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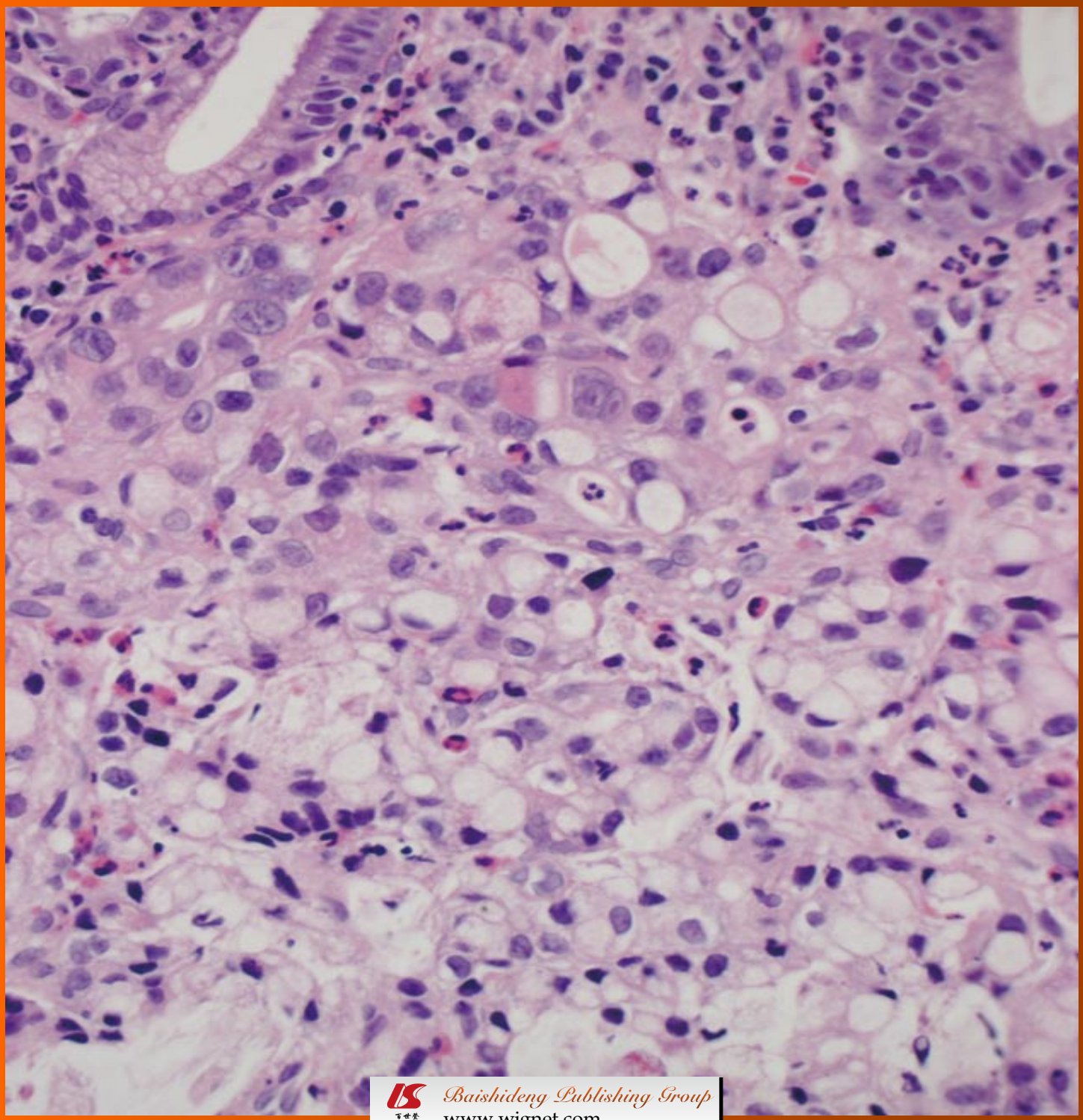

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Contents

Monthly Volume 3 Number 5 May 15, 2011

EDITORIAL

71 Emerging role of bioflavonoids in gastroenterology: Especially their effects on intestinal neoplasia

Hoensch HP, Oertel R

CASE REPORT

75 Pedunculated gastric tube interposition in an esophageal cancer patient with prepyloric adenocarcinoma

Kanda T, Sato Y, Yajima K, Kosugi S, Matsuki A, Ishikawa T, Bamba T, Umezu H, Suzuki T, Hatakeyama K

Contents

World Journal of Gastrointestinal Oncology
Volume 3 Number 5 May 15, 2011

ACKNOWLEDGMENTS I Acknowledgments to reviewers of *World Journal of Gastrointestinal Oncology*

APPENDIX I Meetings

I-V Instructions to authors

ABOUT COVER Kanda T, Sato Y, Yajima K, Kosugi S, Matsuki A, Ishikawa T, Bamba T, Umezu H, Suzuki T, Hatakeyama K. Pedunculated gastric tube interposition in an esophageal cancer patient with prepyloric adenocarcinoma.
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Telephone: +86-10-8538-1892
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Room 903, Building D, Ocean International Center,
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Telephone: +86-10-8538-1891
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EDITORIAL

Emerging role of bioflavonoids in gastroenterology: Especially their effects on intestinal neoplasia

Harald P Hoensch, Reinhard Oertel

Harald P Hoensch, Marien-Hospital, Private Practice of Internal Medicine, Martinspfad 72, D-64285 Darmstadt, Germany
Reinhard Oertel, Department of Clinical Pharmacology, Technical University of Dresden, Fiedler Str. 27, D-01307 Dresden, Germany
Author contributions: Hoensch HP made substantial contributions to the conception and design, drafted the article and provided the final approval of the version to be published; Oertel R acquired the data and did the analysis of serum levels of apigenin.
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Correspondence to: Harald P Hoensch, MD, Professor, Marien-Hospital, Private Practice of Internal Medicine, Marienhospital Darmstadt, Martinspfad 72, D-64285 Darmstadt, Germany. h.p.hoensch@t-online.de
Telephone: +49-6152-56768 Fax: +49-6152-56742
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Abstract

