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Acute abdominal emergency due to infectious enteritis: an observational study comparing Campylobacter spp. to other enteric pathogens in children

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Summary

BACKGROUND: *Campylobacter* spp. are a frequent cause of gastroenteritis, presenting in some patients as an acute abdominal emergency. Here we describe the distinctive clinical characteristics of these patients.

METHODS: We designed a retrospective, single-centre, observational study. Children and adolescents under 18 years of age who had positive stool cultures for *Campylobacter* spp. during the period between June 1, 2008 and May 31, 2016 were identified from our database. Hospitalised patients with *Campylobacter* spp. were then matched for age and gender with patients hospitalised for gastroenteritis of other or unknown aetiology. Patients who had undergone abdominal radiographic investigation or had received a surgery consultation were included as "acute abdomen" (AA) cases. Demographics, clinical characteristics and management were compared between AA and non-AA cases.

RESULTS: One hundred and forty-one patients with cultures positive for *Campylobacter* spp. were included in the analysis. Nineteen patients were identified as AA cases. Fewer of these had diarrhoea (14/19, 74% vs 117/121, 97%; p = 0.02) and more reported a lower sense of general wellbeing (8/18, 44% vs 8/108, 7%; p <0.001). Localised pain (9/18, 50% vs 20/115, 17%; p = 0.002) and abdominal tenderness (2/18, 11% vs 0/111; p = 0.02) were also more common among AA cases. Forty-four patients with *Campylobacter* spp. infections were hospitalised and matched with 44 patients with gastroenteritis of other or unknown aetiology. *Campylobacter* spp. infection (risk ratio 3.6, 95% CI 1.3–9.7; p = 0.01) was positively correlated with being seen by a surgeon and/or a prescription for radiological examination.

CONCLUSIONS: We identified a subset of patients with *Campylobacter* spp. gastroenteritis who present as an acute abdominal emergency. The presentation of these patients was characterised mainly by the nature of the associated abdominal pain.

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Introduction

Campylobacter species are a major cause of acute bacterial enterocolitis. Most infections are acquired through the consumption and handling of poultry. In Europe, the annual incidence varies from 1 case per 100,000 in Poland to more than 50 cases per 100,000 in Germany and the Netherlands [1]. In Switzerland, the incidence of campylobacteriosis increased to 100 cases per 100,000 inhabitants in 2016 [2].

The major symptom of campylobacteriosis is diarrhoea. In a school outbreak in Spain, 93.6% of 81 cases reported diarrhoea. Abdominal cramps, fever, nausea and vomiting were present in 89.6%, 61.5%, 29.7% and 28% of cases, respectively [3]. Asymptomatic carriage is also well described. In a prospective, population-based cohort study of gastroenteritis conducted in the UK, *Campylobacter* spp. documentation was reported in 0.7% of 2,264 controls compared to in 12.2% of 2,893 cases [4]. Asymptomatic carriage may be even more common in low-income countries. In a cohort study conducted in Mexico, only 30% of children under five years of age with positive stool cultures for *Campylobacter* spp. were symptomatic [5].

Previous studies have reported that *Campylobacter* spp. enteritis (CSE) may present as an acute abdominal emergency. In several case reports and series of patients hospitalised with CSE, authors reported variable numbers of patients in whom appendicitis was suspected, necessitating radiological work-up and the scheduling of surgical procedures [6–16]. In most such cases, the possibility of appendicitis was ultimately excluded. According to Lamps, even when a *Campylobacter* spp. was isolated from the appendix, the latter was mostly macroscopically normal, without transmural inflammation [17].

In our regular paediatric practice, we observed some cases of CSE that presented as acute abdominal emergencies,

necessitating radiological work-up and/or surgical evaluation. The main objective of our study is to describe the distinctive clinical characteristics of these patients in comparison to the other patients with CSE. Our secondary objective is to compare hospitalised patients with CSE with patients hospitalised for gastroenteritis from other causes to determine whether the clinical presentation as an "acute abdomen" (AA) case can distinguish those two groups.

Patients and methods

Design, setting and population

This study is a retrospective, single-centre, observational study performed in a tertiary care hospital (Lausanne University Hospital, Lausanne, Switzerland). Children and adolescents under 18 years of age who had positive stool cultures for *Campylobacter* spp. between June 1, 2008 and May 31, 2016 were eligible for inclusion in our study. Due to its retrospective design, with no harm to the patients anticipated, no informed consent was requested. This study was approved by the institutional ethics committee and was conducted in accordance with the principles of the Declaration of Helsinki, the Good Clinical Practice standards, and Swiss regulatory requirements.

