *et al*[12] (1990)	34	F	8	-	-	Resection PVA Splenectomy
Hagiwara *et al*[13] (1991)	34	M	2.7	-	-	Observation
Dognini *et al*[14] (1991)	67	F	2.4	-	-	Sphincterotomy
Glazer *et al*[15] (1991)	26	F	7	-	+	Thrombectomy Aneurysmorrhaphy
Brock *et al*[16] (1997)	72	F	6	-	-	Resection PVA
Present case	78	F	6	-	-	Observation

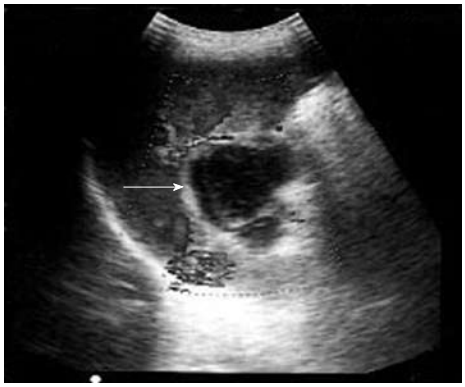

Figure 1 Ultrasound oblique scan through the long axis of the portal vein.

gone further examination until this hospitalization. On admission, the patient was 137 cm in height and weighed 40 kg, having exhibited no weight loss. Her consciousness was alert. Results of a general physical examination were otherwise normal; the palpebral conjunctiva was not anemic and the bulbar conjunctiva was not icteric. The liver and spleen were not palpable, and the abdomen was soft and flat, with no palpable tumor. No vascular bruit was heard. Laboratory test results included aspartate transaminase of 22 U/L, alanine transaminase of 63 U/L, and alkaline phosphatase of 430 U/L. Abdominal ultrasonogram showed a hypoechoic mass connected with the main portal trunk (Figure 1). Computed tomography scan revealed a mass of 6 cm in a diameter in the porta hepatis (Figure 2A). Developed collateral vessels around the mass were detected on the delay phase by intravenous contrast material, suggesting thrombosis of the portal aneurysm (Figure 2B). The venous phase of a superior mesenteric artery angiogram showed obstruction of the portal vein, and that intrahepatic portal flow was maintained by collateral supply (Figure 3). There was no arterial or venous fistula. As the patient's liver function was almost within normal limits and she showed no symptoms of portal hypertension, we decided not to intervene surgically but to carefully monitor her clinical course. She was well when discharged, and has remained symptom-free for 5 years since leaving the hospital.

DISCUSSION

Since the natural history of PVA is not clear, it is difficult to determine the strategy of treatment for this disease.

The 18 reported cases of extrahepatic PVA, including our case[1-16], are listed in Table 1. The age of patients in these cases varies from 5 to 78 years old, with our patient being oldest. The PVA ranged from 1.9 to 8 cm in a diameter, with an average diameter of 5.3 cm. Six of the 18 cases revealed underlying liver disease[1-4,6,7], and seven were associated with portal hypertension[1-4,6,15]. The advance of radiological diagnosis has resulted in the identification of a number of PVA without liver disease since 1986. Surgical treatments were performed on 13 out of the 18 cases, and direct surgery for PVA was carried out in four[10,12,15,16]. Resection of PVA was done in three cases, and thrombectomy and aneurysmorrhaphy were performed on one case with thrombus. On the other hand, five cases including our case, were managed by observation[7,8,11,13]. A PVA of 4 cm in diameter monitored for 2 years did not change in size[8] and aneurysms reported by Lee and Hagiwara did not change in size after 5 years of observation[11,13]. These aneurysms were smaller than the one observed in our patient.

Excluding our case, three out of 17 patients had throm-

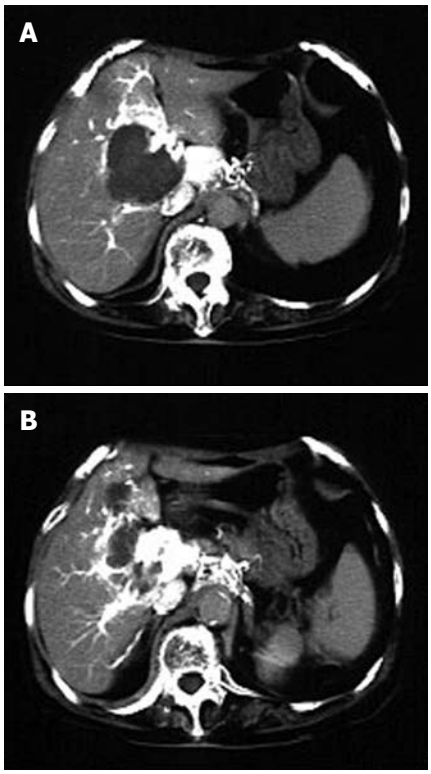

Figure 2 Abdominal computed tomography scan with contrast. A: A hypovascular mass in the porta hepatis; B: Developed collateral vessels around the mass.