Flavonoids, secondary plant products which could be essential for normal physiology in humans and animals, may be the vitamins of the next century. Flavonoids belong to the polyphenols and possess antioxidative, anti-inflammatory, antimutagenic and anticarcinogenic properties. Among the various flavonoid species, tea flavonoids such as apigenin (from camomile) and epigallocatechin gallate (EGCG from green tea) can be used for the prevention of intestinal neoplasia, especially for adenoma and cancer prevention in the gastrointestinal tract. Numerous experimental studies with molecular and biological end points support the therapeutic efficacy of bioflavonoids. Clinical studies with cohorts and case-control trials suggest that flavonoids are effective in tertiary bioprevention but, as yet, there are no controlled randomized clinical trials. Flavonoids can inhibit inflammatory pathways and could be useful for chronic inflammatory bowel diseases. Flavonoid deficiency syndromes could be therapeutic targets in the future.

Key words: Flavonoids; Intestinal neoplasia; Cancer prevention; Apigenin; Epigallocatechin gallate; Inhibition of colon cancer cells

Peer reviewer: Seung Joon Baek, PhD, Associate Professor, Department of Pathobiology, College of Veterinary Medicine, The University of Tennessee, 2407 River Drive, Rm A228, Knoxville, TN 37996, United States

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INTRODUCTION

At a conference of nutritional science a few years ago, it was suggested that flavonoids could become the vitamins of the next century. At that time I thought that this is an outrageous claim. However, since then there is mounting evidence that flavonoids could fulfil this promise.

FLAVONOID SOURCES

Flavonoids are secondary plant metabolites that are ubiquitous in fruits, vegetables, nuts, seeds and as plants, are parts of human nutrition[1,2]. Some of them exhibit a broad spectrum of physiological and pharmacological properties such as antioxidant, radical-scavenging, anti-inflammatory, anti-viral and -bacterial, anti-atherogenic and anti-carcinogenic activities[3]. It is reported that human intake of all flavonoids is a few hundred milligrams to 650 mg/d in our western diet. Their entry into the body takes place *via* the gastrointestinal tract and therefore this organ and especially the epithelial lining cells are exposed to fairly high concentrations of flavonoids[4].

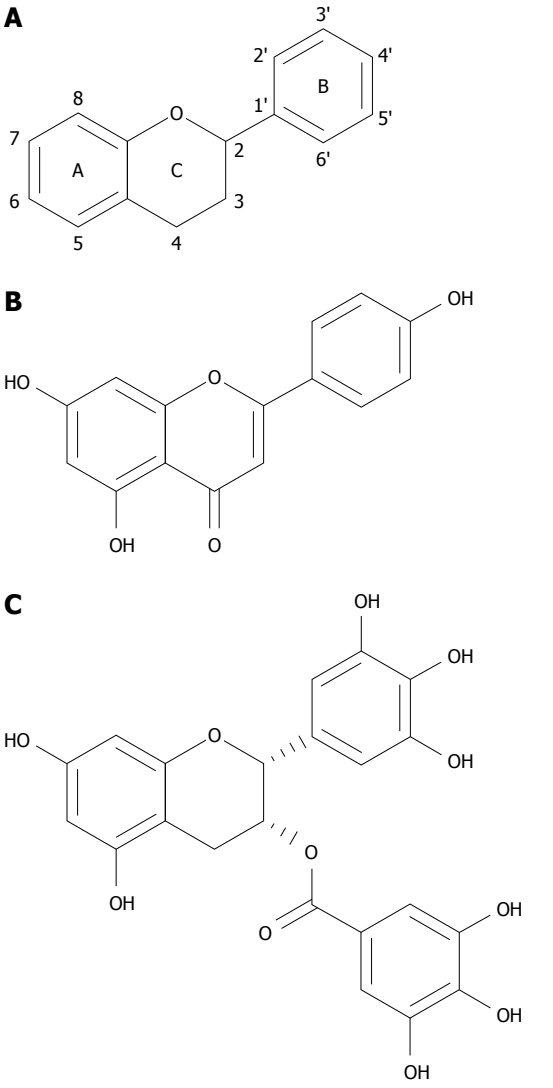

Figure 1 Chemical structure of flavonoids (A), apigenin (B) and epigallocatechin gallate (C).

There is some controversy about the bioavailability of nutritional flavonoids and about which type of flavonoids will be taken up and absorbed from the average food that we consume. Data on bioavailability vary wildly depending on many factors including the source, the specific molecular species, the presence of fat and the degree of food processing. Due to modern food technology, the flavonoid content of food is diminished, as can be shown in chocolate[5] and apple juice[6].

The most abundant flavonoids from nature originate from fruits (apples, onions and citrus fruits), teas (camomile, green tea), vegetables (cabbage, broccoli), seasonings (parsley, celery) and berries. These products of nature contain flavons (apigenin, luteolin), flavonols (quercetin, kaempferol), flavanols (epigallocatechin-3-gallate- EGCG), flavanons (naringenin, hesperidin) and isoflavons (genistein from soja) as well as anthocyanidins (from berries). The basic chemical structure of flavonoids is illustrated in Figure 1A and that of two of its major species (apigenin and EGCG) in Figure 1B and C.

