INFLAMMATORY BOWEL DISEASE

Phenotype at diagnosis predicts recurrence rates in Crohn's disease

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Background: In Crohn's disease (CD), studies associating phenotype at diagnosis and subsequent disease activity are important for patient counselling and health care planning.

Aims: To calculate disease recurrence rates and to correlate these with phenotypic traits at diagnosis.

Methods: A prospectively assembled uniformly diagnosed European population based inception cohort of CD patients was classified according to the Vienna classification for disease phenotype at diagnosis. Surgical and non-surgical recurrence rates throughout a 10 year follow up period were calculated. Multivariate analysis was performed to classify risk factors present at diagnosis for recurrent disease.

Results: A total of 358 were classified for phenotype at diagnosis, of whom 262 (73.2%) had a first recurrence and 113 patients (31.6%) a first surgical recurrence during the first 10 years after diagnosis. Patients with upper gastrointestinal disease at diagnosis had an excess risk of recurrence (hazard ratio 1.54 (95% confidence interval (CI) 1.13–2.10)) whereas age ≥ 40 years at diagnosis was protective (hazard ratio 0.82 (95% CI 0.70–0.97)). Colonic disease was a protective characteristic for resective surgery (hazard ratio 0.38 (95% CI 0.21–0.69)). More frequent resective surgical recurrences were reported from Copenhagen (hazard ratio 3.23 (95% CI 1.32–7.89)).

Conclusions: A mild course of disease in terms of disease recurrence was observed in this European cohort. Phenotype at diagnosis had predictive value for disease recurrence with upper gastrointestinal disease being the most important positive predictor. A phenotypic North-South gradient in CD may be present, illustrated by higher surgery risks in some of the Northern European centres.

Crohn's disease (CD) is a chronic inflammatory condition that can be distributed throughout the entire gastrointestinal tract. There has been controversy regarding its aetiology. Both infectious agents such as *Mycobacterium paratuberculosis*¹ as well as the autoimmune pathogenetic route² have been proposed. The course of CD is variable and has been notoriously difficult to predict based on information acquired at the moment of diagnosis.^{3,4} For clinical purposes, categorisation is desirable to correlate a restricted number of identifiable phenotypic subgroups at diagnosis with clinical outcome measures during subsequent follow up. This may enable the clinician to classify patients at diagnosis into high and low risk groups and to inform patients more precisely, early after diagnosis, about their possible disease course. Furthermore, it may serve as background information to direct trials of long term therapeutic strategies for subgroups of patients early during the course of disease. Correlates of "phenotype at diagnosis" and "disease recurrence" should preferably be evaluated in unselected patient samples, gathered within a restricted time frame, and using uniform diagnostic criteria.⁵

The aim of this study was to evaluate whether CD phenotype at diagnosis correlates with surgical and non-surgical recurrence rates during the first 10 years after diagnosis in a population based European inception cohort.

MATERIALS AND METHODS

Patients and centres

Between October 1991 and September 1993, the European Collaborative Study Group of Inflammatory Bowel Disease (EC-IBD) created a prospective and uniformly diagnosed

population based inception cohort of 2201 patients afflicted with inflammatory bowel disease (IBD) within 20 well described geographical areas in 12 European countries.⁶ In this cohort, 706 CD patients were diagnosed with CD, 1379 with ulcerative colitis, and 116 with indeterminate colitis.⁷

In the present follow up study, all centres that originally participated in the EC-IBD cohort were approached to take part. Thirteen of the original 20 centres from nine countries participated, including 483 of the original 706 CD patients (68%). Study areas and study area populations have been described in detail previously.⁷ Patients were followed up from inception (1 October 1991 to 30 September 1993) until data inclusion (1 August 2002 to 31 January 2004), or any date prior to that data inclusion period, indicated to be either the date of death or "lost to follow up" (LTFU).