bus[1,6,15]. Two of these died of aneurysm rupture[1,6]. Both were less than 30 years old. Their PVA etiology seemed to be acquired, as their PVA was the result of liver disease. On the other hand, the remaining patient was treated by a surgical procedure, and remained symptom free for 10 years after operation[15]. The origin of this case was also acquired. However it is unclear whether operation was needed in this case, given that the PVA contained a large amount of organized clot and the wall of the aneurysm showed normal venous structure with no atrophy of the muscle. Our patient had chronic progress, and PVA etiology was suspected to be of congenital origin. She was saved from rupture of the PVA as collateral vessels had developed around it. We inferred that the patient was symptom free because of the formation of sufficient collaterals. We believe that after short follow-up asymptomatic aneurysm with thrombus can be successfully managed by observation alone.

The decision on surgical treatment depends on the size, anatomy of PVA as well as the symptoms and condition of the patient. In the past, large PVAs over 4 cm in diameter had been operated on. Ours seems to be the first case reported without operative treatment for PVAs over 5 cm in a diameter. Miyauchi *et al*[17] concluded that the indications for surgical interventions in the treatment of PVA with porta hepatic venous fistula were as follows: (1) Patients with symptoms and large shunts; (2) Patients with enlarging fistulae; and (3) Patients with multiple fistulae where angiography shows that the lesions are sufficiently localized that the volume of the shunt cannot be reduced by conservative therapy. Moreover, patients who have biliary tract obstruction and hemobilia caused by PVAs also require operation.

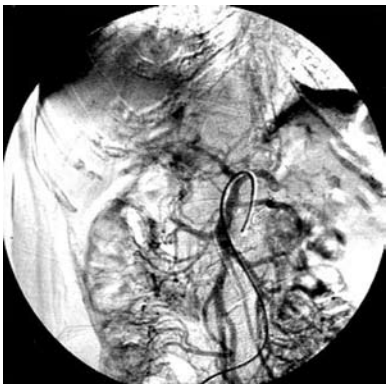

Figure 3 Portal venogram (digital subtraction angiogram) after selective superior mesenteric arteriography.

Since the natural history and incidence of PVA is not well known, it is difficult to decide the best treatment. Prognosis in symptomatic patients treated with surgery is dependent on the underlying liver disease. In a case reported by Brock, the patient underwent PVA resection because of a lack of experience for judging whether the large uncomplicated saccular PVA in his patient should be resected[16]. However, our case indicated a natural history of PVA without arterial or venous fistula, and suggests that surgical treatment is not required for PVA in the absence of portal hypertension. Moreover, our case also suggests that it might be possible to decide PVA treatment based on etiology.

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Online Submissions: http://www.wjgnet.com/1948-9366office
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World J Gastrointest Surg 2011 March 27; 3(3): I
ISSN 1948-9366 (online)

Acknowledgments to reviewers of *World Journal of Gastrointestinal Surgery*

Many reviewers have contributed their expertise and time to the peer review, a critical process to ensure the quality of *World Journal of Gastrointestinal Surgery*. The editors and authors of the articles submitted to the journal are grateful to the following reviewers for evaluating the articles (including those published in this issue and those rejected for this issue) during the last editing time period.

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World J Gastrointest Surg 2011 March 27; 3(3): I
ISSN 1948-9366 (online)

Meetings

Events Calendar 2011

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Gastrointestinal Cancers Symposium (ASCO GI), San Francisco, CA, United States

January 26-30, 2011
5th UK Alpine Liver and Pancreatic Surgery Meeting, Carlo Magno Zeledria Hotel, Madonna di Campiglio, Italy

February 01-03, 2011
6th Annual Academic Surgical Congress, Huntington Beach, CA, United States

February 21-26, 2011
Minimally Invasive Surgery Symposium 2011, The Grand America Hotel, Salt Lake City, Utah, United States

March 03-06, 2011
The Society of Surgical Oncology 63rd Annual Meeting, San Antonio, TX, United States

March 10-13, 2011
The American Hepato-Pancreato-Biliary Association Annual Meeting, Miami Beach, FL, United States

March 14-17, 2011
British Society for Gastroenterology Annual Meeting, International Convention Centre, Birmingham, United Kingdom

March 25-27, 2011
NZAGS Conference 2011 GI Surgery, New Plymouth, New Zealand

March 30-April 02, 2011
The Society of American Gastrointestinal and Endoscopic Surgeons 2011 Annual Meeting, San Antonio Convention Center, San Antonio, TX, United States

April 02-06, 2011
The American Association for Cancer Research 102nd Annual Meeting, Orlando, FL, United States

April 10-12, 2011
The American Association of Endocrine Surgeons 32nd Annual Meeting, Houston, TX, United States

April 14-16, 2011
The American Surgical Association 131st Annual Meeting, Boca Raton, FL, United States

May 07-10, 2011
Digestive Disease Week, Chicago, IL, United States

May 07-10, 2011
45th Annual Meeting of the Pancreas Club, Chicago, IL, United States