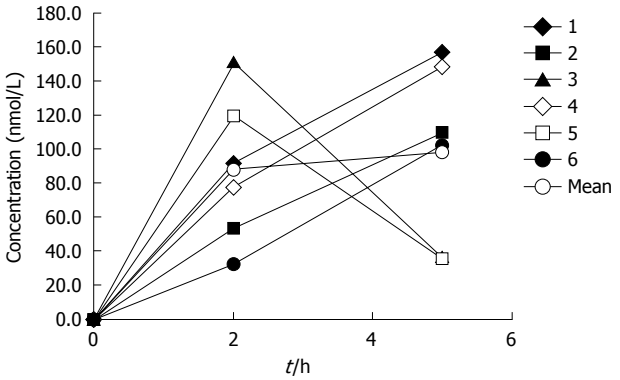

Figure 2 Serum levels of apigenin of six volunteers after ingestion of 5 flavonoid tablets over 5 h.

FLAVONOID SUPPLEMENTATION

Epidemiological studies have indicated decreased cancer occurrence in people who regularly drink green tea and, conversely, low intake could be associated with an enhanced risk of cancer[4]. Several constituents, including apigenin 7-*O*-glucoside and EGCG which accounts for about 60%-70% of total catechins, have been studied with respect to their anti-cancerogenic activities and it appears that these substances could be the most potent compounds in tea for inhibition of proliferation and induction of apoptosis of cancer cells[7]. Thus, regarding their safety, flavonoids are likely to have a potential value in preventive and therapeutic roles in cancerogenic conditions. Apigenin, a flavonoid present in camomile tea, parsley and celery, is a unique flavonoid because it is not detectable in the blood of individuals with a conventional western diet while other compounds like EGCG reach the central compartment and can be found in the serum[8]. We measured apigenin levels (50-150 nmol/L) after ingestion of 5 flavonoid tablets (Figure 2), each containing a mixture of tea flavonoids including apigenin (10 mg), EGCG (10 mg) and other tea flavonoids[9].

The results indicate that the absorption of food flavonoids from a conventional western diet is not sufficient to provide detectable and quantifiable apigenin levels. However, measurable exposure to at least apigenin can be found on nutritional supplements such as extracts from tea plants (camomile, green tea -"Flavo-Natin"®).

EXPERIMENTAL STUDIES

Increasingly, epidemiological and experimental studies demonstrate that modulation of the carcinogenic response by natural phytochemicals plays an important role in the prevention, mitigation and treatment of many diseases, especially in colorectal cancer (CRC)[10]. Exposure of colon-cancer cell lines (Caco-2 and HT-29 cell lines) to apigenin and EGCG from the nutritional supplement (Flavo-Natin®) leads to a significant inhibition of tumor cell proliferation *in vitro* (Figure 3).

Numerous *in vitro* studies elucidated the beneficial

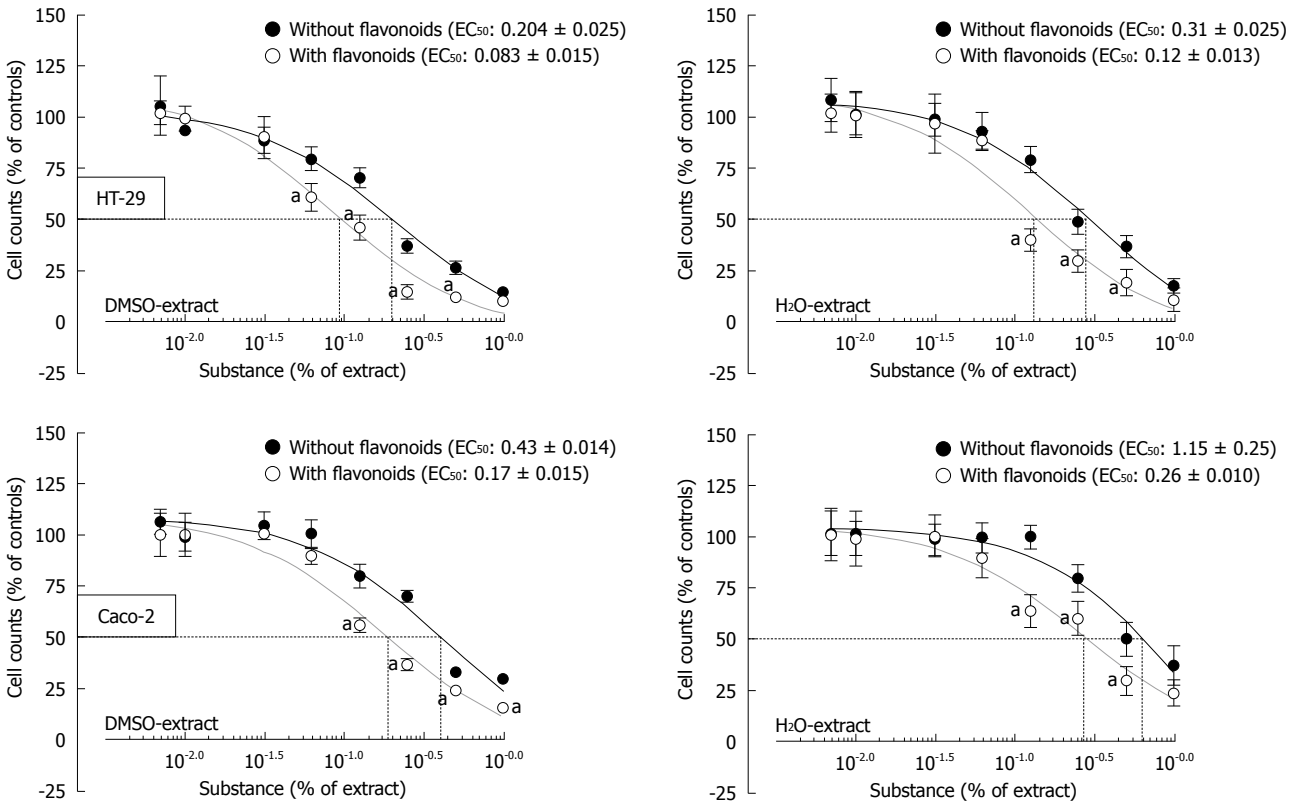

Figure 3 Inhibition of proliferation of Caco-2 cells and HT-29 cells *in vitro* by tea flavonoids used as nutritional supplement. a: significantly different; Grey line: Verum (content of tablets plus matrix); Black line: Placebo (tablet matrix only).

molecular biological effects of flavonoids among which the inhibition of the Wnt/β-cathenin and nuclear factor κB pathways, inhibition of hypoxia-inducible factor-1α and vascular endothelial growth factor expression by blocking phosphatidylinositol-3-kinase/AKT signalling are the most important targets[11]. Moreover, studies with biological end points revealed that flavonoids can suppress neoangiogenesis and increase apoptosis resulting in anti-carcinogenic and anti-mutagenic effects[12].

