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1. RAPD Objectives and characteristics: The Revista Andaluza de Patología Digestiva is the official publication of the Andalusian Society of Digestive Pathology (SAPD), which since 2007 has been published in electronic format only, under the name RAPD Online. Its purpose is to disseminate all epidemiological, clinical, basic and sociological aspects of digestive diseases, through the contributions sent to the journal from Andalusia and from the entire scientific community. The official language for the publication of this journal is Spanish, but some contributions may be accepted in the author's original language in English, French or Italian. RAPD Online is published bimonthly, with one of the issues being specially dedicated to the Annual Meeting of the SAPD and the Editorial Board deciding to reserve one or more issues per year for the monographic development of a topic related to the speciality.

All submitted contributions must be original and not be simultaneously under review for publication in another journal. The publication of abstracts or posters is not considered duplicate publication. Manuscripts will be evaluated by expert reviewers, appointed by the editorial board, before being accepted for publication, in a process that will take less than 30 days.

2. RAPD Contents: *regular numbers of RAPD Online include defined sections such as:*

- *Original articles on clinical or basic research.*
- *Thematic reviews on specific aspects of Gastroenterology.*
- *Consensus documents.*
- *Clinical cases.*
- *Clinical cases with videos or Videoforum.*
- *Images of the month.*
- *News and updates on gastroenterology and hepatology.*
- *Letters to the Editor.*

Other contributions that are considered of interest by the Editorial Board, relating to different aspects of clinical practice in the recent past, biographical comments, or other contents of a cultural nature, or related to scientific activities in any territorial area, will be inserted in RAPD Online in sections designed specifically for this purpose.

3.Submission of manuscripts: *The preferred way to submit manuscripts is through the SAPD website (https://www.sapd. es), by accessing the RAPD Online page and clicking on the "Submit an original" button located on the same access page to the journal. This will take you to the Manuscript Centre, from where you will be able to send manuscripts and all the required documentation. To use this tool you must be previously registered, access requires a username and password. If you are a member of the SAPD, you can use your usual username. If you are not a member, you can request a username for access to the Manuscript Centre using the form on the website. You can write to sulime@sulime.net or RAPDonline@sapd.es, for the solution of any problem in the submission of manuscripts.*

4. Writing standard for manuscripts: *monographic numbers, thematic reviews, updates and annotated articles will be commissioned by the Editorial Board, but the submission of any of these contributions at the request of an author will be considered by the RAPD Online Management and evaluated with great interest for inclusion in the journal.*

All manuscripts will be subject to specific rules, depending on the type of contribution, and to common ethical and legal standards.

A) Specific standard for manuscripts writing

They refer to the recommended length and structure of each type of manuscript. As a basic unit of length for the text, in any of the contributions, a page of 30-31 lines, spaced 1.5 lines apart, with a font size of 12, with 75-80 characters without spaces per line and a total of 400-450 words per page is considered. Texts should be sent spell-checked and in editable format in all their applications (main text, figures, legends or figure captions, tables, graphs, drawings).

Originals: *originals can be up to 12 pages long (5,100 words), excluding bibliographical references and captions to figures and tables. It is not advisable to insert more than 10 images, including tables and figures. Colour illustrations and videos will not represent an economic charge for the authors, but the insertion of videos, for technical reasons, will be previously agreed with the editor. However, the editing method of RAPD Online allows, in specific cases, the acceptance of longer manuscripts, or the inclusion of a greater number of images, provided that the characteristics of the material presented so require. It is not advisable to have more than 9 authors, except in the case of collaborative works. In these originals, the first nine participants will be listed at the head of the paper and the rest of the participants will be listed at the end of the first page of the manuscript.*

Through the Manuscript Centre, the following information will be required for the submission of an original:

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- Main body of the manuscript, containing:

1° Structured abstract in Spanish (optional also in English) and 3-5 keywords. The abstract will have a maximum length of 250 words and should be structured as follows:

a) Introduction and objectives
b) Material and methods
c) Results
d) Conclusions

2° List of abbreviations used in the text.

3° Text: it will include the following sections:
a) Introduction
b) Material and methods
c) Results

d) Discussion
e) Conclusions; each of them appropriately headed.

4° Bibliography: according to the specifications established in the group of common standards (See common standards and other supporting documents).

5° Acknowledgements.

6° Figure captions.

7° Tables and figures in text.

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5° Declaration on the existence or non-existence of a source of funding for the work, or conflicts of interest.

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3° Bibliography: According to the specifications established in the group of common standards (See common standards and other supporting documents).
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1° Structured abstract in Spanish and English. 3-5 key words. The abstract will have a maximum length of 350 words, emphasising the most important aspects of the manuscript.
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Clinical Cases: *the manuscripts included in this section will include 1-5 clinical cases, which due to their infrequent or unusual clinical behaviour, or because they provide some diagnostic or therapeutic novelty, deserve to be reported.*

The length of the texts in the Clinical Cases section should not exceed 5 pages (2,125 words), excluding bibliographical references and captions to figures and tables, and the number of inserted images should not exceed 5, including tables and figures. However, the RAPD Online editing method allows, in specific cases, the acceptance of longer manuscripts, or the inclusion of a greater number of images, provided that the characteristics of the material presented so require. Colour illustrations and videos will not represent a financial charge for authors, but the insertion of videos, for technical reasons, will be previously agreed with the editor. No more than 5 authors will be admitted, except in specific and reasoned cases.

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- Main body of the manuscript, containing:

1° Structured abstract in Spanish and English. 3-5 key words. The abstract will have a maximum length of 250 words.
2° Introduction. To present the clinical problem reported.
3° Description of the clinical case.
4° Discussion. To highlight the peculiarities of the case and its consequences.
5° Bibliography: According to the specifications established in the group of common standards (See common standards and other supporting documents).
6°Acknowledgements.7° Figure captions.
8° Tables and text figures.

Clinical Cases with Videos or Videoforum: *the manuscripts included in this section will include 1-5 clinical cases, which due to their infrequent or unusual clinical behaviour, or because they provide some diagnostic or therapeutic novelty, deserve to be communicated.*
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Videos should be submitted in AVI, MPEG, MP4 OR MOV format, and at a recommended high quality resolution (720p or 1080p). They must not contain personal data of the patients. It is recommended that they be edited to minimise editing time, which should not exceed 10 minutes. If the video includes sound, it must be processed in MP3 format. If the videos to be included are in other formats, please contact the publisher

to verify their validity. They should not exceed 2GB.
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4° Full postal address of the responsible author, to whom correspondence should be addressed, including telephone, fax and email address.

- Main body of the manuscript, containing:

1° Structured abstract in Spanish and English. 3-5 key words. The abstract will have a maximum length of 250 words.
2° Introduction. To present the clinical problem reported. 3° Description of the clinical case.
4° Discussion. To highlight the peculiarities of the case and its consequences.
5° Bibliography: According to the specifications established in the group of common standards (See common standards and other supporting documents).
6°Acknowledgements.7° Figure captions.
8° Tables and figures in text.
9° Videos.

Link tutorial videos: https://www.sapd.es/videoteca/varios/ tutoriales/

Images of the month: *the manuscripts included in this section can take two formats, depending on the authors' preference.*

- Format A. Images with educational value: these shall include images of any kind, clinical, radiological, endoscopic, anatomopathological, macro and microscopic, which contribute to postgraduate training and therefore deserve to be shown because of their peculiarity, or because they represent a characteristic example.
- Format B. Key images for a diagnosis: These will include images of any kind, clinical, radiological, endoscopic, anatomopathological, macro and microscopic, together with a summarised clinical history, which will provide the possible final diagnostic resolution. This will be presented in a separate section in the same issue of the journal.

The length of the texts in the Images of the Month section must not exceed 1 page (425 words) in the clinical approach to the image presented and 2 pages (850 words), excluding bibliographical references and captions to figures and tables, in the commentary on the image (Format A) or in the diagnostic resolution of the case (Format B). However, the RAPD Online editing method allows, in specific cases, the acceptance of longer manuscripts, or the inclusion of a greater number of images, provided that the characteristics of the material presented so require. Colour illustrations and videos will not represent a financial charge for authors, but the insertion of videos, for technical reasons, will be previously agreed with the editor. No more than 3 authors will be accepted, except in specific and reasoned cases.

Through the Manuscript Centre, and for the submission of an Image of the Month, the following information will be required:

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1° Full title of the work in Spanish and English.. 2° Surnames and first names of all authors.
3° Centre(s) of origin (department, institution, city and country).
4° Full postal address of the responsible author, to whom correspondence should be addressed, including telephone, fax and email address.
5° Type of Image of the Month format chosen.

-Main body of the manuscript, containing:

1° Structured abstract in Spanish and English and 3-5 key words. The abstract will have a maximum length of 250 words.
2° Description of the image.
3° Comments on the image.
4° Bibliography: According to the specifications established in the group of common standards (See common standards and other supporting documents).
5° Figure captions.

New developments and updates in gastroenterology and hepatology: this section will be devoted to commenting on the scientific and medical developments that have occurred in recent years in the speciality of Gastroenterology and Hepatology.

This section will systematically and periodically analyse all facets of the speciality.

Texts on " New developments in Gastroenterology" may be up to 5 pages long (2,125 words), excluding bibliographical references and captions to added figures and tables. In both cases the number of inserted images must not exceed 5, including tables and figures. However, the RAPD Online editing method allows,

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4° Full postal address of the responsible author, to whom correspondence should be addressed, including telephone, fax and email address.
5° Declaration on the existence or non-existence of a source of funding for the work, or conflicts of interest.

- Main body of the manuscript, containing:

1° Structured abstract in Spanish and English. 3-5 key words. The abstract will have a maximum length of 250 words.
2° Description of the bibliographic material analysed.
3° Critical comments on the results contained in the selected works.
4° Bibliography: According to the specifications established in the group of common standards (See common standards and other supporting documents). If two or more originals have been chosen for the analysis, it is advisable to divide the section into sections at the authors' discretion.
5° Figure captions.
6° Tables and Figures in text.