Before the start of data collection, an arbitrarily chosen minimal response rate per centre was set at 60%. Centres that did not comply with this minimum response rate were analysed separately.

Ten year clinical follow up project

The present study was part of a large 10 year clinical follow up study project, encompassing multiple aspects of disease outcome in patients with IBD. This study project was launched in 1998, granted by the European Commission in 2000, started in 2001, and was finished in February 2005. Details of the methods used in this project have been

Abbreviations: CD, Crohn's disease; EC-IBD, European Collaborative Study Group of Inflammatory Bowel Disease; PQ, patient questionnaire; IBD, inflammatory bowel disease; LTFU, lost to follow up

extensively described elsewhere.⁸ Apart from collection of clinical data on vital status, disease activity, medication use, and surgical interventions, patients were invited to complete a patient questionnaire (PQ). The PQ had questions on demographic details, lifestyle, and family history. Data inclusion was facilitated by an electronic utility available on an internet based website.⁹

Definitions

Patients were retrospectively characterised according to the Vienna classification.¹⁰ Recurrences were dated on the 10 year follow up time scale and recorded as being either surgical or non-surgical. A surgical recurrence was defined as an episode of increased disease activity requiring surgical intervention either with or without medication change. Surgical recurrence was further classified as resective (part of the bowel was resected) or any other type of surgery (including abscess drainages, fistula operations, etc). A non-surgical recurrence was defined as an episode of increased disease activity requiring only medication change. In the present study, "all type recurrences" (surgical plus non-surgical recurrences), "surgical recurrences" (resective plus non-resective surgical recurrences), and "resective surgical recurrences" are presented as end points.

Statistical analysis

Cox regression analysis was applied to identify risk factors for first, second, and third recurrences. Values for the patient groups with disease location in the terminal ileum, inflammatory disease, and those resident in Vigo were randomly chosen as references to calculate hazard ratios for disease location, disease behaviour, and residential groups, respectively. Survival curves displaying cumulative recurrence rates for each phenotypic subgroup were derived with the Kaplan-Meier method. For both these analyses the Statistical Package for the Social Sciences (SPSS 11.5.1 for Windows; SPSS Inc., Chicago, Illinois, USA) was used. A Cox proportional hazard frailty model¹¹ was applied to identify risk factors for disease recurrence incorporating all recurrences

that occurred throughout the observation time, using the Stata statistical software package.¹²

In both multivariate models, apart from phenotypic characteristics at diagnosis, sex, place of residence, educational status, smoking behaviour, oral contraceptives, appendectomy, and family history of IBD at that moment were considered as confounding factors. In the Cox regression analyses, disease recurrence rates were adjusted for both 5-ASA and azathioprine use.

RESULTS

Follow up

Ten of the participating 13 centres from seven European countries and Israel complied with the minimal 60% response threshold, leaving 380 patients of whom 282 (74%) had a complete dataset at follow up (median follow up duration 123 months (range 107–141)). The remaining 98 patients were LTFU. Nine of these appeared to have a date of last visit the same as date of diagnosis and 13 had an unknown disease location and/or disease behaviour at diagnosis: these 22 patients were excluded from the analysis. The remaining 76/98 patients LTFU with known disease phenotype at diagnosis (median follow up duration 49 months (range 1–120)), including 36 deaths (median time from diagnosis until death 51 months (range 3–120)), were lost at varying times throughout the observation period and remained in the analysis until the time of loss or death. Details of patients who died during this observation have been presented previously.¹³ Patients ≥ 40 years at diagnosis were more likely to be LTFU (41 of 117 (35%) v 35 of 241 (14.5%) for patients < 40 years at diagnosis). The difference appeared to be caused by deaths that occurred in the older patient group (33 deaths in patients ≥ 40 years versus three in patients < 40 years at diagnosis). Furthermore, patients with colonic disease at diagnosis were more likely to be LTFU compared with the other disease location groups (39 of 76 patients LTFU had a colonic disease location (51%) v 112 of 282 (39%) with completed follow up). This was because 21 patients died who had a colonic disease location at diagnosis. Patients