CLINICAL EVIDENCE

Clinical studies such as cohort and case-control trials suggest that increasing dietary flavonoids can be associated with a lower incidence and prevalence of CRC and colon adenomas. EGCG, flavons and flavonols can prevent growth of early forms of neoplasia such as adenomatous polyps and could be used for biological prevention[13,14]. Flavonoid treatment of at risk patients has the potential for tumor suppression in the gastrointestinal tract, as was shown for post surgical patients with resected CRC[15]. Tertiary bioprevention with flavonoids seems to be an appropriate target for improving the outcome of CRC survivors. Beside epidemiological studies, we need controlled randomized clinical trials to answer the question of whether flavonoids are useful for neoplasia prevention. It is unlikely that flavonoids are capable of treating advanced forms of neoplasia.

NEW APPROACHES

There is a great interest in new approaches to prevent and inhibit cancers of the pancreas, the prostate gland and the urinary tract using flavonoids. Some evidence indicates that flavonoids could be a new way to suppress these types of cancer.

Flavonoids belong to the polyphenols and possess anti-inflammatory and anti-oxidative properties. For this reason, they are strong candidates for a new treatment option for patients with chronic inflammatory bowel diseases such as Crohn's disease (CD) and ulcerative colitis (UC). In CD and UC there is a sustained over expression of inflammatory cytokines and chemokines, as well as an accumulation of chronically activated inflammatory cells like lymphocytes and macrophages. Flavonoids can inhibit inflammatory pathways *in vitro* and in animal experiments[16]. Clinical pilot trials with these agents should be performed to assess their clinical activities in CD and UC since these botanicals are without relevant side effects compared to biological agents currently in use.

OUTLOOK

There might be a deficiency of certain flavonoids in our food due to insufficient intake of fruits and vegetables, the methods of industrialized agriculture and the modes of nutritional preservation and storage. Particularly, chemically defined forms of enteral and parenteral nutri-

tion are deficient in flavonoids and other polyphenols[17]. Deficiencies of these compounds and other plant agents could be responsible for derailing the healthy steady state within and outside exposed epithelial cells. Without certain secondary plant metabolites, the microenvironment is disturbed by exogenous and endogenous toxins leading to a proinflammatory and mutagenic milieu with ensuing DNA damage. Neoplastic cells can proliferate in a milieu with inflammatory cytokines, free oxygen species and other radicals. Flavonoids could act as protective agents and block this unfavourable environment by inhibition of mutagenic and carcinogenic pathways. Deprived of these stimuli, neoplastic cells could cease to proliferate. Instead of trying to destroy the neoplastic cells by chemotherapy, secondary plant metabolites such as flavonoids could possibly be used to stabilize the extracellular environment to prevent enhanced carcinogenic cell proliferation.

Plants have developed antioxidative mechanisms for their evolution and to maintain their health. Like vitamins, flavonoids could play an emerging role for human health, cancer prevention and well being.

The first challenge in the future will be to define degenerative diseases for which flavonoids might be useful. This concept could apply to intestinal neoplasia and chronic inflammatory bowel diseases, as well as to neurodegenerative and chronic cardiovascular disorders. The second challenge will be to develop specific flavonoid preparations to deliver these compounds to the target structures and their receptors. If this can be accomplished, it may be possible to use flavonoids as tools for prevention and treatment, much like the vitamins did in the past.

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CASE REPORT

Pedunculated gastric tube interposition in an esophageal cancer patient with prepyloric adenocarcinoma

Tatsuo Kanda, Yu Sato, Kazuhito Yajima, Shin-ichi Kosugi, Atsushi Matsuki, Takashi Ishikawa, Takeo Bamba, Hajime Umezu, Tsutomu Suzuki, Katsuyoshi Hatakeyama

Tatsuo Kanda, Yu Sato, Kazuhito Yajima, Shin-ichi Kosugi, Atsushi Matsuki, Takashi Ishikawa, Takeo Bamba, Katsuyoshi Hatakeyama, Division of Digestive and General Surgery, Niigata University Graduate School of Medical and Dental Sciences, 1-757 Asahimachi-dori, Niigata City 951-8510, Japan
Hajime Umezu, Division of Cellular and Molecular Pathology, Niigata University Graduate School of Medical and Dental Sciences, 1-757 Asahimachi-dori, Niigata City 951-8510, Japan
Tsutomu Suzuki, Department of Nursing, School of Health Science, Niigata University, 2-746 Asahimachi-dori, Niigata City 951-8518, Japan
Author contributions: Kanda T, Yajima K, Kosugi S and Ishikawa T performed surgery in this case; Kanda T, Kosugi S and Suzuki T designed the research; Kanda T, Sato S, Yajima K and Matsuki A drafted the manuscript; Bamba T and Umezu H were responsible for the pathological examinations; Suzuki T and Hatakeyama K revised the implementation of this work and reviewed the manuscript.
Correspondence to: Tatsuo Kanda, MD, PhD, Division of Digestive and General Surgery, Niigata University Graduate School of Medical and Dental Sciences, 1-757 Asahimachi-dori, Niigata City 951-8510, Japan. kandat@med.niigata-u.ac.jp
Telephone: +81-25-2272228 Fax: +81-25-2270779
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Abstract