Letters to the Editor: *this section will be dedicated to comments on any manuscript published in RAPD Online. This section may also include comments of a more general nature, establishing the authors' own hypotheses and suggestions, within the scientific field of Gastroenterology. The length of the texts in this section of Letters to the Editor should not exceed 2 pages (850 words), including bibliographical references. Two figures or tables may be included and the number of authors should not exceed four.*

Through the Manuscript Centre, and for the submission of a Letter to the Editor, the following information will be required:

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2° Surnames and first names of all authors. It is advisable to place a hyphen between the first and second surname.
3° Centre(s) of origin (department, institution, city and country).
4° Full postal address of the responsible author, to whom correspondence should be addressed, including telephone, fax and e-mail address.
5° Declaration on the existence or non-existence of a source of funding for the work, or conflicts of interest.

- Basic body of the manuscript, containing:

1° Text of the manuscript.
2° Bibliography: According to the specifications set out in the common standards group (See common standards and other supporting documents).

B)Common standards and other supporting documents

This refers to the set of mandatory standards, both for uniformity in the presentation of manuscripts and for compliance with current legal regulations. In general, the style of manuscripts should follow the guidelines set out in the Vancouver Agreement of the International Committee of Medical Journal Editors. (http://www.ICMJE.org).

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- Units. Biochemical and haematological parameters shall be expressed in International Units (SI), except haemoglobin which shall be expressed in g/dL. Length, height and weight measurements shall be expressed in decimal metric units and temperatures in degrees Celsius. Blood pressure shall be measured in millimetres of mercury.
There is an aid for the conversion of non-international (non-SI) units into international (SI) units. (http://www.techexpo.com/techdata/techcntr.html).

- Generic names. The generic names of medicinal products, clinical instruments and tools and software shall be used. When a brand name is the subject of research, the brand name and the name of the manufacturer, city and country shall be included in parentheses the first time the generic name is mentioned in the Methods section.

- Abbreviations. Abbreviations should be avoided, but if they have to be used, in order not to repeat long technical names, the full word should appear the first time in the text, followed by the abbreviation in brackets, which will already be used in the manuscript.

Bibliographical references: bibliographical references should be presented in the order in which they appear in the manuscript, with a sequential number, which will appear in the appropriate place in the text, in brackets. This numbering will be maintained and will serve to order the list of all references at the end of the manuscript, as normal text and never as a footnote. Personal communications and unpublished data will not be included in the final list of bibliographical references, although they will be mentioned in the appropriate place in the text, in brackets, as appropriate, i.e. personal communication or unpublished data. When the bibliographic citation includes more than 6 authors, the first 6 authors should be cited, followed by the abbreviation et al.

The style of bibliographic references will depend on the type and format of the source cited:

- Medical journal article: journal names should be abbreviated according to the style of the Index. Medicus/Medline (http://www.ncbi.nlm.nih.gov/journals?itool=sidebar).

- Article already published in print and online journals Internet:
The authors (surname and initial of the first name, comma separation between authors), the full name of the manuscript, the abbreviation of the journal, the year of publication and after a semicolon the volume of the journal and after a colon the complete numbers of the first and last page of the paper.

Kandulsky A, Selgras M, Malfertheiner P. Helicobacter pylori infection: A Clinical Overview. Dig Liver Dis 2008; 40:619-626.

Alvarez F, Berg PA, Bianchi FB, Bianchi L, Burroughs AK,Cancado EL, et al. International Autoimmune Hepatitis Group Report: review of criteria for diagnosis of autoimmune hepatitis. J Hepatol 1999; 31:929-938.

- Admitted article, published only on the Internet, but not yet included in a regular number of the journal: the authors, the full name of the manuscript, the abbreviation of the journal, the year and month since the article is available on the Internet and DOI will be noted. The original paper to which reference is made usually details how to cite the manuscript.

Stamatakos M, Sargedi C, Stefanaki C, Safi oleas C, Matthaiopoulou I, Safi oleas M. Anthelminthic treatment: An adjuvant therapeuticstrategy against Echinococcus granulosus. Parasitol Int (2009), doi:10.1016/j.parint.2009.01.002

Inadomi JM, Somsouk M, Madanick RD, Thomas JP, Shaheen NJ. A cost-utility analysis of ablative therapy for Barrett'sesophagus, Gastroenterology (2009), doi: 10.1053/j.gastro.2009.02.062.

- Article from a journal that is published only on the Internet, but arranged in a conventional way: the authors, the full name of the manuscript, the abbreviation of the journal (can be added in brackets online), the year of publication and after a semicolon the volume of the journal and after a colon the complete numbers of the first and last page of the paper. If the original work being referenced provides the DOI and Internet address (URL), these can be added at the end of the reference.

Gurbulak B, Kabul E, Dural C, Citlak G, Yanar H,Gulluoglu M, et al. Heterotopic pancreas as a leading point for small-bowel intussusception in a pregnant woman. JOP (Online) 2007; 8:584-587.

Fishman DS, Tarnasky PR, Patel SN, Raijman I. Managementof pancreaticobiliary disease using a new intra-ductal endoscope: The Texas experience. World J Gastroenterol 2009; 15:1353-1358. Available from: URL: http://www.wjgnet.com/1007-9327/15/1353.asp. DOI: http://dx.doi.org/10.3748/wjg.15.1353

- Article from a journal that is published only on the Internet, but is not conventionally arranged: the authors, the full name of the manuscript, the abbreviation of the journal, the year of publication and the DOI will be listed.

Rossi CP, Hanauer SB, Tomasevic R, Hunter JO, Shafran I, Graffner H. Interferon beta-1a for the maintenance of remission in patients with Crohn's disease: results of a phase II dose-finding study.BMC Gastroenterology 2009, 9:22doi:10.1186/1471-230X-9-22.

-Article published in an abstract or in a supplement of a journal: the authors (surname and first initial, separated by commas between authors), the full name of the manuscript, the word abstract in square brackets, the abbreviation of the journal, the year of publication and after a semicolon the volume of the journal, followed by the abbreviation Suppl, or Supl, in brackets and after a colon the complete numbers of the first and last page of the paper.

Klin M, Kaplowitz N. Differential susceptibility of hepatocytesto TNF-induced apoptosis vs necrosis [Abstract]. Hepatology 1998; 28(Suppl):310A.

- Books: the authors of the book (surname and first initial, comma separated by the authors), the title of the book, the city where it was published, the name of the publisher and the year of publication will be listed.

Takada T. Medical Guideline of Acute Cholangitis and Cholecystitis. Tokyo: Igaku Tosho Shuppan Co; 2005.

- Chapter of a book: the authors of the chapter should be listed (surname and first initial, separated by commas between authors), followed by In: the names of the editors of the book and, after a full stop, the name of the book. The city where it was published, the name of the publisher, the year of publication and after a colon the complete numbers of the first and last page of the work.

Siewert JR. Introduction. In: Giuli R, Siewert JR, Couturier D, Scarpignato C, eds. OESO Barrett's Esophagus. 250 Questions. Paris: Hors Collection, 2003; 1–3.

- Information from a document produced at a meeting: this type of reference should be avoided whenever possible. However, if it has to be cited, the title of the subject, the name of the meeting and the city where it was held should be given. The entity that organised the meeting, and the year. The e-mail address through which the document can be accessed.

U.S. positions on selected issues at the third negotiating session of the Framework Convention on Tobacco Control. Washington, D.C.: Committee on Government Reform, 2002. (Accessed March 4, 2002, at:http://www.house.gov/reform/min/inves_tobacco/index_accord. htm.)

Figures, tables and videos: iconography, whether photographs, radiographs, diagrams or graphs, should be referred to under the generic name of "Figure". References to figures, tables and videos should be highlighted in bold. They should be numbered with Arabic numerals, according to their order of appearance in the text. Panels of two or more photographs grouped together will be considered as a single figure and may be referred to as "Figures 1A, 1B, 1C".

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Endoscopic images and other techniques generating colour images, if not directly available in electronic format, shall be scanned in colour. Special details shall be indicated by arrows, using for these and any other type of symbols the maximum contrast plot with respect to the figure.

Figure files will be identified according to their order of appearance in the text, with the name of the file, its number and surname of the first signatory (e.g.: fig1_Gómez.jpg) or the title of the article. Each image must have an associated figure caption that serves as a description. Figure captions must be submitted in a separate text document with clear reference to the figures to which they refer. Images may be inserted in Word/PowerPoint files to facilitate their identification or association with the figure captions, but they must always be submitted as separate images in the formats mentioned above.

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REVIEW ARTICLE

INITIAL MANAGEMENT OF ACUTE PANCREATITIS

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Abstract

The management of acute pancreatitis (AP) has undergone significant changes in recent years, driven by new research that challenges previous practices. Moderate-intensity, targeted fluid therapy is now favoured over more aggressive strategies. While evidence on the ideal type of fluid is limited, lactated Ringer's solution may be associated with better outcomes. Effective pain control and early oral nutrition, when possible, are key components of treatment. Antibiotic use should be limited to proven or highly suspected infections, avoiding prophylactic use. Urgent endoscopic retrograde cholangiopancreatography (ERCP) is indicated in patients with acute cholangitis. Cholecystectomy performed during the same admission for mild biliary AP is safe and effectively prevents relapses.

Keywords: acute pancreatitis, management, prognosis.

Introduction

Acute pancreatitis (AP) is a common disease that represents one of the leading causes of hospitalisation for gastrointestinal disorders, with significant associated costs and an increasing incidence[1]. Although most cases have a mild course, about one third of patients develop local complications or organ failure, which worsens the prognosis[2].