Table 1 Number of patients with complete follow up and lost to follow up according to disease phenotype at diagnosis and place of residence

Total No of patients (n = 358)	Total	Complete follow up	Lost to follow up	
			Lost during course	Dead
Age at diagnosis				
≤ 40 y	241	206 (86)	32 (13)	3 (1)
≥ 40 y	117	76 (65)	8 (7)	33 (28)
Disease location at diagnosis				
Terminal ileum	53	45 (85)	5 (9)	3 (6)
Colon	151	112 (74)	18 (12)	21 (14)
Ileocolon	134	110 (82)	14 (10)	10 (8)
Upper gastrointestinal	20	15 (75)	3 (15)	2 (10)
Disease behaviour at diagnosis				
Inflammatory	264	207 (78)	29 (11)	28 (11)
Strictureing	57	47 (82)	4 (7)	6 (11)
Penetrating	30	23 (77)	5 (17)	2 (6)
Strictureing and penetrating	7	5 (71)	2 (19)	0
Residence				
Almada, Portugal	9	5 (55)	4 (45)	0
Beer Sheva, Israel	20	15 (75)	4 (20)	1 (5)
Copenhagen, Denmark	58	42 (72)	6 (10)	10 (18)
Cremona, Italy	9	8 (89)	0	1 (11)
Ioannina, Greece	6	4 (66)	1 (17)	1 (17)
Heraklion, Greece	15	8 (53)	4 (27)	3 (20)
Oslo, Norway	103	85 (82)	8 (8)	10 (10)
Reggio Emilia, Italy	32	24 (75)	5 (16)	3 (9)
South Limburg, the Netherlands	77	66 (86)	7 (9)	4 (5)
Vigo, Spain	30	25 (83)	2 (7)	3 (10)

Values are number (percentage of total).

Figure 1 Cumulative first, second, and third recurrence rates for the entire cohort for all recurrences (A) and surgical recurrences (B). n, number of patients at risk.

≥ 40 years at diagnosis were more likely to have colonic disease ($p = 0.03$). Patients LTFU were not different compared with patients with complete follow up in terms of sex and disease behaviour at diagnosis. Details of the 358 patients remaining for recurrence analyses are summarised in table 1.

Recurrence rates

Figure 1 shows cumulative recurrence rates for the entire cohort for all recurrences and surgical recurrences. Of 358 patients observed in this population based cohort, 96 patients (26.8%) experienced no recurrence, 63 (17.6%) had one recurrence, and 45 (12.6%) had two all type disease recurrences during the first 10 years after diagnosis.

First all type cumulative recurrence rates were 34%, 69.2%, and 77.5% one, five, and 10 years after diagnosis, respectively, whereas first surgical recurrence rates were 9.4%, 27.3%, and 33.5% one, five, and 10 years after diagnosis, respectively. Of 358 patients at risk, 25% had an all type recurrence after seven, 50% after 27, and 75% after 104 months whereas 25% were operated on within the first 58 months after diagnosis. The majority of patients (96 of 110) with a first surgical recurrence had resective surgery; first resective surgical recurrence rates were 7.1%, 21.2%, and 28.9% one, five, and 10 years after diagnosis, respectively. Within the first 89 months after diagnosis, 25% underwent a surgical procedure entailing bowel resection.