Data collection

All eligible patients with CSE were identified from the database associated with the microbiological laboratory at our institution. The database, named MOLIS, includes all the patients and all information on laboratory tests, from the initial specimen to the final result (preanalytic, analytic and postanalytic). The database did not undergo any changes during the period stated above.

To verify whether the AA presentation is specific to CSE, we compared hospitalised CSE patients with patients hospitalised for gastroenteritis of other aetiologies. To limit selection bias (notably a younger age for patients hospitalised with viral gastroenteritis), we matched each CSE patient with a control patient. For this, we identified all patients hospitalised for gastroenteritis of a different or unknown aetiology by searching our computerised patient records by diagnostic code (ICD 10 diagnostic codes: A04.0, A04.1, A04.2, A04.3, A04.4, A04.6, A04.7, A04.8, A08.0, A08.1, A08.3, A08.4, A08.8, A09.0, A09.9). We then manually selected the individual patients whose age and gender best matched each hospitalised patient with CSE. Relevant information relating to clinical presentation, orientation, work-up, and management were retrospectively retrieved from the medical records. The same methods of clinical information sourcing and the same measurements were used for all the participants. Children with no clinical information were subsequently excluded. All patients who had undergone abdominal radiography (ultrasound or computed tomography) or had received a surgical consultation were identified as AA cases.

Statistical analysis

Demographic information (e.g., gender, origin, age), clinical characteristics (e.g., presenting symptoms, comorbidities), and management (e.g., hospitalisation, hydration, antibiotic treatment) were compared between the groups. Student's T-test or the Kruskal-Wallis test was used for continuous variables and a chi-square test or Fisher's exact test was used for categorical variables. To determine significant predictors of the primary outcome (AA presentation) in the CSE population, we performed a multivariate logistic regression with the potential predictors identified in the univariate analyses (with no additional covariates) and calculated the adjusted odds ratios (ORs) and 95% confidence intervals (CIs). For our matched hospitalised population, as our matching was based on an exposure variable we used a conditional Poisson regression model with grouping on the matched sets to estimate the risk of having an AA presentation with CSE compared to with gastroenteritis of other origin [18]. Risk ratios were calculated and adjustments were then made sequentially for other possible predictors of AA presentation, as identified in the CSE population.

All tests were two-tailed, and a P-value ≤ 0.05 was considered statistically significant. The statistical analyses were computed using Stata software (Stata/IC 11.2 for Mac; StataCorp, Lakeway, TX).

Results

CSE population

Between June 1, 2008 and May 31, 2016, 143 positive cultures for *Campylobacter* spp. were identified in 143 paediatric patients (figure 1). Two patients were excluded due to the absence of medical records. Nineteen patients had undergone abdominal imaging (n = 18) and/or received a surgical consultation (n = 15) and were identified as AA cases. There were 122 non-AA CSE cases. Demographic information was similar between the two groups (table 1). Median age was 8 years (IQR 2–12.8 years); and the population included 60 females (43%) and 81 males (57%). There were 16 patients (14%) with comorbidities. Two patients had gastrointestinal diseases (one case of hypoganglionosis, one of anorectal malformation); two patients suffered from congenital heart disease; six patients were immunosuppressed (three cases of cancer, one case of sickle cell disease, one splenectomy for chronic idiopathic thrombocytopenic purpura, one renal transplantation); four patients had neuropathy (one case of muscular dystrophy, one case of chromosomal anomaly with epilepsy and developmental delay, two developmental delays of unknown origin); one patient had bladder exstrophy; and one patient had interstitial desquamative pneumopathy. Comorbidities were more common among AA than among non-AA cases (4/19, 21% vs 12/99, 12%), but the difference was not statistically significant (table 1).

Clinical presentation of CSE patients

The most frequent complaint was diarrhoea (131/140, 94%), followed by loss of appetite (62/72, 86%), fever (111/132, 84%), abdominal pain (101/132, 77%), and vomiting (59/118, 50%) (table 1). Of these, only the frequency of diarrhoea differed significantly between the two groups (14/19, 74% for cases vs 117/121, 97% for controls; p = 0.02). The vast majority of patients (95 out of 118, 80%) had bloody and/or mucous diarrhoea. General wellbeing

was worse in AA cases (8/18, 44%) than in non-AA cases (8/108, 7%; p <0.001). Although the proportion of patients reporting abdominal pain was similar between the two groups, the character of the pain differed significantly. Nine out of 18 AA cases (50%) compared to 20 out of 115 non-AA cases (17%) had localised pain (p = 0.002); and 2/18 AA cases (11%) compared to 0/111 non-AA cases had abdominal tenderness (p = 0.02)(table 1). There was, however, no difference in rebound pain, which was present in only five patients. Finally, there was no difference in maximum temperature, dehydration state, or the duration of symptoms before consultation (table 1).