This article provides an evidence-based approach to the initial management of AP, addressing also the aetiological evaluation to prevent recurrences.

Early prognosis

After the initial diagnosis of AP, it is critical to assess the risk of the patient developing moderately severe or severe forms of the disease, which will guide treatment strategies.

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Factors such as obesity, advanced age and elevated blood urea nitrogen (or urea) and haematocrit have been associated with poor outcome[3,4]. Although multiple severity prediction systems exist, none have demonstrated clear superiority over others, and their positive predictive value is often limited[3].

Fluid therapy

Fluid therapy has been considered one of the mainstays of treatment in the early phase of AP. Traditionally, it has been believed that aggressive fluid replacement could improve blood flow to the pancreas, thereby reducing the risk of pancreatic necrosis[5]. However, recent studies, including our group's WATERFALL clinical trial, suggest that a moderate fluid therapy strategy (1.5 ml/kg/h and a bolus of 10 ml/kg in case of hypovolaemia), offers better results than aggressive fluid therapy, reducing fluid overload and associated complications [6,7]. Lactated Ringer's solution appears to be preferable to normal saline due to its anti-inflammatory properties[8].

Pain management

Pain is one of the most relevant symptoms in AP, significantly impacting patient experience. Opioids are commonly used due to their potent analgesic effect, although their use has been associated with worsening disease severity in some studies[9]. This result may be biased, as the most severe cases are those most frequently receiving opioids. Other alternatives such as non-steroidal anti-inflammatory drugs (NSAIDs) have been shown to be equally effective in controlling pain during the first 24 hours[10]. In a pilot study by our group, metamizole tended to be more effective than morphine, but the sample size was very small[11]. Epidural anaesthesia may be an effective option in cases of uncontrolled pain in intensive care units[10]. Finally, COX-2 inhibitors may have beneficial effects on disease progression[12].

Early nutrition

Historically, it was believed that 'pancreatic rest', avoiding oral food intake, was essential in the treatment of AP in order to prevent pancreatic enzyme stimulation and secretion. However, recent studies have challenged this belief. Numerous randomised clinical trials in good prognosis pancreatitis have shown that early feeding, even with a solid or soft diet, is safe and associated with a reduction in length of hospitalisation and costs, without increasing rates of oral intolerance or complications[13]. Evidence shows that it is not necessary to start with a liquid diet and gradually progress to solid foods but that early introduction of a solid or soft diet is well tolerated and shortens hospital stay[13]. In patients with severe or moderate pancreatitis, who cannot tolerate oral feeding, enteral nutrition is still useful, but in patients who tolerate oral intake, it does not change the course of the disease and its widespread use should be avoided[14]. In the absence of duodenal stenosis, there is no advantage for the use of nasojejunal tube nutrition over nasogastric tube nutrition[15,16]. Parenteral nutrition should generally be avoided in favour of oral nutrition, or enteral if not tolerated[17], as it is associated with poorer outcomes. The use of immunonutrients, such as L-glutamine and omega-3 fatty acids, requires further evaluation in larger studies to establish their true benefit in patients with AP. While some preliminary studies suggest potential benefits, heterogeneity in results and small sample sizes have limited the generalisability of immunonutrient use. In particular, probiotics, once considered a possible treatment option, have been shown to be potentially dangerous in patients with severe AP, with increased mortality[18].

Antibiotic use and antifungal therapy

The use of antibiotics in AP is a controversial issue due to their overuse. While some patients may benefit from their use in cases of confirmed or suspected infection, prophylactic administration of antibiotics in AP without evidence of infection has been shown to be ineffective. Several meta-analyses have concluded that prophylactic use does not reduce the incidence of infected necrosis or improve other clinical outcomes[19].

Deciding when to administer antibiotics in patients with AP who present with signs of inflammation, such as fever or leukocytosis, but without clear evidence of infection, remains a clinical challenge. In these cases, the use of the bacterial infection marker procalcitonin (with a cut-off point of 1 ng/ml) has been proposed as a useful tool to guide the decision to start or stop antibiotics, thus reducing unnecessary antibiotic use without compromising patient safety[20].

In terms of antifungal therapy, fungal infections, such as invasive candidiasis, can develop in patients with necrotising AP, particularly those patients in the intensive care unit who require central venous catheters, parenteral nutrition or broad-spectrum antibiotics. However, antifungals should also not be administered prophylactically; their use should be reserved for confirmed fungal infections[21].

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Endoscopic retrograde cholangiopancreatography (ERCP)

Studies suggest that early ERCP in patients with cholangitis is associated with a significant reduction in hospital mortality, 30-day mortality, organ failure and length of hospital stay[22-24].

However, the use of early ERCP (first 24-48h) in patients with biliary AP without cholangitis has been a matter of debate. Several randomised clinical trials have shown that early ERCP does not improve clinical outcomes in these patients, even in those with gallstones or gallbladder sludge[25-27]. Therefore, early ERCP should be reserved for those with suspected cholangitis. In the presence of choledocholithiasis, it is common sense that should guide when to perform ERCP, as no study has shown it to be harmful either.

Aetiological assessment

Identifying the cause of AP is essential to prevent recurrent episodes. Biliary aetiology is the most common cause, responsible for approximately 60% of cases[2]. Abdominal ultrasound has limited sensitivity for the diagnosis of gallbladder biliary sludge, so in the absence of biliary lithiasis or other aetiologies in the ultrasound, it is recommended to perform an endoscopic ultrasound, as it allows diagnosis, in addition to accurately assessing the pancreatic parenchyma and its ductal system. In cases of mild biliary AP, cholecystectomy during the same hospital admission is more effective in preventing relapses and gallstone-related complications compared to delayed cholecystectomy[28]. In cases of AP with collections/necrosis, it is recommended to delay cholecystectomy until local complications are controlled, usually 4-6 weeks after the initial episode[28,29]. Imaging tests will give an idea of the regression of collections so that the patient can be operated on.

Another important cause of AP is alcohol consumption, which accounts for 15-20% of cases[2,30]. Approximately 50% of patients who continue to consume alcohol after an episode of AP will have recurrences, whereas abstinence significantly reduces this risk[31]. Repeated interventions to promote abstinence from alcohol, even after discharge, have been shown to be more effective than single interventions during hospitalisation[32].

Hypertriglyceridaemia (HTG) is a less common cause of AP in Western countries, accounting for 2-5% of cases, although in countries such as China, its prevalence is much higher[33]. Triglyceride levels above 1000 mg/dL are usually required to induce an episode of AP[33]. HTG-induced AP has been shown to be associated with a worse prognosis and increased risk of recurrence[33,34].

The initial management of AP due to HTG is similar to that of other aetiologies. However, in these patients it may be necessary to prolong fasting to reduce triglyceride levels. In addition, the use of insulin and plasmapheresis have been used as strategies to lower triglycerides, although the evidence supporting these treatments in terms of improving clinical outcomes is limited, and even plasmapheresis may be detrimental[35,36]. To avoid recurrences, it is essential to implement changes in diet, lifestyle and the use of lipid-lowering drugs, with the aim of keeping triglyceride levels ideally below 200 mg/dL[37].

Conclusions

The initial management of acute pancreatitis continues to evolve, with new evidence challenging previous practices and influencing current clinical strategies. Current tools for predicting disease severity remain imperfect; modest goal-directed fluid therapy has replaced aggressive strategies due to the detrimental outcomes associated with the latter. Effective pain control and early oral nutrition are essential components in the management of AP. The use of antibiotics and antifungal therapies should be limited to cases with proven or highly suspected infections, avoiding their prophylactic use. Procalcitonin has been proposed as a useful tool to decide when to start or stop antibiotics. Early ERCP should be reserved for cases of acute cholangitis, and the aetiology of AP should be addressed to prevent relapse. Same-admission cholecystectomy in cases of mild biliary AP may reduce disease recurrence.

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CLINICAL CASE

GASTROINTESTINAL BLEEDING SECONDARY TO DUODENAL VASCULAR ANOMALY DUE TO MEDIAN ARCUATE LIGAMENT SYNDROME

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Abstract

We present the case of a 52-year-old woman with a history of CADASIL syndrome and dependence for basic activities of daily living who was admitted to the hospital due to asthenia, dizziness and anemia. During hospitalization imaging tests were performed, detecting stenosis of the celiac trunk secondary to compression by the median arcuate ligament, as well as dilatation of the pancreaticoduodenal arcades and aneurysm of a submucosal pyloroduodenal artery. Subsequently, she started with melena and hemodynamic instability, for which reason oral endoscopy was performed with the finding of a visible vessel in the anterior wall of the duodenal bulb. Hemostatic treatment was applied and the clinical-radiological evolution was favorable, dismissing other more invasive options due to the patient's fragility.

Keywords: gastrointestinal bleeding, median arcuate ligament syndrome, aneurysm.

Introduction

The celiac trunk is an artery that arises in close proximity to the diaphragm and has an important anatomical and pathophysiological relationship with this structure. On the other hand, the median arcuate ligament (MAL) is a fibrous arch at the base of the diaphragm that connects the right and left diaphragmatic crura at the aortic hiatus[1,2]. Certain anatomical variants of this ligament can sometimes cause direct compression of the celiac trunk[1,3]. However, if this compression is significant, it can manifest as median arcuate ligament syndrome (MALS) and its clinical features are derived from foregut ischaemia, i.e. postprandial epigastralgia, weight loss and sitophobia (fear of eating)[1,4]. Other less frequent but more serious manifestations are related to the development of aneurysms, especially in the pancreaticoduodenal arteries, either in the form of gastrointestinal or retroperitoneal haemorrhage. These constitute less than 2% of visceral arterial aneurysms and are usually due to atherosclerosis, pancreatitis,

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trauma and fungal or bacterial infections. There are also case reports describing an association between MALS and aneurysms of the pancreaticoduodenal artery[4].