Second all type cumulative recurrence rates were 40.2%, 76.9%, and 82.6% one, five, and 10 years after the first recurrence, respectively, whereas second surgical recurrence rates were 17.8%, 32.3%, and 53.6% one, five, and 10 years after the first surgical episode, respectively. Of 258 patients at risk, 25% had an all type second recurrence after seven, 50% after 23, and 75% after 63 months following the first episode, whereas in the case of second surgical recurrences in 110 patients at risk, 25% recurred within 39 and 50% within 115 months following the first surgical episode. All type (both surgical and non-surgical) recurrence rates were 5.7%, 37%, and 63% one, five, and 10 years after the first surgical

episode, respectively. Second resective surgical recurrence rates were 2.2%, 18.5%, and 35.9% one, five, and 10 years after the first bowel resection, respectively. Within the first 88 months after the first resective surgery, 25% of 93 patients at risk had been operated on for a second bowel resection. All type (both surgical and non-surgical) recurrence rates were 1.1%, 36.8%, and 57.4% one, five, and 10 years after the first resective surgical episode, respectively.

Third all type cumulative recurrence rates were 45.9%, 62.9%, and 76.4% one, two, and five years after the second recurrence, respectively, whereas third surgical recurrence rates were 15.5%, 21.3%, and 49.6% one, two, and five years after the second surgical episode, respectively. Of 187 patients at risk, 25% had an all type third recurrence after six, 50% after 14, and 75% after 68 months following the second episode, whereas in the case of third surgical recurrences of 39 patients at risk, 25% recurred within 35 and 50% within 76 months following the second surgical episode. All type (both surgical and non-surgical) recurrence rates were 34.6% and 66.2% one and five years after the second surgical episode, respectively. Third resective surgical recurrence rates were 5.9%, 13.1%, and 39.8% one, two, and five years after the second bowel resection, respectively. Twenty five per cent of the 17 patients at risk had been operated on for a third bowel resection within the first 40 months after the second resective surgery. All type (both surgical and non-surgical) recurrence rates were 11.8% and 61.8% one and five years after the second resective surgical episode, respectively.

Risk factors

Patients with upper gastrointestinal disease location at diagnosis were found to have an increased risk for the first all type recurrence ($p = 0.05$). Patients using 5-ASA treatment were more likely to experience a first ($p < 0.01$) and second ($p = 0.02$) all type and first ($p < 0.01$) and second ($p < 0.01$) surgical type of recurrence. Azathioprine use was not associated with either an increased or decreased recurrence risk. In 116 of the 358 (32%) patients, data concerning smoking behaviour during the observation time

were not available. Apart from the 76 patients LTFU, 40 other patients whose clinical data were traced did not complete the PQ. Multivariate analysis of the 242 patients with complete PQ data showed that those who had stopped smoking after diagnosis were protected from first ($p = 0.01$) and second ($p = 0.05$) disease recurrences whereas in smokers no significant effect was observed. Patients older than 40 years at diagnosis were protected from a second all type recurrence ($p = 0.02$). More surgical ($p = 0.02$) and resective surgical first recurrences ($p < 0.01$) were reported from Copenhagen.

When all recurrences that had occurred throughout the entire observation period were combined in the Cox proportional hazard frailty model, upper gastrointestinal disease location at diagnosis was the only significant positive predictor for all types of recurrences ($p < 0.01$). Nearly all patients (19/20) with upper gastrointestinal disease location had non-penetrating disease. All seven patients within this group who were operated on had resective surgery. These seven had an excess risk for a second surgical recurrence. The Cox proportional hazard frailty model revealed age ≥ 40 years at diagnosis to be a protective characteristic with regard to disease recurrence of any type ($p = 0.01$). Colonic disease at diagnosis protected from resective surgery ($p < 0.01$). In both all type and resective surgical categories, significantly more recurrences were reported from Copenhagen ($p = 0.04$ and $p = 0.01$, respectively). Significantly less recurrences of all type were reported from Ioannina ($p = 0.03$), Oslo ($p < 0.01$), and Reggio Emilia ($p = 0.04$).

DISCUSSION

This European population based clinical 10 year follow up study of patients with CD showed a relatively benign disease course overall and provided evidence that phenotype at diagnosis has a predictive value for disease recurrence.

