**References**
1. Sandborn WJ, *et al. Gastroenterology*, 2017;152:S1308–9.

## P274

## Factors driving treatment escalation in Crohn's disease in the CALM trial

W. Reinisch[1]*, R. Panaccione[2], P. Bossuyt[3], F. Baert[4], A. Armuzzi[5], X. Hebuterne[6,7], S. Danese[8], W.J. Sandborn[9], S. Schreiber[10], G. D'Haens[11], S. Berg[12], Q. Zhou[13], J. Petersson[13], E. Neimark[13], A.M. Robinson[13], J.-F. Colombel[14]
[1]*Medical University of Vienna, Vienna, Austria,* [2]*University of Calgary, Calgary, Canada,* [3]*Imelda General Hospital, Bonheiden, Belgium,* [4]*AZ Delta Roeselare-Menen, Menen, Belgium,* [5]*Presidio Columbus, Fondazione Policlinico Gemelli Università Cattolica, Rome, Italy,* [6]*Service de Gastroentérologie et Nutrition Clinique, Nice, France,* [7]*Université de Nice-Sophia-Antipolis, Nice, France,* [8]*Istituto Clinico Humanitas, Milan, Italy,* [9]*University of California San Diego, La Jolla, USA,* [10]*University Hospital Schleswig-Holstein, Kiel, Germany,* [11]*Academic Medical Center, Amsterdam, The Netherlands,* [12]*AbbVie AB, Solna, Sweden,* [13]*AbbVie Inc., North Chicago, USA,* [14]*Icahn School of Medicine at Mount Sinai, New York, USA*

**Background:** Patients with Crohn's disease (CD) from the tight control (TC) management group of CALM, whose treatment was escalated based on clinical symptoms and biomarkers (CD Activity Index [CDAI], C-reactive protein [CRP], faecal calprotectin [FC] and/or prednisone use), achieved better endoscopic outcomes than patients whose treatment was escalated based on CDAI and prednisone use only.[1] The majority of decisions to escalate in the TC group included FC followed by CRP, CDAI, and prednisone use, however, a detailed algorithm of treatment decisions based on the four criteria has not been described. This analysis reports treatment escalation decisions in the TC group in more detail.
**Methods:** A total of 122 patients naïve to biologics and immunosuppressants with or without prior prednisone use were randomised to the TC group. At randomisation, patients received treatment with adalimumab (ADA) induction 160/80 mg and then 40 mg every other week (EOW) if they met at least one criterion, i.e., CDAI ≥150, CRP ≥5 mg/l, FC ≥250 µg/g, and prednisone use. At 12, 24, and 36 weeks after randomisation, patients, who met at least one of the CDAI, CRP, FC, or prednisone criteria were escalated consecutively to the next treatment option (ADA 40 mg every week [EW], followed by ADA 40 mg EW+2.5 mg/kg azathioprine). Patients who did not meet a treatment escalation criterion were to stay at the same treatment. CDAI, FC, CRP and prednisone use were assessed one week before randomisation and treatment escalation. Dose escalation criteria were summarised and reported as observed in all patients who met escalation criteria at each time point.
**Results:** Over 70% of patients who qualified to receive ADA at randomisation met three to four criteria (Table). After randomisation, approximately 50% or more patients were escalated to the next treatment option based on one criterion. FC was the most frequent single reason of escalation followed by CRP and CDAI at 12 and 24 weeks. At 36 weeks, there were fewer escalations to which FC, CRP, and CDAI contributed equally. Among two reasons for escalation, the FC+CRP combination was the most prevalent. Overall, prednisone use was the least frequent criterion for escalation.