Clinical case

A 52-year-old woman with a personal history of CADASIL syndrome (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy) under follow-up by Neurology and dependent for basic activities of daily living was admitted for asthenia, dizziness and anaemia (Hb 5.1 g/dl), with no evidence of bleeding. A thoracoabdominal CT scan was performed with a finding of compression of the celiac trunk by the median arcuate ligament. Therefore, a further study was carried out by CT angiography, confirming significant non-atherosclerotic stenosis at the exit of the celiac trunk due to compression by the median arcuate ligament of the diaphragm. In addition, the celiac trunk was visualised in the form of a 'hook', with post-stenotic dilatation of the pancreaticoduodenal arcades and an aneurysm of a submucosal pyloroduodenal artery (Figure 1). The day after the CT angiography, the patient began to develop melena and haemodynamic instability, for which reason oral endoscopy was performed and a large clot was found in the anterior aspect of the duodenal bulb, which was removed and a visible vessel was visualised. It was treated with diluted perilesional adrenaline and three haemostatic clips, with good endoscopic results (Figure 2). The patient's subsequent clinical evolution was favourable, with progressive recovery of haemoglobin levels. However, given the radiological findings, the possibility of embolisation and surgery was considered with interventional radiology, but both approaches were rejected given the good evolution of the patient and considering her fragility. Subsequently, CT angiography was repeated without observing the submucosal aneurysmal arterial vascular structure in the duodenal bulb due to probable resolution after endoscopic treatment. Finally, the patient was discharged from hospitalisation and presented no clinical incidents in her subsequent follow-up.

Discussion

This case draws attention to the complexities of MALS and its association with complications such as aneurysms, particularly of the pancreaticoduodenal artery[4]. Approximately 7-15% of these aneurysms are associated with haemorrhage, particularly in the retroperitoneal space. When an aneurysm ruptures, mortality rates can be as high as 50% [4,5].

In MALS, blood flow to the splanchnic organs is redirected to the superior mesenteric artery (SMA) and the pancreaticoduodenal arcades due to the lowering of blood

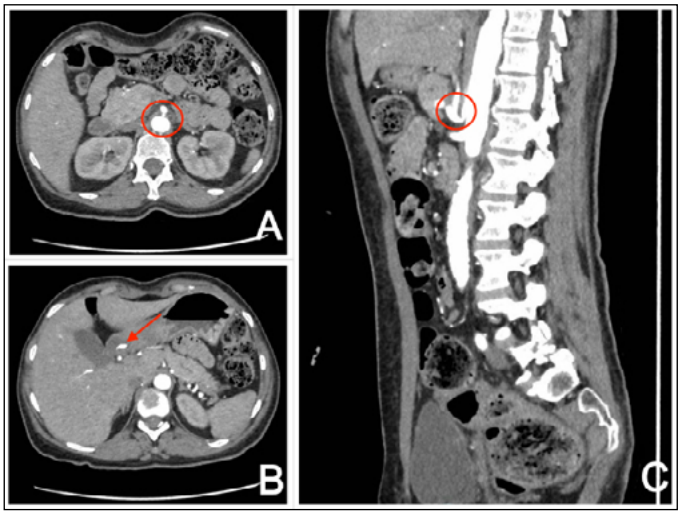

Figure 1. ***Images obtained from CT angiography showing the characteristic "hook" shape of the celiac trunk during expiration with a red circle. The submucosal pyloroduodenal artery with aneurysmal dilatation is indicated by a red arrow.***

flow in the celiac trunk. Although there is no proven causal mechanism for this relationship, it is thought that these pancreaticoduodenal arcades and other more distal branches would not support this increased flow, leading to increased shear stress on the arterial wall, formation of pseudoaneurysms, growth of these and eventually spontaneous rupture[2,4]. Unlike other types of aneurysms, the risk of rupture is not directly proportional to the diameter of the aneurysm itsel[3,5,6].

The diagnosis of MALS is made by a combination of symptoms and compatible imaging findings. It is sometimes difficult and is made by chance or after the occurrence of a

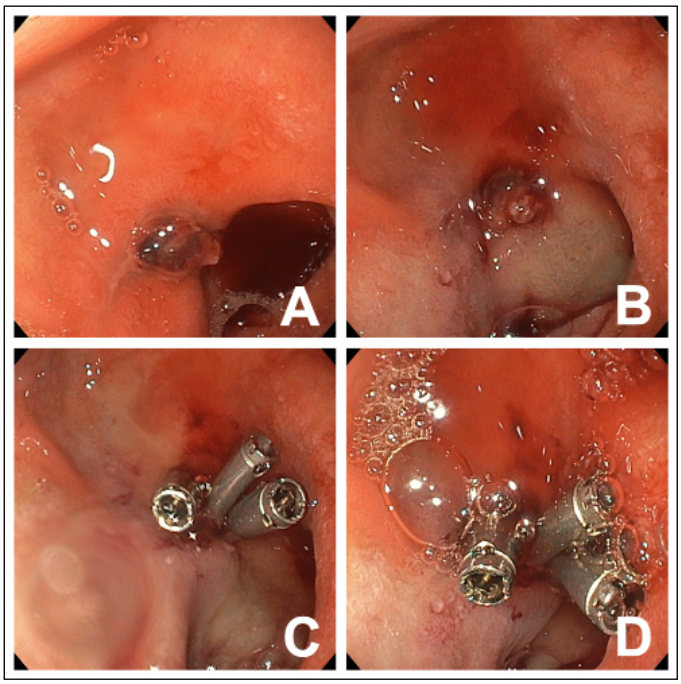

Figure 2. ***Endoscopic image showing: A) Clot with visible vessel in duodenal bulb; B) Injection of diluted perilesional adrenaline; C and D) Application of three haemostatic clips with sealing of the lesion.***

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complication. The gold standard for the diagnosis of MALS is CT angiography where the characteristic ''hook'' shape of the celiac trunk is identified during expiration. However, Doppler ultrasound is a non-invasive method and is more widely available, which would be a valid alternative in certain cases[2,4].

Treatment of MALS consists primarily of surgical release of the ligament, thus improving blood flow. Endovascular techniques are available as alternatives, but they have higher rates of therapeutic failure[4]. However, there are currently no consensus or treatment protocols for the management of this type of aneurysm, thus posing a major therapeutic challenge. Early treatment of these aneurysms is critical to prevent serious complications, as in our case, due to the high risk of spontaneous rupture. Some authors propose embolisation of the aneurysm as the sole treatment while others suggest releasing the stenosis from the celiac trunk. If the stenosis-relieving approach is chosen, there is a better chance of avoiding aneurysm recurrence. In contrast, in asymptomatic individuals with incidental finding of MALS there seems to be no indication for treatment[4,7].

In conclusion, MALS is an underestimated clinical entity and with this clinical case we want to increase the index of suspicion in the medical population, as it can lead to serious complications.

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CLINICAL CASE

JEJUNAL ADENOCARCINOMA: A RARE ENTITY.

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Abstract

We present the case of a 39-year-old woman with epigastralgia, hypermesis and severe weight loss of 6 month´s evolution. Laboratory tests, upper endoscopy, colonoscopy together with inconclusive imaging techniques were performed. Finally exploratory laparotomy was carried out, revealing intestinal obstruction secondary to jejunum tumor. The pathological analysis of the surgical specimen confirmed the diagnosis of invasive jejunal adenocarcinoma.

Keywords: jejunal adenocarcinoma, intestinal obstruction, constitutional syndrome.

Introduction

Jejunal adenocarcinoma is a very rare entity, the most common intestinal adenocarcinoma being that of the duodenum. It is more prevalent in males between the 6th-7th stages of life and usually presents asymptomatically and indolently, with intestinal obstruction associated with abdominal pain and anaemia with an iron deficiency profile being the most frequent forms of presentation. Imaging tests together with a high level of clinical suspicion and thorough anamnesis are the pillars on which the diagnosis of this type of tumour is based, with anatomopathological analysis being essential for definitive diagnosis. The treatment and initial prognosis at diagnosis depend on the tumour stage, with those in a more advanced stage having a worse prognosis.

Clinical case

39-year-old woman, with no family history of interest and a personal history of left hemithyroidectomy for multicystic multinodular goitre 8 years earlier. After presenting with miscarriage due to anembryonic gestation 5 months prior to the onset of the clinical picture, she presented diffuse abdominal

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pain, postpartum vomiting, liquid stools and weight loss of up to 26 kg in the 6 months prior to coming to our clinic.

Primary care performed an abdominal ultrasound with findings of cholelithiasis, upper gastrointestinal endoscopy and colonoscopy, with no abnormalities. Complete analytical study and normal coeliac profile. Calprotectin 846 µg/g.

Given the persistence of symptoms and abdominal pain, prophylactic cholecystectomy was performed in a private centre 2 months prior to the consultation, without any improvement in the clinical symptoms despite the same.

The patient repeatedly visited the emergency department due to persistent abdominal pain, with hyperemesis and progressive weight loss, and was referred to Internal Medicine, which, after a negative study, consulted our department and decided to admit her to the Digestive Department.

Physical examination revealed diffuse abdominal pain, with no clear peritonism, but focusing on the right iliac fossa, even with negative Blumberg, and preserving hydro-aerial sounds. In analytical tests, he already showed signs of renal insufficiency with creatinine at 1.90 mg/dl, GFR 63 ml/min/1.73m2, the rest normal.

The haemogram showed a thrombocytosis of 554,000 /mm3 with normal tumour markers and no other alterations. The abdominal CT scan without contrast showed dilated small bowel loops (jejunum) with liquid content and hydroaerial levels and mucous hyperenhancement, identifying a sudden change in calibre located at the hypogastric level, data compatible with small bowel obstruction secondary to flanges or focal jejunal stenotic segment without being able to determine its nature (Figure 1).