Previous studies reporting on surgical and non-surgical recurrence rates for different phenotypes (being either population based or from referral centres) differed in terms of geographical origin, selection of patients, era of observation, duration of follow up, treatment habits, and surgical techniques applied. The use of population based cohorts is obligatory to describe true phenotype disease outcome correlations. Most previous population based studies reporting on disease outcome and analysing inception periods of one or more decades were from a single geographical area⁵ in contrast with the present continent wide approach.

Our study had several strong methodological characteristics. Firstly, a population based prospectively and uniformly diagnosed inception cohort of CD patients was used, originating from 10 well described geographical areas in seven European countries and Israel. Secondly, patient inclusion took place during a short period of two years. All patients were followed throughout the same time period and had approximately identical observation times, except for those who died or were lost to follow up. This minimised differences in treatment practices that could have influenced outcome during the course of the study as may be encountered in observational cohort studies that enrol new patients over decades. Thirdly, data collection was uniform and efficient. Fourthly, to our knowledge, no previous cohort study had the opportunity to correlate disease phenotype at diagnosis according to the Vienna classification with disease recurrence.

There were some limitations. Firstly, even though the total number of patients as a whole was large, some centres and clinical subgroups had low participation rates. Secondly, 50% of the original EC-IBD centres participated in this study (52% of the original CD patients). Possibly centres with more of an interest in IBD and therefore with more expertise in treatment participated, which might have been a source of

bias. The seven centres that refrained from participation did so because of technical and/or logistic reasons. However, this did not jeopardise the population based character of the study as all of the original participating centres had individually met the criteria for population based patient inclusion when the cohort was formed in 1991–1993. Thirdly, treatment modalities such as surgery and change in prescribed medication were used as indicators of disease activity. Inter-physician variability in surgical treatment of increased disease activity in CD has been demonstrated.¹⁴ Furthermore, recordings in patients' files concerning non-surgical recurrences may have varied between doctors and centres and thus hampered the analysis. In all centres involved in this study, physicians instructed their patients to treat themselves without prior consultation in cases of mildly increased disease activity. Such episodes of increased disease activity were mostly not reported in the patient files and were thus not included as recurrences. Therefore, the actual number of mild flares was possibly underestimated in this follow up study. However, there is no reason to assume that this underestimation would have been different between the different phenotypic groups. Fourthly, CD patient classification by pattern of disease behaviour at diagnosis was previously shown to yield only fair interobserver agreement.¹⁵ In the present study, uniform definitions were used to classify patients at diagnosis regarding disease location and disease behaviour. These findings were clearly reported in the patient files. Accordingly, the risk of interpretative classification was minimised. Fifthly, even though recent observations have correlated disease phenotype in conjunction with genotype and disease course,^{16–18} this study was not designed to incorporate genotypic features into the analyses.

In the present observation, the time intervals needed for 50% of patients at risk to suffer recurrence decreased from the first to the second to the third episodes for both all type recurrences (27, 23, and 14 months) and surgical recurrences (58, 39, and 35 months). This finding supports the previously reported phenomenon in CD patients of previous relapse rates positively influencing relapse rates during subsequent courses.^{3, 19} However, the course of disease in terms of disease recurrence, as observed in this European cohort, must be considered mild, as more than half (57%) of all patients had two recurrences or less during the 10 year observation period. The resective surgical disease recurrence rate in particular was low (28.9%) compared with previous population based observations, with recurrence rates varying between 34% and 71% 10 years after diagnosis.⁵ However, reoperation rates were comparable with previous observations.^{3, 20–21} Between 54% and 57% of patients who needed resective surgery during the first 15 years after diagnosis were found to be operated on within the first year after diagnosis in previous series.^{3, 21–22} This was not confirmed in the present study as approximately 25% of patients needing resective surgery within the first 10 years after diagnosis was operated on during the first year after diagnosis. Improved and possibly more aggressive early initial maintenance therapy policies during the last decade compared with earlier decades may have contributed to these differences. Additionally, increasing reluctance towards surgery in CD patients in general may have played a role.