**Table.** Number of patients in the TC group who met escalation criteria at and after randomisation.

| Escalation Criteria Met | Patients who received ADA at randomisation and were escalated after randomisation | | | |
|---|---|---|---|---|
| | Randomisation n (%) n=111 | 12 weeks n (%) n=76 | 24 weeks n (%) n=55 | 36 weeks n (%) n=20 |
| **1 criterion** | 7 (6.3) | 35 (46.1) | 34 (61.8) | 15 (75.0) |
| CDAI | 3 (2.7) | 8 (10.5) | 9 (16.4) | 4 (20.0) |
| Prednisone | 0 | 2 (2.6) | 1 (1.8) | 2 (10.0) |
| CRP | 2 (1.8) | 10 (13.2) | 9 (16.4) | 5 (25.0) |
| FC | 2 (1.8) | 15 (19.7) | 15 (27.3) | 4 (20.0) |
| **2 criteria** | 23 (20.7) | 25 (32.9) | 14 (25.5) | 4 (20.0) |
| CDAI+Prednisone | 1 (0.9) | 4 (5.3) | 2 (3.6) | 0 |
| CDAI+CRP | 5 (4.5) | 2 (2.6) | 3 (5.5) | 0 |
| CDAI+FC | 7 (6.3) | 6 (7.9) | 3 (5.5) | 1 (5.0) |
| Prednisone+CRP | 2 (1.8) | 1 (1.3) | 0 | 0 |
| Prednisone+FC | 0 | 3 (3.9) | 0 | 0 |
| CRP+FC | 8 (7.2) | 9 (11.8) | 6 (10.9) | 3 (15.0) |
| **3 criteria** | 43 (38.7) | 11 (14.5) | 5 (9.1) | 1 (5.0) |
| CDAI+Prednisone+CRP | 9 (8.1) | 0 | 0 | 0 |
| CDAI+Prednisone+FC | 13 (11.7) | 0 | 0 | 0 |
| CDAI+CRP+FC | 17 (15.3) | 10 (13.2) | 3 (5.5) | 0 |
| Prednisone+CRP+FC | 4 (3.6) | 1 (1.3) | 2 (3.6) | 1 (5.0) |
| **4 criteria (CDAI+Prednisone+CRP+FC)** | 38 (34.2) | 5 (6.6) | 2 (3.6) | 0 |

**Conclusions:** The data from CALM suggest that biomarkers are important for guiding treatment decisions in patients with early CD after their symptoms are controlled by treatment. These results underscore the importance of monitoring biomarkers along with clinical symptoms to achieve better clinical and endoscopic outcomes.
**Reference**
1. Colombel J-F, *et al. Lancet* [published online on October 31]

## P275

## Clinical characteristics, associated malignancies and management of primary sclerosing colangitis in inflammatory bowel disease patients: A Spanish nationwide study based on the ENEIDA registry

I. Guerra[1]*, L. Bujanda[2,3], J. Castro[4], O. Merino[5], J. Tosca[6], B. Camps[7], A. Gutiérrez[8], J. Gordillo[9], L. de Castro[10], M. Iborra[3,11], A.Y. Carbajo[12], C. Taxonera[13], I. Rodriguez - Lago[14], F. Mesonero[15], R. de Francisco[16], G.J. Gómez-Gómez[17], M. Chaparro[3,18], C.A. Tardillo[19], M. Rivero[20], A. Algaba[1], E. Martín-Arranz[21], F. Cañete[3,22], R. Vicente[23], B. Sicilia[24], B. Antolín[25], V. Prieto[26], L. Márquez[27], J.M. Benítez[28], P. Camo[29], M. Piqueras[30], C.J. Gargallo[31], E. Hinojosa[32], J.M. Huguet[33], J.L. Pérez Calle[34], M. Van Domselaar[35], C. Rodriguez[36], X. Calvet[3,37], M.C. Muñoz[38], M.F. García-Sepulcre[39], F. Bermejo[1], J.M. Bañales[40], J.P. Gisbert[3,18]
[1]*Hospital Universitario de Fuenlabrada, Department of Gastroenterology, Fuenlabrada, Spain,* [2]*Hospital Donostia/Instituto Biodonostia, Universidad del País Vasco UPV/EHU, Department of Gatroenterology, Donostia-San Sebastian, Spain,* [3]*Centro de Investigación Biomédica en Red de Enfermedades Hepáticas y Digestivas (CIBERehd), Spain, Spain,* [4]*Hospital Clinic de Barcelona, Department of Gastroenterology, Barcelona, Spain,* [5]*Hospital Universitario de Cruces, Department of Gastroenterology, Baracaldo, Vizcaya, Spain,* [6]*Hospital Universitario Clínico de*