General surgery was contacted for assessment, initially rejecting surgical intervention and opting for conservative treatment. Empirical antibiotic therapy (Ciprofloxacin and Metronidazole) and intravenous corticotherapy were started. The biological study showed only normocytic normochromic anaemia. Tumour markers and calprotectin were normal on this occasion. An attempt was made to initiate oral tolerance, but the patient presented intolerance to this with abdominal pain, vomiting and liquid stools, so a new control abdominal CT scan was performed, which showed persistent dilatation of the proximal jejunal loops of up to 45 mm with the presence of free liquid in the pelvis (Figures 2 and 3). Surgery was contacted urgently and exploratory laparoscopy was performed, identifying after systematic exploration of the small intestine from the ileocecal valve, the stenosis described in the middle jejunum, which caused the retrograde dilatation of the proximal intestinal loops visible in the CT scan up to the angle of Treitz. Two reactive adenopathies were identified adjacent to the vascular axis of the root of the meso of the loop on which the lesion depended.

Figure 1. ***Coronal abdominal CT scan showing dilatation of the jejunal loops (blue arrow).***

Segmental small bowel resection was performed for excision of the lesion with safety margins, with mechanical laterolateral jejunojejunal anastomosis and regional mesenteric lymphadenectomy. The anatomopathological report of the surgical specimen was invasive jejunal adenocarcinoma (Figure 4), well differentiated (G1), reaching the muscularis propria layer (pT2), respecting surgical edges and vascular spaces. Negative lymph nodes and MISMATCH REPAIR, MMR not altered (Figure 5). Subsequently, a complete extension study was performed with thoracic CT, ruling out distant disease, so that, together with the anatomopathological data, the patient was diagnosed with stage I adenocarcinoma of the jejunum. The case was presented to the multidisciplinary tumour committee and the patient did not require chemotherapy, and is currently being monitored by the Digestive System, in High Risk Digestive Cancer consultations.

Discussion

Small bowel tumours are a rare entity within the gastrointestinal tract, constituting 5% of all gastrointestinal neoplasms and 1-3% of malignant neoplasms in this location. In general, they are more frequent in males, from 60 years of age onwards. The most frequent malignant tumours of the small intestine are adenocarcinoma and lymphoma,

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Figure 2. ***Axial abdominal CT scan showing jejunal loop dilatation (green arrow) up to 45 mm in calibre with abundant liquid content.***

Figure 3. ***Sagittal abdominal CT scan showing small bowel dilatation, some parietal thickening and mucosal hyperenhancement, identifying a point of change of calibre in the jejunum (blue arrow).***

with sarcoma, GIST and carcinoids being less frequent. Adenocarcinoma of the small intestine is the most common malignant tumour, usually originating from adenoma and most commonly located in the duodenum, with adenocarcinoma of the jejunum being less common. Associated risk factors are smoking, obesity, celiac disease, Crohn's disease, a history of colorectal cancer and familial adenomatous polyposis, with the age of onset being lower in patients with these pathologies[1].

The clinical presentation of jejunal adenocarcinoma is non-specific, mostly asymptomatic, although they usually present with abdominal pain in later stages, nausea, vomiting,

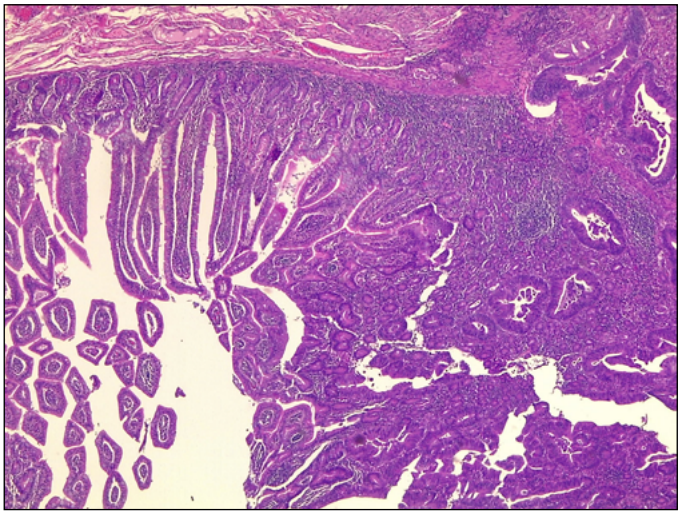

Figure 4. ***Histological section showing morphological changes typical of invasive, well-differentiated intestinal adenocarcinoma, with more than 95% glandular formation.***

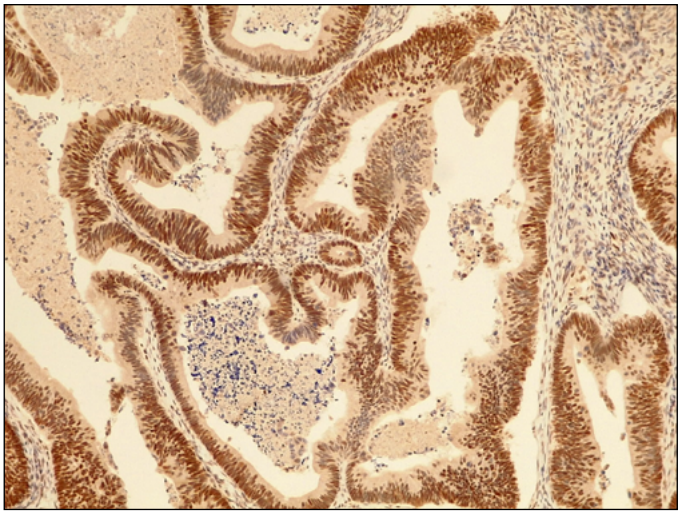

Figure 5. ***Tumour cells showed unaltered immunoexpression of MISMATCH REPAIR.***

weight loss, anaemia, asthenia, anorexia, and may cause pseudo-occlusive symptoms and intestinal obstruction, given that these types of tumours present annular growth, as occurred in our case. More than half of the patients have advanced disease at the time of diagnosis. Malignant jejunal tumours are an entity that are rarely diagnosed preoperatively due to their rarity and non-specific clinical features[2].

The most important causes associated with small bowel obstruction that should be taken into account in the differential diagnosis are intestinal adenocarcinoma, carcinoid neoplasia, lymphoma, gastrointestinal stromal tumours, inflammatory bowel disease, bridles, hernias, biliary ileus, as well as benign neoplasms, although the latter do not usually behave aggressively. There are several theories that attempt to shed light on the low prevalence of these tumours, including rapid transit, liquid intestinal contents, a high concentration

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of digestive enzymes in this tract that detoxify potential carcinogens along with low presence of bacterial populations and the abundant presence of lymphoid tissue[3,4].

Abdominal CT is the main imaging technique for identifying the primary tumour and assessing its extension; sometimes magnetic resonance imaging or capsule endoscopy is used to locate the tumour, although caution should be exercised in patients with clinical suspicion of intestinal obstruction or subocclusive symptoms[5]. What is exceptional in our case is that the patient had no previous potentially obstructive pathologies, at the infrequent age of onset, which led to a delay in diagnosis, with cholecystectomy the previous 2 months via laparoscopy, which we believe also constituted a confounding factor in the delay, since the pain persisted - attributed to the postoperative period/potential flanges - although it was within the average 6 months in which it is usually diagnosed. The striking weight loss and sudden onset anorexia - which could initially be attributed to an eating disorder - were the guiding symptoms that eventually led to the diagnosis.

The treatment of these types of tumours is mainly based on extensive surgery to resect the tumour with regional lymphadenectomy. If there is lymph node involvement, treatment is completed with postoperative chemotherapy. Treatment depends on the tumour stage of the patient. However, the use of neoadjuvant chemotherapy is not yet well defined in these tumours. In unresectable adenocarcinomas, palliative chemotherapy is indicated, and if there is also extensive occlusion of the jejunal lumen, palliative resection is indicated.

In any case, given the infrequency of primitive tumours of the small intestine, they constitute a real diagnostic challenge and their paucisymptomatic onset up to advanced stages requires a thorough anamnesis and a high index of initial suspicion to avoid delays in treatment, with exploratory laparoscopy being necessary in some cases, such as ours, to identify the lesion and analyse the surgical specimen.

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CLINICAL CASE

PULMONARY GRANULOMATOSIS IN ULCERATIVE COLITIS, AN UNCOMMON EXTRAINTESTINAL MANIFESTATION

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Abstract

This is an ex-smoker patient with a history of long-standing ulcerative colitis with persistent activity and refractory to several biological therapies. She has ongoing symptoms, such as bloody diarrhea, weight loss, and severe anemia that require regular intravenous iron transfusions. Despite attempts to control the disease with different medications, including Infliximab and Vedolizumab, the patient remains symptomatic and elevates markers of disease activity, such as fecal calprotectin.

At follow-up, the patient presented with a chronic cough, which led to studies that revealed the presence of a hypermetabolic pulmonary nodule in the left lower lobe, suspected of primary pulmonary neoplasia, and an atypical pulmonary resection was performed.

Diagnosis reveals possible respiratory granulomatous intestinal epithelial metaplasia due to histological findings in the lung, consistent with an uncommon extraintestinal manifestation in the context of uncontrolled ulcerative colitis.

Finally, a change of therapeutic target with Ustekinumab was initiated due to the previous failure of anti-TNF and Vedolizumab treatments in the control of ulcerative colitis, with clinical improvement in the digestive and respiratory spheres.

Keywords: ulcerative colitis, granulomas, chronic cough.

Introduction

Inflammatory Bowel Disease (IBD) is characterised by chronic recurrent intestinal inflammation. There are two well-defined entities: Crohn's Disease (CD) and Ulcerative Colitis (UC). While CD causes discontinuous transmural inflammation in any section of the digestive tract and may be

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associated with fistulas, abscesses or strictures, UC causes a continuous alteration of the ultrastructure of the colon.