Disease recurrence after diagnosis in CD, either surgical plus non-surgical^{3, 23} or surgical alone,^{3, 4, 20–24} and phenotypic risk factors at diagnosis have previously been studied in population based cohorts. Although not consistently reported in all of these studies, small bowel involvement, perianal disease, and age 45–59 years at diagnosis were found to be risk factors for surgical and/or non-surgical disease recurrence. The present study found upper gastrointestinal disease location and young age at diagnosis to be risk factors. Disease behavioural characteristics at diagnosis were not found to be

risk factors in the present study. This could be explained by the previously shown instability of these features during subsequent courses. In a large number of patients, inflammatory disease behaviour at diagnosis evolves into either stricturing or penetrating categories during the first 10 years after diagnosis.^{25–26}

The correlation between disease phenotype at diagnosis and surgical recurrence rates was reported in one referral centre based study, showing penetrating disease to be a risk factor for surgical recurrence.²⁷ In a larger number of referral centre based patient samples, post-surgical recurrence rates were correlated with phenotypic traits at first surgery.^{19–28–31} Established phenotypic risk factors for post-surgical disease recurrence were young age³² and perforating disease.^{19–27–29–33–34} The first mentioned finding was confirmed in the present study.

Other factors and disease related or therapeutic conditions have been related to reduction or increase in risk. Smoking is a firmly established lifestyle related risk factor for general or postoperative disease recurrence. In the present study, not smoking protected against disease recurrence. Smokers were not found to have an increased recurrence risk but this may have been influenced by the considerable number of patients whose data on smoking behaviour were not available.

The widespread introduction of different immunomodulatory maintenance treatment modalities over the last four decades has reduced both general and postoperative disease recurrence rates.^{35–38} In this study, adjustment for concomitant 5-ASA and azathioprine use during the follow up period was performed. 5-ASA use appeared to be positively correlated with disease recurrence. Distinction between remission induction and maintenance 5-ASA therapy was not possible. However, in most circumstances a combination of both goals is served by 5-ASA prescription to CD patients. As such, 5-ASA use must be regarded as an epiphenomenon of recurrent disease rather than a risk factor. Azathioprine use did not appear to be associated with such a positive effect on disease recurrence risk. This could be a sign of its potent immunomodulatory maintenance effect.

Patients from Northern European centres, especially Copenhagen, had the higher risk of surgical interventions. This may reflect differences in the prevalence of genetic defects and possibly a more aggressive disease phenotype in the North, but could also be a manifestation of different treatment policies. However, stenotic symptoms not alleviated by medical treatment and prestenotic dilatation were indicative of resections in patients from Copenhagen³⁹ and these criteria were no different from those used by other centres.

In conclusion, overall, a relatively benign disease course in a European population based cohort of CD patients was observed, as more than half of all patients had two recurrences or less during the first 10 years after diagnosis. Phenotype at diagnosis appeared to have clear predictive value for disease recurrence, with upper gastrointestinal disease being the most important positive predictor of disease recurrence. A phenotypic North-South gradient in CD may be present, as reflected by higher surgery risks in some of the Northern European centres compared with those in the South. Genotypic differences may explain this phenotypic gradient and should be the subject of further study.