Gastric carcinoma is one of the malignancies that are most frequently associated with esophageal carcinoma. We describe herein our device for advanced esophageal cancer associated with early gastric cancer in the antrum. A 57-year-old man presenting with dysphagia and upper abdominal pain was admitted to our hospital. Preoperative examinations revealed locally advanced squamous cell carcinoma (SCC) of the middle thoracic esophagus (T3N0M0 Stage ⅡA) and mucosal signet-ring cell carcinoma of the gastric antrum (T1N0M0 Stage ⅠA). Although the gastric tumor appeared to be an intramucosal carcinoma, its margin was obscure, so endoscopic *en-bloc* resection was considered inadequate. We chose surgical resection of the gastric tumor as well as the esophageal SCC after neoadjuvant chemotherapy with 5-fluorouracil and cisplatin for advanced esophageal cancer. Following transthoracic esophagectomy with three-field lymph node dissection, the gastric carcinoma was removed by gastric antrectomy, which preserved the right gastroepiploic vessels, and a pedunculated short gastric tube was used as the esophageal substitute. Twenty-eight months after the surgery, the patient is well with no evidence of cancer recurrence. Because it minimizes surgical stress and organ sacrifice, gastric tube interposition is a potentially useful technique for esophageal cancer associated with localized early gastric cancer.

Key words: Antrectomy; Early gastric cancer; Esophageal cancer; Esophageal reconstruction; Gastric tube

Peer reviewer: Markku Voutilainen, MD, Department of Internal Medicine, Central Hospital Central Finland, Keskusairaalantie 19, Jyväskylä FIN-40620, Finland

Kanda T, Sato Y, Yajima K, Kosugi S, Matsuki A, Ishikawa T, Bamba T, Umezu H, Suzuki T, Hatakeyama K. Pedunculated gastric tube interposition in an esophageal cancer patient with prepyloric adenocarcinoma. *World J Gastrointest Oncol* 2011; 3(5): 75-78 Available from: URL: http://www.wjgnet.com/1948-5204/full/v3/i5/75.htm DOI: http://dx.doi.org/10.4251/wjgo.v3.i5.75

INTRODUCTION

Esophageal reconstruction with a gastric tube is a well-established surgical procedure following radical esophagectomy for carcinoma of the thoracic esophagus. In cases where the stomach is unavailable, for example, patients with a history of gastrectomy, concurrent gastric diseases,

or tumor involvement of the stomach, a colonic conduit is preferentially selected as an esophageal substitute because a long segment can be harvested and brought up to the neck without microvascular surgery[1]. However, this procedure is more surgically invasive than gastric pull-up and the functional loss of both stomach and colon may potentially deteriorate patient's nutritional status.

Gastric adenocarcinoma is the second greatest cause of cancer death in the world[2]. Furthermore, it is second most common malignancy associated with esophageal carcinoma[3,4]. Thus, esophageal cancer associated with gastric adenocarcinoma is not exceptionally rare, particularly in East Asia where both malignancies are more common than in western countries[5].

We present herein details of surgical treatment of a patient with locally advanced squamous cell carcinoma (SCC) of the mid-thoracic esophagus and early gastric adenocarcinoma of the prepyloric region. The patient underwent radical esophagectomy with three-field lymph node dissection and the stomach tumor was excised by gastric antrectomy, which preserved the right gastroepiploic artery and vein. The remaining pedunculated short gastric tube was used as the esophageal substitute in conjunction with Roux-en-Y gastrojejunostomy.

CASE REPORT

In November 2008, a 57-year-old man presenting with dysphagia was diagnosed with locally advanced SCC of the mid-thoracic esophagus by endoscopic examination of the upper gastrointestinal tract. The endoscopic examination simultaneously revealed the coexistence of an irregular depressed lesion in the prepylorus (Figure 1). Biopsy specimens taken from the gastric lesion showed poorly differentiated adenocarcinoma with signet ring carcinoma cells (Figure 2). Computed tomography showed neither distant metastasis nor lymph node metastasis to the mediastinum or the perigastric area. A clinical diagnosis of double cancer was made - locally advanced esophageal SCC (T3N0M0 Stage ⅡA) and early gastric adenocarcinoma of the prepylorus (T1N0M0 Stage ⅠA). Further endoscopic examination was carried out to look into the possibility of endoscopic treatment of the gastric tumor. Although the gastric tumor was endoscopically estimated to be an intramucosal carcinoma unassociated with ulcer scar, it was histologically a signet-ring cell carcinoma and the tumor boundary was poorly defined. As wide resection with the endoscopic submucosal dissection (ESD) technique would cause stenosis of the gastric outlet[6,7], we selected surgical resection of the gastric tumor.

Surgery

The surgery consisted of transthoracic radical esophagectomy with three-field dissection and resection of the pyloroantral region of the stomach. As an esophageal substitute, we selected a short gastric tube using the right gastroepiploic artery as a vascular pedicle, considering the low incidence of lymph node metastasis from gastric carcinoma. The surgery was performed 26 d after neoadjuvant chemotherapy with 5-fluorouracil and cisplatin for advanced esophageal cancer. After completion of the radical esophagectomy, the stomach was mobilized as for the construction of a conventional gastric tube. No lymph node metastasis was found macroscopically along the gastroepiploic arteries. The branches of the right gastroepiploic vessels were carefully cut at 7 cm distal from the pylorus (Figure 3A). This procedure was extended downward to the pylorus. The duodenum was divided immediately distal to the pyloric ring and the duodenal stump was closed (Figure 3B). The distal part of the gastric tube was resected along with suprapyloric lymph nodes (Figure 3C). The distal stump of the gastric tube was anastomosed to the upper jejunum for a Roux-en-Y reconstruction (Figure 3D). The gastric tube was pulled up through the posterior mediastinum and anastomosed to the cervical esophagus. The cervical anastomosis was easy because separation from the duodenum increased the flexibility of the pedunculated short gastric tube. The operation time was 630 min and blood loss was 635 mL.