June 15-18, 2011
19th International Congress of the European Association for Endoscopic Surgery, in collaboration with and incorporating the 15th National Congress of the Italian Society of Endoscopic Surgery, Torino, Italy

September 10-14, 2011
International Congress of Endoscopy, Los Angeles, CA, United States

September 22-24, 2011
5th joint EAES and ESGE, European Workshop on NOTES, Frankfurt, Germany

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September 23-27, 2011
ECCO-European Society for Medical Oncology Congress, Stockholm, Sweden

October 23-27, 2011
The American College of Surgeons 97th Annual Clinical Congress, San Francisco, CA, United States

November 02-05, 2011
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World J Gastrointest Surg 2011 March 27; 3(3): I-V
ISSN 1948-9366 (online)

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Name of journal

World Journal of Gastrointestinal Surgery

Serial publication number

ISSN 1948-9366 (online)

Indexing/abstracting

PubMed Central, PubMed, Digital Object Identifer, and Directory of Open Access Journals.

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Chinese journal article (list all authors and include the PMID where applicable)

2 **Lin GZ**, Wang XZ, Wang P, Lin J, Yang FD. Immunologic effect of Jianpi Yishen decoction in treatment of Pixu-diarrhoea. *Shijie Huaren Xiaohua Zazhi* 1999; **7**: 285-287

In press

3 **Tian D**, Araki H, Stahl E, Bergelson J, Kreitman M. Signature of balancing selection in Arabidopsis. *Proc Natl Acad Sci USA* 2006; In press

Organization as author

4 **Diabetes Prevention Program Research Group**. Hypertension, insulin, and proinsulin in participants with impaired glucose tolerance. *Hypertension* 2002; **40**: 679-686 [PMID: 12411462 PMCID:2516377 DOI:10.1161/01.HYP.0000035706.28494.09]

Both personal authors and an organization as author

5 **Vallancien G**, Emberton M, Harving N, van Moorselaar RJ; Alf-One Study Group. Sexual dysfunction in 1, 274 European men suffering from lower urinary tract symptoms. *J Urol* 2003; **169**: 2257-2261 [PMID: 12771764 DOI:10.1097/01.ju.0000067940.76090.73]

No author given

6 21st century heart solution may have a sting in the tail. *BMJ* 2002; **325**: 184 [PMID: 12142303 DOI:10.1136/bmj.325.7357.184]

Volume with supplement

7 **Geraud G**, Spierings EL, Keywood C. Tolerability and safety of frovatriptan with short- and long-term use for treatment of migraine and in comparison with sumatriptan. *Headache* 2002; **42** Suppl 2: S93-99 [PMID: 12028325 DOI:10.1046/j.1526-4610.42.s2.7.x]

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8 **Banit DM**, Kaufer H, Hartford JM. Intraoperative frozen section analysis in revision total joint arthroplasty. *Clin Orthop Relat Res* 2002; (**401**): 230-238 [PMID: 12151900 DOI:10.1097/00003086-200208000-00026]

No volume or issue

9 Outreach: Bringing HIV-positive individuals into care. *HRSA Careaction* 2002; 1-6 [PMID: 12154804]

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Personal author(s)

10 **Sherlock S**, Dooley J. Diseases of the liver and billiary system. 9th ed. Oxford: Blackwell Sci Pub, 1993: 258-296

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11 **Lam SK**. Academic investigator's perspectives of medical treatment for peptic ulcer. In: Swabb EA, Azabo S. Ulcer disease: investigation and basis for therapy. New York: Marcel Dekker, 1991: 431-450

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12 **Breedlove GK**, Schorfheide AM. Adolescent pregnancy. 2nd ed. Wieczorek RR, editor. White Plains (NY): March of Dimes Education Services, 2001: 20-34

Conference proceedings

13 **Harnden P**, Joffe JK, Jones WG, editors. Germ cell tumours V. Proceedings of the 5th Germ cell tumours Conference; 2001 Sep 13-15; Leeds, UK. New York: Springer, 2002: 30-56

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14 **Christensen S**, Oppacher F. An analysis of Koza's computational effort statistic for genetic programming. In: Foster JA, Lutton E, Miller J, Ryan C, Tettamanzi AG, editors. Genetic programming. EuroGP 2002: Proceedings of the 5th European Conference on Genetic Programming; 2002 Apr 3-5; Kinsdale, Ireland. Berlin: Springer, 2002: 182-191

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15 Morse SS. Factors in the emergence of infectious diseases. Emerg Infect Dis serial online, 1995-01-03, cited 1996-06-05; 1(1): 24 screens. Available from: URL: http://www.cdc.gov/ncidod/eid/index.htm

Patent (list all authors)

16 **Pagedas AC**, inventor; Ancel Surgical R&D Inc., assignee. Flexible endoscopic grasping and cutting device and positioning tool assembly. United States patent US 20020103498. 2002 Aug 1

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Write as mean ± SD or mean ± SE.

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