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Table 1A Acknowledgements

Centre	Investigators	Contribution
Maastricht (project coordinator) Department of Gastroenterology and Hepatology, University Hospital Maastricht, Maastricht, The Netherlands	Mia Cilissen Marielle Romberg	Collected data Collected data
Almada Departments of Gastroenterology and Pathology, Almada Regional Health Department, Portugal	Paula Borralho Nunes	Collected data
Beersheva Department of Gastroenterology	Hagit Yona	Collected data
Copenhagen Department of Medical Gastroenterology, Herlev Hospital, University of Copenhagen, Herlev, Denmark	Vibeke Binder Pia Munkholm Lene Riis	Scientific advisor Scientific advisor Collected data
Cremona Servizio di Gastroenterologia, Ospedale di Cremona, Cremona, Italy	Paolo Bodini	Scientific advisor
Dublin Adelaide and Meath Hospital, Department of Gastroenterology, Trinity College, Tallaght, Dublin, Ireland	Colm O'Morain Angie Harrington	Scientific advisor Collected data
Firenze UO di Gastroenterologia, Policlinico di Careggi, Firenze, Italy	Guiseppe d' Albasio Andrea Messori	Scientific advisor Scientific advisor
Global Vitis Wattstraat 52, Sassenheim, the Netherlands	Frederik Wessels Ed van Hees Robert van Hees	Developed data acquisition tools
Heerlen Department of Pathology, Atrium Medisch Centrum Heerlen, the Netherlands	Marius Nap	Scientific advisor
Heraklion Department of Gastroenterology, University General Hospital, Heraklion, Crete, Greece	Iannis Mouzas, Athanasios G Pallis Ioannis G Vlachonikolis Maria Tzardi	Scientific advisor Collected data Scientific advisor Scientific advisor
Ioannina Division of Internal Medicine, University of Ioannina, Ioannina, Greece	Kostas Katsanos Michel Economou	Collected data Collected data
Leuven Department. of Gastroenterology, UZ Gasthuisberg, Universiteit Leuven, Leuven, Belgium	Paul Rutgeerts Sofie Joossens Greet Claessens	Scientific advisor Collected data Collected data
Maastricht MEMIC University of Maastricht, Maastricht, the Netherlands	Charles Limonard	Scientific advisor
Milan Universita degli Studi di Milano, Cattedra di Gastroenterologia, Milano, Italy	Tullio Ranzi Claudio Cortelezzi	Scientific advisor Collected data
Oslo Medical Department A, Rikshospitalet, University of Oslo, Oslo, Norway	Idar Lygren, Camilla Solberg Morten Vatn	Collected data, Collected data Scientific advisor

Table 1A Continued

Centre	Investigators	Contribution
Reggio Emilia Department of Internal Medicine and Gastroenterology, Arcispedale S Maria Nuova, Reggio Emilia, Italy	Giovanni Fornaciari Maria Grazia Mortilla	Scientific advisor, Collected data
Torino Divisione di Gastroenterologica, Largo Turati 62, Torin, Italy	Angelo Pera	Scientific advisor
Vigo Hospital Xeral de Vigo, Vigo, Spain	Juan Clofent, Mercedes Butron Victor Ruiz Ochoa	Collected data, Collected data Scientific advisor

EDITOR'S QUIZ: GI SNAPSHOT

An unusual chest infection

Clinical presentation

A 53 year old man fell down a flight of stairs. He presented to casualty complaining of shoulder and back pain. He was discharged with a diagnosis of musculoskeletal pain. Six weeks later he presented with pleuritic chest pain, dyspnoea, and indigestion. On examination he was tachypnoeic, tachycardic, febrile, and hypotensive. Chest x ray (fig 1) demonstrated right lower lobe consolidation and a pleural effusion. He was diagnosed with sepsis secondary to pneumonia and treated with antibiotics. A chest drain was inserted and drained purulent fluid which grew *Candida* and therefore fluconazole was commenced. Over the next few days his condition failed to improve. Further biochemical analysis of the pleural fluid indicated the presence of triglycerides and cholesterol, raising the suspicion of a chyle leak. Repeat chest radiograph showed re-accumulation of right pleural effusion. The chest drain was resited and it drained in excess of 4 litre over 24 hours.

Figure 1 Chest x ray showing right lower lobe consolidation and a pleural effusion.

Question

What was going on here? Was this a simple pneumonia?

See page 1164 for answer

This case is submitted by:

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