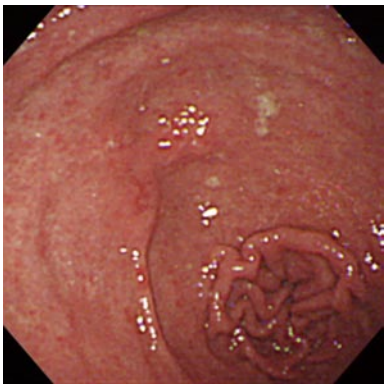

Figure 1 Endoscopic finding of gastric adenocarcinoma. Gastrofiberscopy revealed an irregular depressed lesion with an obscure boundary in the lesser curvature of the prepyloric region.

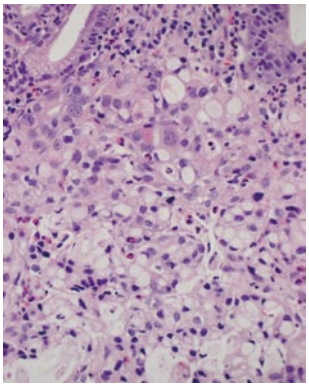

Figure 2 Histological finding of biopsy specimens. Histological examination of biopsy specimens taken from the gastric lesion showed poorly differentiated adenocarcinoma that partially exhibited signet-ring cell morphology (Hematoxylin-and-eosin; original magnification, × 400).

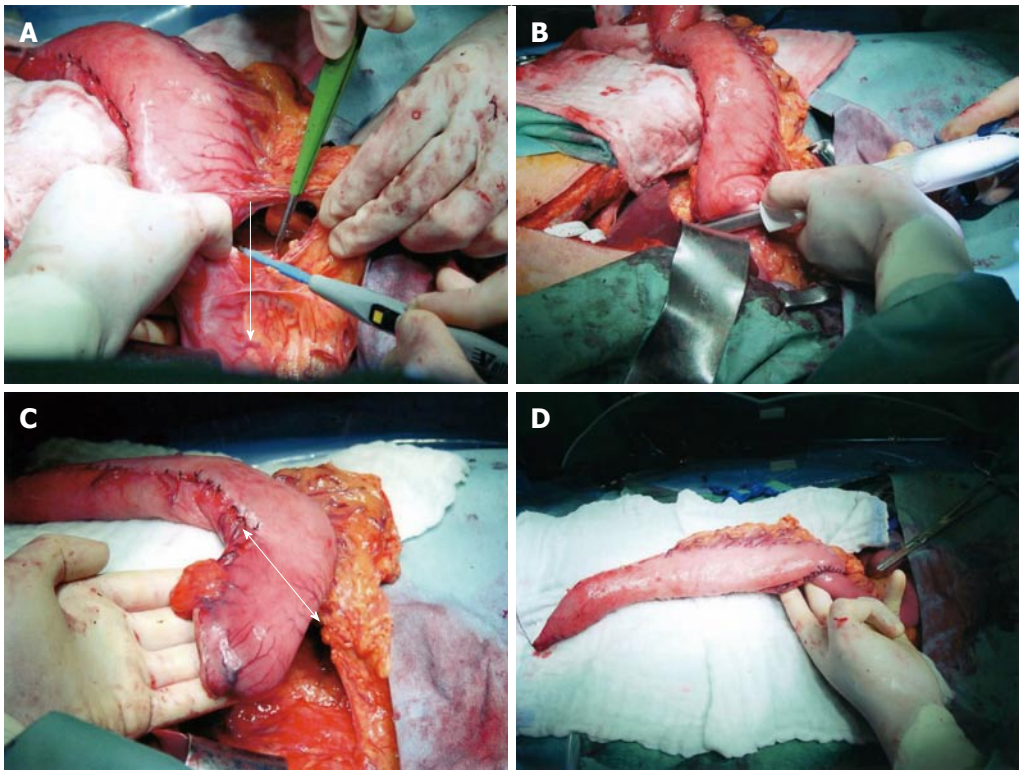

Figure 3 Intraoperative findings. After a conventional gastric tube was prepared, the branches of the right gastroepiploic vessels supplying the pyloric lesion were cut along the greater curvature: The arrow line indicates the direction and range of devascularization (A). The duodenum was divided immediately distal to the pyloric ring and the duodenal stump was closed (B). The distal part of the gastric tube was resected to remove the early gastric adenocarcinoma of the prepyloric region and the suprapyloric lymph nodes: the arrow line indicates the cutting line for antrectomy (C). The distal stump of the gastric tube was anastomosed to the upper jejunum for a Roux-en-Y reconstruction prior to gastric pull-up to the neck (D).

Pathology and postoperative course

Pathological examination of surgical specimens revealed that the esophageal tumor was a well-differentiated SCC that had spread to the esophageal adventitia in depth (pT3) and was associated with no lymph node metastasis. The gastric tumor was a moderately differentiated adenocarcinoma with a mucinous component and was localized in the mucosa. No vascular or lymphatic invasion was found. Pathological examination revealed a gastric tumor measuring 1.5 cm × 1.0 cm in size and complete tumor-free margins. No lymph node metastasis was found in the suprapyloric lymph nodes dissected.

The patient suffered from minor leakage from the esophagogastrostomy but soon recovered non-surgically. He was discharged on the 39th postoperative day. At the time of writing, i.e. 28 mo after the surgery, the patient is well with no evidence of cancer recurrence.