In clinical terms, persistent abdominal pain, weight loss, asthenia, fever or feverishness or diarrhoea with pathological products are the most frequent symptoms at the onset of IBD, the intensity of which correlates with greater severity[1].

Other clinical manifestations associated with IBD are extraintestinal manifestations (EIMs). The prevalence of EIMs ranges from 6% to 47%, affecting more women (50%) than men (34%)[2]. However, in a not insignificant proportion of cases (25.8%), EIMs appear before formal diagnosis of IBD, with a diagnostic delay of up to 5 months and as long as 2 years[3].

EIMs can affect any system or apparatus[4], with musculoskeletal manifestations (peripheral arthritis type 1 or 2 and HLA B27 axial spondyloarthropathy), cutaneous (erythema nodosum, pyoderma gangrenosum, aphthous stomatitis, psoriasis and Sweet's syndrome), ophthalmological (episcleritis, scleritis and uveitis), hepatobiliary (primary sclerosing cholangitis), renal and pulmonary, the latter being more rare.

Infrequent EIMs include those affecting the pulmonary sphere. It is essential to recognise the common presenting pulmonary symptoms (dyspnoea, wheezing, dry or productive cough, pleuritic pain, fever or stridor). However, between 37% and 55% of patients with IBD may present with abnormalities on pulmonary function tests, chest imaging or histopathology in the absence of respiratory symptoms[5].

Pulmonary EIMs is very diverse and can affect the airways (granulomatous or obliterative bronchiolitis, bronchiectasis, chronic bronchitis), the parenchyma (organised interstitial, lymphocytic or eosinophilic pneumonia and granulomatous interstitial lung disease) or diffusely (interstitial pneumonitis, abscesses, granulomas)[6].

The prevalence of pulmonary EIMS is not clearly defined in the literature, with a low prevalence found among adolescents, a group in which IBD is very common[7]. A greater association with CD has been seen, although they may appear independently of IBD activity, which makes their suspicion and management even more difficult.

Early detection of EIMs is of vital importance, as it affects the overall management of IBD patients[8]. On the one hand, IBD increases the overall burden of disease, leading to increased morbidity, reduced quality of life and the development of complications. In addition, such detection requires a multidisciplinary approach involving a range of specialists to achieve optimal management of intestinal and extraintestinal pathology. Patient education and support is also important to control modifiable environmental factors and ensure adherence to treatment.

A fundamental pillar is the medical treatment of IBD9. There are currently several therapeutic steps ranging from topical treatments (suppositories, enemas or Mesalazine foams) to oral treatment (mesalazine, beclomethasone, budesonide or prednisone). If clinical, analytical or endoscopic remission is not achieved, it is necessary to resort to biological therapies and small molecules, with a proven efficacy and safety profile in routine clinical practice. This allows better control of IBD, as well as those EIMs that are parallel and dependent on digestive activity (peripheral arthritis type 1, erythema nodosum, aphthous stomatitis or episcleritis, among others).

We present the clinical case of a patient with uncontrolled UC refractory to conventional treatment and various biological lines who, during close follow-up in the clinic, presented with a chronic cough attributable to pulmonary granulomatosis, a rare pulmonary EIM.

Clinical case

The patient is a 22-year-old former smoker of (half a pack a day), with no medical history of interest in the digestive sphere, who was diagnosed in 1998 with UC. Since then, the patient has presented a variegated clinical course that has required the administration and substitution of multiple treatments: Oral and topical mesalazine, beclomethasone, continuous rounds of oral corticosteroids up to mercaptopurine, which was substituted to infliximab due to lack of improvement, initiated after performing the prebiological study which ruled out contraindications (Among them, the presence of latent tuberculosis). Despite maximum intensification of infliximab, remission of the disease was not achieved either, changing the therapeutic target to vedolizumab, with the same clinical, analytical and endoscopic results despite maximum intensification. This led the patient to continually visit the emergency department, with multiple hospitalisations since the onset of the disease.

Even with all the therapeutic adjustments described, the patient has presented multiple outbreaks over the years, with persistent symptoms such as severe bloody diarrhoea (more than 8 bowel movements per day), significant weight loss (10 kg), faecal incontinence and severe anaemia that has required periodic intravenous iron transfusions. In addition, he has suffered on 2 occasions from infectious colitis secondary

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to *Clostridium Difficile*, which responded adequately to vancomycin.

From the analytical point of view, the difficulty in controlling the disease translates into a persistent and not very fluctuating elevation of acute phase reactants: faecal calprotectin around 2000-4000 IU/L, anaemia (haemoglobin around 10 mg/dL), hypoalbuminaemia and thrombocytosis. All this indicates a high inflammatory burden of UC, which requires endoscopic controls in the face of continuous changes in treatment, with findings of diffuse UC with mild-moderate activity in the last colonoscopy performed in 2024.

During close follow-up in the gastroenterology department, the patient began with a productive brown phlegm cough of 2 months' duration that did not improve with inhalation treatment or antibiotic therapy. Additional tests included negative sputum cultures, negative Mantoux and IGRA, normal respiratory function tests and a chest CT scan showing two nodular images in the right lower lobe (RIL) and another in the left lower lobe (LIL) of approximately 12 mm with indeterminate characteristics (Figure 1).

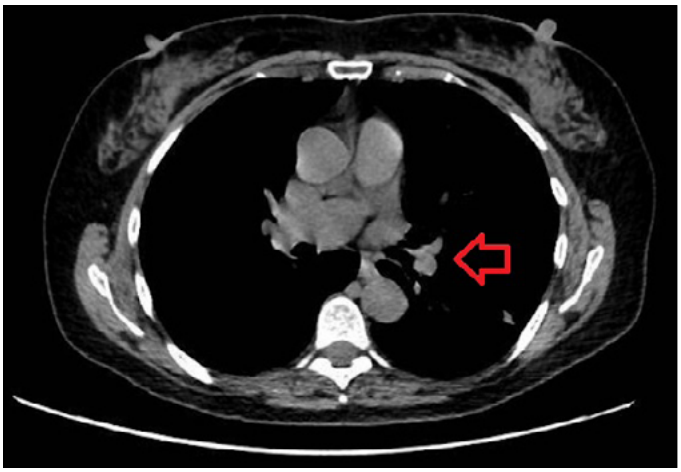

Figure 1. ***Cross-sectional CT scan of the chest showing a nodular image in LIL of about 12 mm of undetermined characteristics (red arrow).***

Positron Emission Tomography (PET-CT) was performed and showed a hypermetabolic pulmonary nodule in LIL (Figure 2). Heterogeneous intestinal metabolic activity with hypermetabolic mesenteric and retroperitoneal adenopathies was also identified, and a multidisciplinary assessment was recommended for suspected primary or metastatic pulmonary neoplasia. Given the patient's immunosuppression due to biologic therapy, the differential diagnosis included pulmonary tuberculosis. This diagnostic possibility was ruled out in view of the negative Mantoux and IGRA results, as well as the lower and single location of the nodule, which differed from the usual behaviour of tuberculosis.

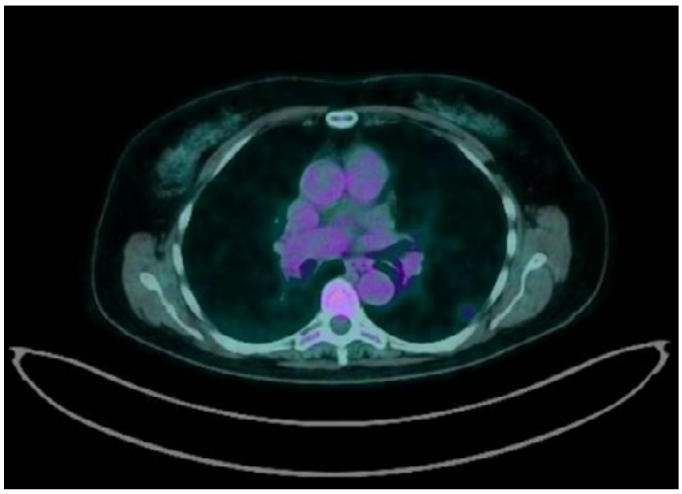

Figure 2. ***Thoracic cross-sectional PET-CT scan showing a 12mm hypermetabolic pulmonary nodule in LIL. No other findings suggestive of macroscopic neoplastic lesions or high metabolic grade were observed.***

In view of these findings, the case was presented to the multidisciplinary committee for thoracic tumours and a decision was made to perform surgery consisting of atypical resection of the LIL nodule with intraoperative analysis and, depending on the results, to decide to extend the resection. The results showed lung fragments with necrotising granulomas and surrounding hyalinising fibrosis, compatible with an uncontrolled active UC EIM, ruling out neoplastic disease.

Following the anatomopathological findings, it was decided to change the biological target to Ustekinumab due to the previous failure of Infliximab and Vedolizumab as advanced therapies, as well as other conventional treatments. In addition, given the reduced lung capacity after resection, the patient was instructed on the use of inhalers and measures to improve her lifestyle were recommended.

At the last consultation review, post-surgical changes were observed in the left lung in the control chest CT scan, together with a stable RIL nodule, as described above (Figure 3). In addition, the patient has shown some clinical bowel improvement after the last change of therapeutic target, with a reduction of the stool rhythm (4-5 bowel movements per day with minimal pathological products) and analytical improvement. From the respiratory point of view, the cough she had suffered in recent months has disappeared, so it was decided to maintain the current treatment and to carry out a close multidisciplinary follow-up in the Digestive and Pneumology departments.