*Valencia, Department of Medicine, University of Valencia, Valencia, Spain, [7]Hospital Universitari de Bellvitge-IDIBELL, Department of Gastroenterology, Barcelona, Spain, [8]Hospital General Universitario de Alicante, Department of Gastroenterology, Alicante, Spain, [9]Hospital de la Santa Creu i Sant Pau, Department of Gastroenterology, Barcelona, Spain, [10]Complexo Hospitalario Universitario de Vigo, Instituto de Investigación Biomédica. Estructura Organizativa de Xestión Integrada de Vigo, Department of Gastroenterology, Vigo, Spain, [11]Hospital Universitari i Politecnic La Fe, Department of Gastroenterology, Valencia, Spain, [12]Hospital Universitario Rio Hortega, Department of Gastroenterology, Valladolid, Spain, [13]Hospital Clínico San Carlos and Instituto de Investigación del Hospital Clínico San Carlos (IdISSC), Department of Gastroenterology, Madrid, Spain, [14]Hospital de Galdakao, Department of Gastroenterology, Galdakao, Vizcaya, Spain, [15]Hospital Universitario Ramón y Cajal, Department of Gastroenterology, Madrid, Spain, [16]Hospital Universitario Central De Asturias, Department of Gastroenterology, Oviedo, Spain, [17]Hospital 12 de Octubre, Department of Gastroenterology, Madrid, Spain, [18]Hospital Universitario de La Princesa, Gastroenterology Unit, IIS-IP, Madrid, Spain, [19]Hospital Universitario Nuestra Señora Candelaria, Department of Gastroenterology, Santa Cruz De Tenerife, Spain, [20]Hospital Universitario Marques de Valdecilla, Department of Gastroenterology, Unit EII, Santander, Spain, [21]Hospital Universitario La Paz, Department of Gastroenterology, Madrid, Spain, [22]Hospital Universitari Germans Trias I Pujol, Department of Gastroenterology, Badalona, Spain, [23]Hospital Universitario Miguel Servet, Department of Gastroenterology, Zaragoza, Spain, [24]Hospital Universitario de Burgos, Department of Gastroenterology, Burgos, Spain, [25]Hospital Clínico Universitario de Valladolid, Department of Gastroenterology, Valladolid, Spain, [26]Hospital Universitario de Salamanca, Department of Gastroenterology, Salamanca, Spain, [27]Parc de Salut Mar, Department of Gastroenterology, Barcelona, Spain, [28]Hospital Universitario Reina Sofia, Department of Gastroenterology, Córdoba, Spain, [29]Hospital General San Jorge, Department of Hepatology and Gastroenterology, Huesca, Spain, [30]Consorci Sanitari Terrassa, Terrassa, Spain, [31]Hospital Clínico Universitario "Lozano Blesa" and Aragón Health Research Institute (IIS Aragón), Department of Gastroenterology, Zaragoza, Spain, [32]Hospital de Manises, Department of Gastroenterology, Valencia, Spain, [33]Hospital General Universitario de Valencia, Department of Gastroenterology, Valencia, Spain, [34]Hospital Universitario Fundación Alcorcón, Department of Gastroenterology, Madrid, Spain, [35]Hospital Universitario de Torrejón, Department of Gastroenterology, Torrejón De Ardoz, Spain, [36]Complejo hospitalario de Navarra, Department of Gastroenterology and Hepatology, Pamplona, Spain, [37]Corporació Sanitària Universitària Parc Taulí, Universitat Autònoma de Barcelona, Sabadell, Spain, [38]Hospital Universitario de Basurto, Department of Gastroenterology, Bilbao, Spain, [39]Hospital de Elche, Department of Gastroenterology, Alicante, Spain, [40]Head, Liver Diseases Group Biodonostia Research Institute-Donostia University Hospital- Assistant Professor of Medicine/Sciences University of Navarra, USA., Donostia, Spain*