DISCUSSION

It is not uncommon that patients with esophageal carcinoma suffer from other malignancies synchronously and/or metachronously. According to the Comprehensive Registry of Esophageal Cancer in Japan[8], 18.5% of patients with esophageal carcinoma simultaneously had multiple primary cancers, in which gastric carcinoma was the most common (4.7%), followed by head and neck cancer (2.7%), colorectal carcinoma (1.2%), and lung cancer (0.7%). These data suggest the need to devise an adequate treatment plan for patients having double cancer. In the case of combination with gastric carcinoma, reconstruction following radical esophagectomy is problematic because the conventionally used gastric pull-up is unavailable. Recently, endoscopic mucosal resection has become the standard treatment for gastric intramucosal carcinoma with a differentiated morphology. The development of ESD, in which the mucosa containing tumors is dissected along the submucosal layer, allows for *en-bloc* resection of large tumors and extends the application of endoscopic treatment to early gastric cancer with a very low risk of regional lymph node metastasis[9]. Endoscopic resection would potentially have been suitable for our case. However, the tumor in the present case was determined to be a small intramucosal carcinoma whose boundary could not be identified. Furthermore, it was located in the prepyloric region. Based on these conditions, the gastrointestinal cancer board in our hospital, which consists of esophagogastric surgeons, gastrointestinal endoscopists, and radiologists, determined that endoscopic resection should be avoided because it had a high risk of incomplete resection and/or stenosis following wide resection.

Interposition with a pedunculated gastric tube in a Roux-en-Y fashion was reported for the first time by Yamagishi *et al*[10] in 1970. They used this technique originally for the bypass surgery of advanced esophageal cancer, and then

extended it to normal esophageal reconstruction following radical esophagectomy, noticing that this technique improved gastric tube flexibility and enabled safe anastomosis at the lower level of the gastric tube where blood flow is rich. Hanyu *et al*[11] applied the gastric tube interposition to patients with esophageal carcinoma associated with early carcinoma that was located in the lesser curvature of the stomach. They presented this as a procedure that keeps the radicality of the regional lymph node dissection for coexisting early gastric cancer. In their procedure, tumors at the gastric angle were excised in the process of making a short gastric tube using a part of the greater curvature of the stomach. In 2006, Motoyama *et al*[12] reported distal gastrectomy with preservation of the gastroepiploic artery in two cases of adenocarcinoma that metachronously occurred in a gastric tube following esophagectomy. The surgery in the present case is essentially the synchronous counterpart of that of Motoyama *et al*[12].

In the present case, we omitted the dissection of lymph nodes along the right gastroepiploic artery, station nos. 4d and 6 in the Japanese Classification of Gastric Carcinoma[13], in which lymph node metastasis is frequently found in the carcinoma of the lower third of the stomach. However, there are data showing that the incidence of lymph node metastasis from an intramucosal carcinoma without ulcer scar was approximately 4% even if the tumor was morphologically classified as the diffuse type[14]. The esophageal cancer in the present case was diagnosed, both clinically and pathologically, as Stage ⅡA. The 5-year survival rate of patients with esophageal cancer at Stage ⅡA is reported to be 52%-84%[8,15-17] and approximately one third of the patients show recurrence despite undergoing radical esophagectomy with extended lymphadenectomy. Thus, the risk of lymph node recurrence after local resection of early gastric cancer is acceptable, compared with the prognosis of patients with advanced esophageal cancer. Our treatment choice offers certain benefits to the patient, including low invasiveness and good nutritional status although we need to conduct continuous follow-up to determine whether or not our treatment choice is indeed warranted.

The indication of the technique presented here is limited to tumors located in the distal stomach, early stage carcinomas with a low risk of lymph node metastasis, and widely spreading tumors unmanageable by endoscopic resection. However, pedunculated gastric tube interposition combined with gastric antrectomy is a minimally invasive surgical technique that minimizes organ sacrifice. Clinicians involved in esophagogastric cancer treatment should consider this technique as a possible alternative to colonic interposition following radical gastrectomy for gastric carcinoma associated with advanced esophageal cancer.

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Events Calendar 2011

January 20-22, 2011
Gastrointestinal Cancers Symposium 2011, San Francisco, CA, United States

January 27-28, 2011
Falk Workshop, Liver and Immunology, Medical University, Regensburg, Germany

February 17-20, 2011
APASL 2011-The 21st Conference of the Asian Pacific Association for the Study of the Liver, Bangkok, Thailand

February 21-21, 2011
International Conference on Modern Cancer Management-Joint Symposium, Abuja, Nigeria,

February 26-March 1, 2011
Canadian Digestive Diseases Week, Westin Bayshore, Vancouver, British Columbia, Canada

March 11-12, 2011
First Integrative Care for the Future: The future of cancer care, Arnhem, The Netherlands
http://www.integrativecareftfuture.org/

March 14-17, 2011
British Society of Gastroenterology Annual Meeting 2011, Birmingham, England, United Kingdom

March 24-25, 2011
Advanced Cancer Course "International Clinical Trials Workshop", Punta del Este, Uruguay

April 6-7, 2011
IBS-A Global Perspective, Milwaukee, WI, United States

April 6-8, 2011
Third Latin American Symposium on Gastrointestinal Oncology-Chilean Foundation for Oncology Development Joint Symposium, Vina Del Mar, Chile

April 15-16, 2011
Falk Symposium 177, Endoscopy Live Berlin 2011 Intestinal Disease Meeting, Maritim Hotel Berlin, Stauffenbergstr. 26, 10785 Berlin, Germany

April 20-23, 2011
9th International Gastric Cancer Congress, COEX, World Trade Center, Samseong-dong, Gangnam-gu, Seoul 135-731, South Korea