Discussion

The case presents several challenging clinical and therapeutic aspects that merit discussion. The presence

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Figure 3. ***Sagittal CT scan of the chest showing post-surgical changes after atypical resection in LIL. 9 mm stable and known nodule in the right lung base.***

of long-standing UC with persistent inflammatory activity despite multiple treatments, including biologic agents such as Infliximab and Vedolizumab, underscores the complexity of managing this chronic bowel disease. The need for intravenous iron transfusions due to severe anaemia and the persistence of severe gastrointestinal symptoms, such as bloody diarrhoea and faecal urgency, significantly affect the patient's quality of life.

The incidental finding of a hypermetabolic pulmonary nodule, together with the chronic respiratory symptoms, raises concern for the possibility of primary pulmonary neoplasia, although the finding of necrotising granulomas on lung biopsy suggests possible respiratory intestinal epithelial epithelial metaplasia, compatible with pulmonary EIM, an option that should be included in the differential diagnosis in this case.

The decision to switch to Ustekinumab, due to the previous failure of the biologic agents used, highlights the importance of considering alternative therapeutic options in cases of refractory IBD. However, the persistent elevation of activity markers, such as faecal calprotectin, indicates the need for careful monitoring and possible further evaluation of treatment effectiveness.

By consulting the existing literature, this case addresses the complex and unusual extraintestinal pulmonary clinical presentation of IBD and highlights the importance of a multidisciplinary approach involving gastroenterologists, pulmonologists and thoracic surgeons, among other specialists, to address the different aspects of the disease and its potential complications.

Such multidisciplinary management is key to achieving control of the disease in the digestive and extradigestive sphere, which has a positive impact on patients' quality of life.

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CLINICAL CASE

ISCHEMIC COLITIS SECONDARY TO ARTERIOVENOUS MALFORMATION, A RARE CAUSE OF GASTROINTESTINAL BLEEDING

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Abstract

We present the case of a 65-year-old patient who presents with rectorrhagia with hemodynamic compromise, caused by ischemic colitis secondary to a vascular malformation at the level of the inferior mesenteric artery.

The presence of arteriovenous malformations in the abdominal territory is rare and it can lead to ischemic colitis as a consequence of the arteriovenous shunt and the decrease in oxygen supply to the surrounding tissue. Since this condition can simulate other more common inflammatory pathologies, it is usually a diagnostic challenge for the clinician and it is necessary to keep it in mind for a correct therapeutic approach.

Keywords: ischemic colitis, arteriovenous malformation, hemicolectomy.

Introduction

Arteriovenous malformations in the abdominal territory can give rise to ischaemic pictures that can simulate other more common inflammatory pathologies, posing a diagnostic challenge for the clinician. Their therapeutic approach is also complex, with intestinal resection of the affected segment and vascular embolisation being necessary in most cases.

Clinical case

A 65-year-old patient with no past medical history of interest was admitted for two weeks of diarrhoea and rectal bleeding, presenting with a palpable, non-painful mass in the left flank and anaemia in transfusion range.

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A colonoscopy was performed, showing an oedematous appearance of the mucosa with abundant excavated ulcers, suggestive of inflammatory colitis without being able to rule out other causes (Figure 1). The abdominal CT scan showed thickening of the walls of the descending colon, sigma and rectum, with an adjacent image suggestive of an extensive arteriovenous malformation in the territory irrigated by the inferior mesenteric artery (Figure 2), suggesting a probable ischaemic origin of the colitis. The study was completed with an abdominal angiography showing a vascular malformation whose arterial supply was seen at the level of the branches dependent on the sigmoid artery, with respect to the rectal artery, and the compromised branches were embolised with coils (Figure 3). After 48 hours, the patient presented with frank rectal bleeding and haemodynamic instability, so surgery was performed, with left hemicolectomy extended to the upper rectum and resection of the AVM, with a good clinical evolution.

Figure 1. ***Endoscopic image with findings of colitis.***

Figure 2. ***Arteriovenous malformation visualised by abdominal CT scan.***

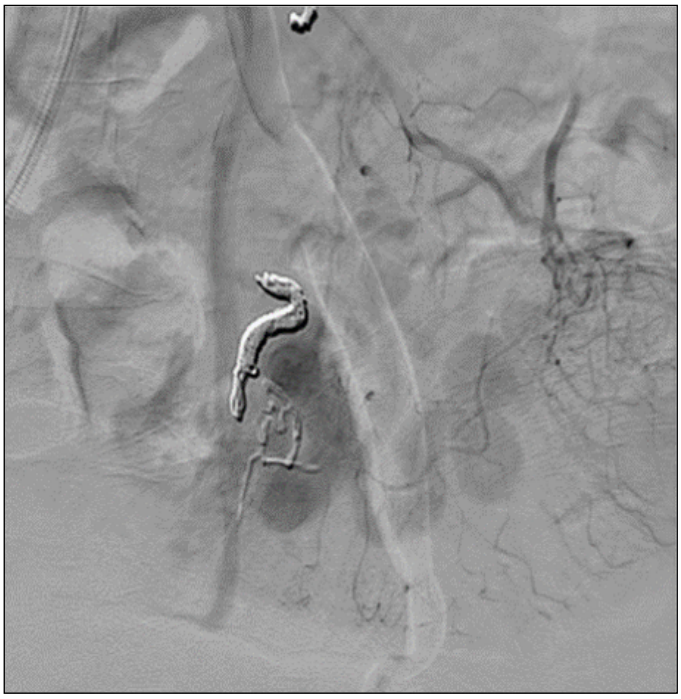

Figure 3. ***Arteriography image showing vascular malformation.***

Discussion

Arteriovenous malformations and fistulas are aberrant vascular connections between arteries and veins, and may be primary or congenital, or secondary to previous surgery or trauma[1]. They can result in a steal phenomenon with disruption of normal blood flow creating a left-to-right shunt leading to a decrease in oxygen supply to the surrounding tissue. When this occurs in the territory supplied by the inferior mesenteric artery, ischaemic colitis may be triggered, but this location is extremely rare[2].

The associated symptomatology can be varied and non-specific, with clinical manifestations of abdominal pain, intestinal bleeding, ischaemic colitis or the presence of an abdominal mass. Portal hypertension has been described in up to 50% of patients with arteriovenous fistulas of the splanchnic territory[3] and, in the most severe cases, may contribute to the development of heart failure[4].

The diagnosis of ischaemic colitis secondary to AVM can be complicated in some cases and can be confused with other inflammatory conditions. The therapeutic options available are percutaneous endovascular embolisation, resection of the affected bowel segment (left hemicolectomy in most cases) or a combination of both[5]. Embolisation may be the definitive treatment or a bridge to surgical intervention, however, it may carry a risk of intestinal ischaemia and recurrence of the AVM in some cases[1].

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The presence of AVMs in the abdominal territory represents a challenge for the clinician, highlighting the need to reach a diagnosis in order to offer appropriate treatment, as it can be life-threatening.

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CLINICAL CASE

CHRONIC MESENTERIC ISCHEMIA, AN UNDERVALUED CAUSE OF CONSTITUCIONAL SYNDROME

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Abstract

Chronic mesenteric ischaemia (CMI) is a rare gastrointestinal pathology, the prevalence of which varies according to case series. It is caused by reduced flow in the main mesenteric vessels, namely the celiac artery (CA), superior mesenteric artery (SMA) and inferior mesenteric artery (IMA), the most common aetiology being atherosclerosis. Pathophysiologically, there is an increase in collateral vessels between the CA, SMA and IMA, and the patient may be asymptomatic until advanced stages or only show symptoms in situations of increased flow demand, such as ingestion or physical exercise. These characteristics mean that CMI is an underdiagnosed pathology, frequently confused with other clinical entities such as constitutional syndrome and whose delayed diagnosis is associated with high morbidity and mortality.

We present the case of a patient with multiple vascular risk factors who was incidentally diagnosed with CMI during the endoscopic study due to clinical suspicion of constitutional syndrome. However, given the advanced stage of the patient, he finally required hospitalisation and surgery, which shows the importance of early diagnosis of this clinical entity.

Keywords: endoscopy, chronic mesenteric ischemia, constitutional syndrome.

Introduction

Chronic mesenteric ischaemia (CMI) is an uncommon digestive pathology in clinical practice, caused by a decrease in flow, due to low output or the presence of stenosis, in the main mesenteric vessels[1], the anatomy of which is detailed in table 1.

Clinically, less than 2% of patients present with the characteristic triad of abdominal pain, sitophobia and

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Celiac Trunk (CT)	It supplies the liver, stomach, pancreas (mainly pancreatic head), first-second duodenal portion and spleen.
Superior Mesenteric Artery (SMA)	It supplies the third-fourth duodenal portion, pancreas (mainly pancreatic tail), the entire small intestine and the right hemicolon.
Inferior mesenteric artery (IMA)	It supplies the distal colon.

Table 1. ***Visceral irrigation of each of the arterial vascular structures that constitute the mesenteric vascular territory.***

unintentional weight loss. This lack of symptoms is due to the fact that the bowel is protected from ischaemia by the existence of multiple collaterals between its main vessels, such as the pancreatoduodenal artery between the CA and the SMA or the marginal artery and the arc of Riolan between the SMA and the IMA (Figure 1). However, an increase in incidence has been observed over the last decade, secondary to population ageing and the increased prevalence of cardiovascular risk factors such as hypertension, diabetes or obesity, which is why it is increasingly diagnosed in gastroenterology consultations or endoscopy units[2].