**Background:** Primary sclerosing colangitis (PSC) is a chronic cholestasic liver disease usually associated with inflammatory bowel disease (IBD). An increased risk of malignancies (colorectal cancer and cholangiocarcinoma) has been reported in IBD patients with PSC. Our aim was to determine the clinical characteristics, associated malignancies and management of PSC in Spanish IBD patients.
**Methods:** PSC patients with IBD were identified from the Spanish prospectively maintained ENEIDA registry of GETECCU. Additional data were collected using REDCap electronic data capture tool hosted at AEG (Asociación Española de Gastroenterología).
**Results:** We identified 245 patients with PSC and IBD, 69% males, 96% Caucasian, 67% with ulcerative colitis, 29% Crohn's disease and 4% with unclassified colitis. Median age at diagnosis of PSC was 38 years (IQR 27–51). PSC was diagnosed after a median of 60 months (IQR 1–147) of IBD diagnosis, being PSC diagnosed before IBD in 12% of patients. Magnetic resonance cholangiopancreatography was the most common diagnostic technique used (77%). Liver biopsy was obtained in 72 patients (29%). Most of the patients (65%) were asymptomatic at PSC diagnosis. In symptomatic patients, pruritus was reported in 28 patients (11%), abdominal pain in 23 (9.4%), jaundice in 18 (7.3%), asthenia in 17 (6.9%) and cholangitis in 9 (3.7%). Phosphatase alkaline was permanently elevated in 53% of patients. IgG4 was positive in 20% of patients, ANA in 38%, SMA in 24% and AMA in 3.1%. Treatment with ursodeoxycholic acid was prescribed in 206 patients (84%) during a median of 70 months (IQR 36–130), with a mean dose of 14 ± 4mg/kg/d. Twenty-one patients (8.6%) required liver transplantation during follow-up, with post-transplantation complications in 9 of them (43%). Sixty-eight percent of patients followed a screening program with colonoscopy every 1–2 years, detecting 9 patients with high-grade dysplasia or adenocarcinoma (5.4%). Thirteen patients (5.3%) developed colorectal cancer, 85% after PSC diagnostic, and 7 patients (2.9%) were diagnosed with cholangiocarcinoma (3 intrahepatic, 4 extrahepatic). Surgery was performed in 2 patients with cholangiocarcinoma, 2 were endoscopically resected and 2 patients received chemotherapy and/or radiotherapy. Having a longer evolution of IBD was the only factor associated with the appearance of colorectal cancer in the multivariate analysis ($p = 0.01$), without finding any factor associated with cholangiocarcinoma development.
**Conclusions:** In this nationwide study it is confirmed that the association of IBD and PSC is more common in males with ulcerative colitis, being PSC usually asymptomatic at diagnosis. Colorectal cancer is more frequent in patients with a longer evolution of IBD and after PSC diagnosis.

## P276

## Risk matrix model for prediction of one-year surgery in Crohn's disease

N. Imperatore*, A. Rispo, A. Testa, L. Bucci, G. Luglio, G.D. De Palma, M. Rea, O.M. Nardone, N. Caporaso, F. Castiglione
*School of Medicine Federico II of Naples, Department of Clinical Medicine and Surgery, Naples, Italy*

**Background:** Having a simple score able to predict the risk of surgery, combining clinical, endoscopic and imaging features, could tailor the management of patients with Crohn's disease (CD).
Aim: to prospectively evaluate the one-year risk factors for surgery in CD and to build a risk matrix model for predicting the 1-year probability of surgery.
**Methods:** Enrolled CD patients underwent prospectively clinical, laboratory, endoscopy and bowel sonography (BS) examinations within one week. Firstly, the optimal cut-off values for Simple Endoscopic Score for CD (SES-CD), bowel wall thickness (BWT) at BS and disease extension at BS in predicting surgery were identified by ROC curves; then binary logistic regression and Cox's regression were conducted. Finally, the probabilities of surgery were computed for selected baseline levels of covariates and results were arranged in a prediction matrix model.