May 8-12, 2011
ESTRO International Oncology Forum, London, United Kingdom

May 19-22, 2011
1st World Congress on Controversies in the Management of Viral Hepatitis (C-Hep), Palau de Congressos de Catalunya, Barcelona, Spain

May 25–27, 2011
9th CIMT Annual Meeting, Targeting Cancer, Road-Maps for Success, Mainz, Germany

May 25-28, 2011
4th Congress of the Gastroenterology Association of Bosnia and Herzegovina with international participation, Sarajevo, Bosnia and Herzegovina

June 3-7, 2011
2011 ASCO Annual Meeting, Chicago, IL, United States

June 18-24, 2011
13th Joint ECCO-AACR-EORTC-ESMO Workshop on "Methods in Clinical Cancer Research", Flims, Switzerland

June 22-25, 2011
ESMO 13th World Congress on Gastrointestinal Cancer, Barcelona, Spain

July 9-10, 2011
Best of ASCO China, Hengzhou, China

July 21-23, 2011
ASCO-JSMO Joint Symposium, Yokohama, Japan

August 25-28, 2011
VII Peruvian Congress SPOM: Toward personalized Oncology-Endorsement, Lima, Peru

September 2-3, 2011
Falk Symposium 178, Diverticular Disease, A Fresh Approach to a Neglected Disease, Martinstr. 29-37, 50667 Cologne, Germany

September 10-14, 2011
ICE 2011-International Congress of Endoscopy, Los Angeles Convention Center, 1201 South Figueroa Street, Los Angeles, CA, United States

September 15-17, 2011
2011 Gastrointestinal Oncology Conference, Sheraton Crystal City, Arlington, VA, United States

September 30-October 1, 2011
Falk Symposium 179, Revisiting IBD Management: Dogmas to be Challenged, Place Rogier 3, 1210 Brussels, Belgium, Germany

October 6-7, 2011
IV InterAmerican Oncology Conference: Current Status and Future of Anti-Cancer Targeted Therapies, Buenos Aires, Argentina

October 14-15, 2011
New Trends in the Medical Treatment of Solid Malignancy-Romanian Society for Medical Oncology Joint Symposium, Bucharest, Romania

October 27-29, 2011
EORTC-NCI-ASCO Annual Meeting on Molecular Markers in Cancer, Brussels, Belgium

November 11-12, 2011
Falk Symposium 180, IBD 2011: Progress and Future for Lifelong Management, 1-12-33 Akasaka, Minato-ku, Tokyo 107-0052, Japan

November 30-December 3, 2011
8th International Cancer Conference "Entering the 21st Century for Cancer Control in Africa"-African Organization for Research and Training in Cancer Joint Symposium, Cairo, Egypt

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Macaulay Onuigbo, MD, MSc FWACP FASN, Department of nephrology, Midelfort clinic, Mayo Health System, Eau Claire, WI 54702, United States

Yo-ichi Yamashita, MD, PhD, Department of Surgery, Hiroshima Red Cross Hospital and Atomic Bomb Survivors Hospital, Senda-machi 1-9-6, Naka-ku, Hiroshima 730-8619, Japan

Runjan Chetty, Professor, Department of Pathology and Gene Regulation, University of Glasgow, Western Infirmary (Pathology), Dumbarton Road, Glasgow, G11 6NT, Scotland, United Kingdom

Xiao-Chun Xu, Associate Professor, Department of Clinical Cancer Prevention, The University of Texas M. D. Anderson Cancer Center, 1515 Holcombe Boulevard, Unit 1360, Houston, TX 77030, United States

Kwang-Huei Lin, PhD, Professor, Associate Dean, College of Medicine, Chang-Gung University, 259 Wen-Hwa 1st Road, Taoyuan 333, Taiwan, China

Barbara W Chwirot, Professor, Department of Medical Biology, Institute of General and Molecular Biology, Nicolaus Copernicus University, Gagarina 9, Torun 87-100, Poland

Jian-Kun Hu, MD, PhD, Associate Professor, Department of Gastrointestinal Surgery, West China Hospital, Sichuan University, Chengdu 610041, Sichuan Province, China

Antonio Russo, MD, PhD, Associate Professor, Genetic and Molecular Oncology Unit, Interdepartmental Center of Research in Clinical Oncology, School of Medicine, University of Palermo, Via del Vespro 127-90127 Palermo, Italy

Goran Stanojevic, MD, PhD, Proffesor, Department of Surgery, Clinical Centre Nis, Bul Zorana Djindjica 48, 18000 Nis, Serbia

Fahd Al-Mulla, PhD, Associate Professor, Department of Molecular Pathology, Kuwait University, Faculty of Medicine, Safat 13110, Kuwait

Ioannis A Voutsadakis, MD, PhD, Department of Medical Oncology, University Hospital of Larissa, Larissa 41110, Greece

John Griniatsos, MD, Assistant Professor, Department of Surgery, University of Athens, Medical School, 1st LAIKO Hospital, 17 Agiou Thoma str, GR 115-27, Athens, Greece

Seung Joon Baek, PhD, Associate Professor, Department of Pathobiology, College of Veterinary Medicine, The University of Tennessee, 2407 River Drive, Rm A228, Knoxville, TN 37996, United States

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Acknowledgments

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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]

Issue with no volume

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]

Books

Personal author(s)

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

Chapter in a book (list all authors)

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

Author(s) and editor(s)

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

Conference paper

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

Electronic journal (list all authors)

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

Statistical data

Write as mean ± SD or mean ± SE.

Statistical expression

Express *t* test as *t* (in italics), *F* test as *F* (in italics), chi square test as χ^2 (in Greek), related coefficient as *r* (in italics), degree of freedom as υ (in Greek), sample number as *n* (in italics), and probability as *P* (in italics).

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Quantities: *t* time or temperature, *c* concentration, *A* area, *l* length,

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