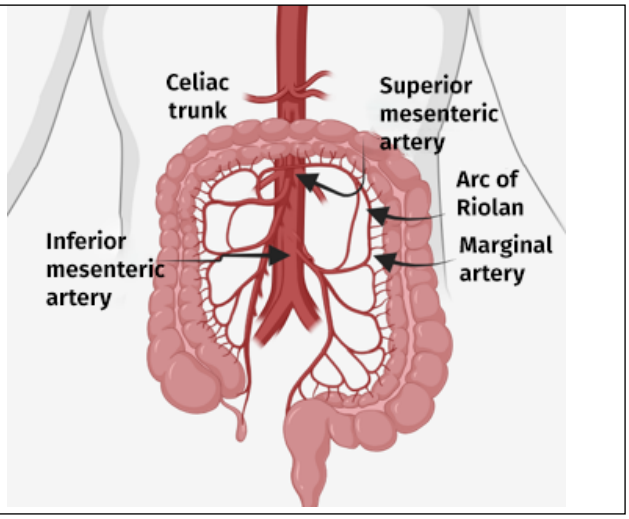

Figure 1. ***Diagram of the mesenteric arterial circulation.***

The characteristic symptoms of CMI are frequently observed in patients with constitutional syndrome, and CMI and other non-neoplastic digestive pathologies, such as peptic disease or chronic pancreatitis, should be included in the differential diagnosis[3]. All of these pathologies, including CMI, are associated with a high morbidity and mortality rate, which is directly proportional to the delay in diagnosis and initiation of therapeutic measures. Initially, conservative management with hygienic-dietary measures and control of cardiovascular risk factors is chosen, opting for a surgical approach in the case of symptomatic patients or those with significant vascular involvement[4].

Clinical case

We present the case of a 65-year-old patient with several cardiovascular risk factors, including arterial hypertension, dyslipidaemia, type II diabetes and active smoking. He had a history of chronic ischaemia in the lower limbs secondary to generalised atherosclerotic disease, and was being treated with cilostazol and pentoxifylline.

The patient came to the outpatient department of the Digestive System, referred from Primary Care, due to abdominal pain in the mesogastrium, predominantly postprandial, together with hyporexia and weight loss of up to 20 kg in the last three months. Laboratory tests showed hypoproteinemia (6 g/dl) with hypoalbuminemia (2.8 g/dl) and slight alteration of the liver profile (GOT 48 U/L, GPT 58 U/L, GGT 188 U/L, FA 91 U/L) with no other significant alterations.

Given the existence of alarm symptoms, it was decided to perform a complete endoscopic study with gastroscopy and colonoscopy. In this study, patchy areas of erythema and whitish mucosa were observed together with fibrinous ulcers-erosions, some of them excavated, with congestive edges, suggesting an ischaemic origin. They are mainly located in the distal gastric body, gastric antrum, duodenum and ascending colon (Figures 2-5). Histological study was compatible with chronic inflammatory infiltrate without signs of intestinal metaplasia and lymphoplasmacytic colitis in the proximal colon samples. Given the unspecificity of the histological findings and the high suspicion of an underlying ischaemic disorder, AngioCT was requested to complete the aetiological study.

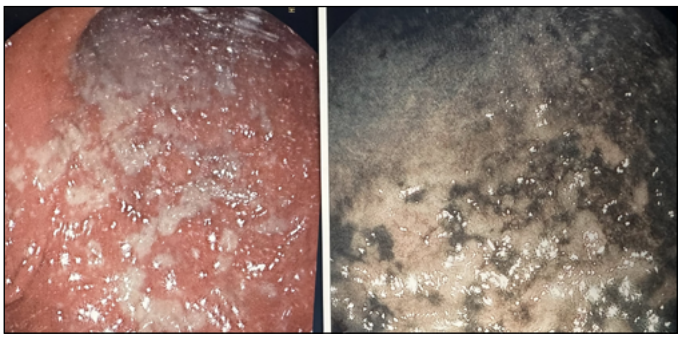

Figure 2. ***Distal gastric mucosa at its junction with gastric antrum with patchy areas of whitish mucosa more marked towards the lesser curvature and antrum that appear ischaemic in nature visualised with white light (left) and with NBI light (right).***

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Figure 3. ***Retro-vision image showing involvement of the distal gastric body, with respect for the fornix and proximal gastric body.***

Figure 4. ***Second duodenal portion with patchy areas of erythema and areas of whitish mucosa, with isolated fibrinous ulcer-erosions.***

A few days after the assessment in the consultation room, the patient came to the emergency department with severe abdominal pain and postprandial vomiting of food, with no other symptoms of interest. Examination revealed generalised pain on palpation with no signs of peritonism. An urgent abdominal CT scan showed critical stenosis at the origin of the celiac trunk with filiform filling of distal arteries and complete proximal occlusion of the superior mesenteric artery with signs of distal repermeabilisation by collaterals (a finding that suggests chronic ischaemia), both vascular occlusions caused by atheromatous plaques (Figure 6). Secondary signs of hepatic and splenic hypoperfusion are observed, without signs of intestinal loops suffering.

Figure 5. ***Angio-CT reconstruction with critical stenosis at the origin of the celiac trunk and proximal occlusion of the superior mesenteric artery with filiform filling of the distal arteries.***

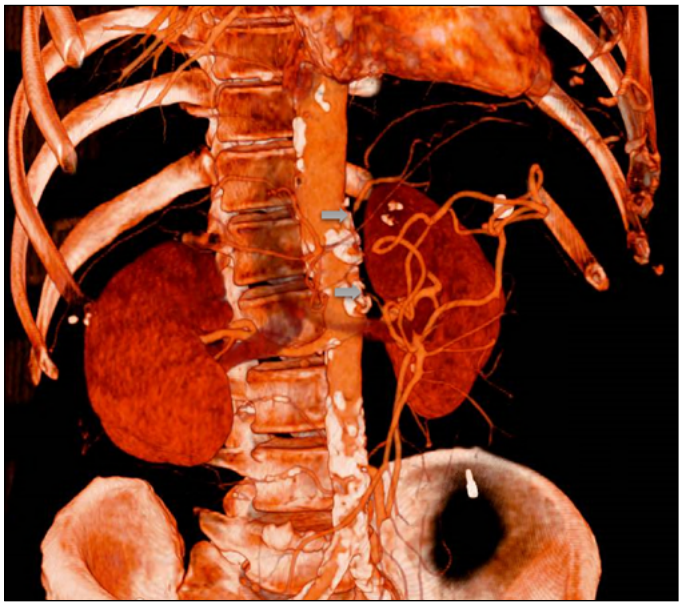

Figure 6. ***Proximal ascending colon with patchy areas of whitish mucosa and some isolated fibrinous erosions, more marked in the ileocaecal valve and cecum.***

The patient was admitted and initially opted for endovascular surgical treatment with the intention of recanalising the superior mesenteric artery, which failed, with persistent postprandial abdominal symptoms. Finally, it was decided to recanalise the superior mesenteric artery by means of an aorto-mesenteric bypass, and the patient subsequently showed a slight clinical improvement.

Currently, the patient continues to be followed up by the Angiology and Vascular Surgery Department. However, given that he continues to smoke, postprandial symptoms have reappeared, together with worsening of the paraesthesias in the lower limbs.

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Discussion

Chronic mesenteric ischaemia (CMI) is an underestimated digestive pathology, the late diagnosis of which is associated with an increased risk of disability and mortality. CMI occurs due to occlusive or non-occlusive causes, and the overall prevalence is unknown. In post-mortem forensic series, a prevalence of 6-29% is estimated, exceeding 67% in series of patients over 80 years of age. However, less than 2% of patients with critical stenosis of the mesenteric vessels will present with symptoms as the intestinal vasculature is characterised by collateral circulation[2]. Chronic occlusive mesenteric ischaemia may be secondary to atherosclerosis, compression of the arcuate ligament of the diaphragm, fibrodysplasia, as well as, other rare causes, such as vasculitis or thrombotic disease. Arteriosclerosis is the most common aetiology, being characteristic in women and associated with cardiovascular risk factors such as smoking, hypertension, diabetes, dyslipidaemia or family history of cardiovascular disease[4].

The most characteristic symptom of CMI is the onset of predominantly postprandial abdominal pain, starting 15-30 min after ingestion and lasting 2-4 hours (74-100% of cases). As a result, 90% of patients eventually develop a phobia of eating, reducing the frequency and quantity of their meals. Secondarily, involuntary weight loss of up to 15 kg occurs (61-94% of cases), thus completing the characteristic clinical triad: predominantly postprandial abdominal pain, sitophobia and weight loss[5]. Thus, CMI can easily be confused with a constitutional syndrome, also known as asthenia-anorexia-cachexia syndrome, for which the diagnostic criterion is a loss of at least 5% of body weight for at least 6 months[6]. Although it is generally accepted that, a priori, a neoplastic process underlies every constitutional syndrome, we must not forget other clinical entities such as non-neoplastic digestive diseases, especially CMI.

There are multiple non-standardised diagnostic algorithms that recommend imaging tests (abdominal ultrasound or abdominal CT) and/or endoscopic techniques in the presence of a constitutional syndrome, in order to avoid under-diagnosing digestive causes of neoplastic or non-neoplastic origin. The characteristic endoscopic findings of CMI are oedema (35%), erythema (42%), mucosal atrophy and erosions or ulcers (60-80%); and they usually show continuous involvement, depending on the vascular territory affected[2].

Treatment of early-stage CMI includes control of cardiovascular risk factors and lifestyle modification, especially smoking cessation. However, when symptoms appear, a surgical approach using endovascular or open procedures is recommended[1,2].

In conclusion, CMI is an infrequent pathology with a high percentage of asymptomatic patients and whose clinical presentation resembles constitutional syndrome, we should include it in the differential diagnosis of all patients with involuntary weight loss, especially if there are added cardiovascular risk factors. On the other hand, despite the lack of specificity of the endoscopic and histological findings, endoscopists should consider it especially if these findings are limited to a specific vascular territory, as was the case in our patient, and when other more frequent causes such as neoplasms, *H. pylori* infection or consumption of non-steroidal anti-inflammatory drugs have been ruled out. High clinical suspicion and endoscopic findings were crucial in this case, and complications and high morbidity and mortality secondary to diagnostic delay could be avoided.

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