In a multivariate logistic regression model including the following three variables associated with an AA presentation, we confirm that localised pain (OR 7.4, 95% CI 2–27.4; p = 0.003), absence of diarrhoea (OR 19.4, 95% CI 3–126.6; p = 0.002), and poor general wellbeing (OR 6.1, 95% CI 1.4–26.3; p = 0.02) were positively correlated with being seen by a surgeon and/or a prescription for radiological examination.

Figure 1: Flowchart of inclusion and group assignment. Abbreviations: CS: Campylobacter species; CSE: CS enteritis; AGE: acute gastroenteritis; AA: acute abdomen * Patients whose age and gender best match the 44 CSE inpatients.

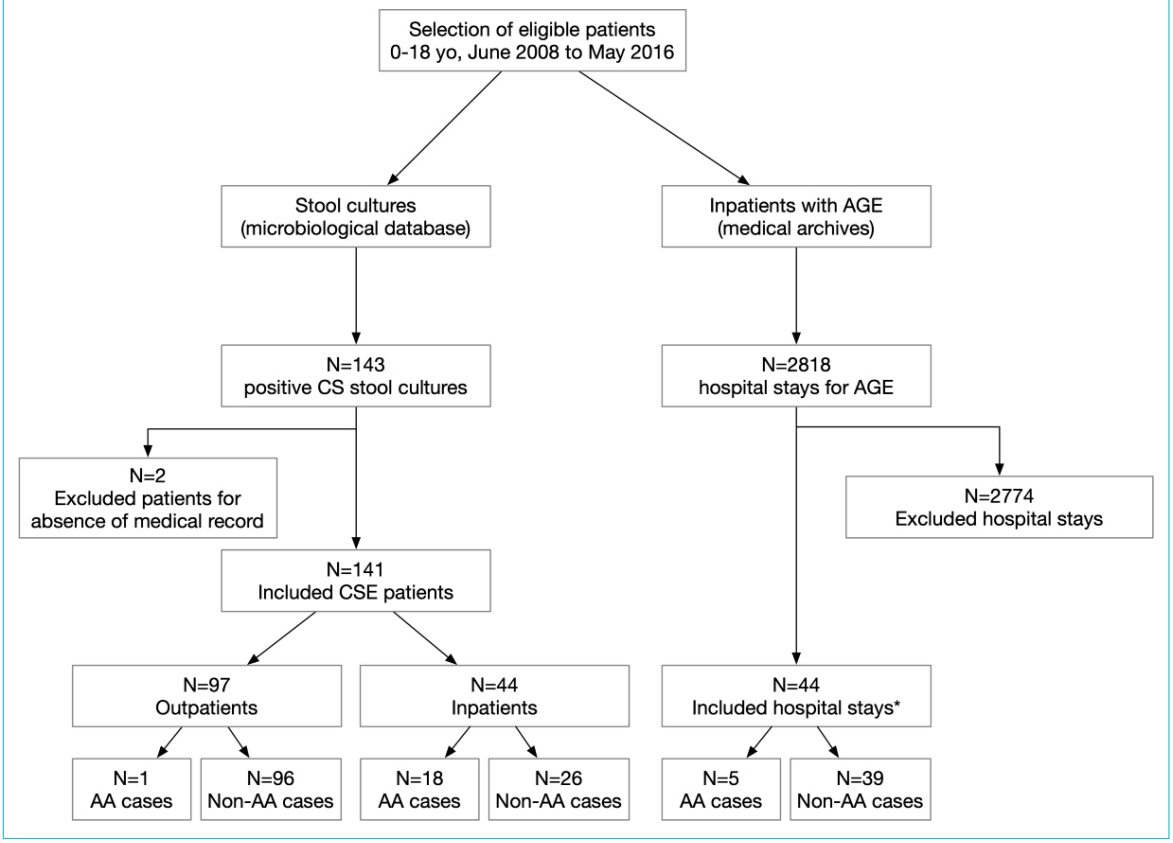

Table 1:
Clinical and demographic characteristics of the study population.

Characteristic	All subjects (n = 141)	Non-AA cases (n = 122)	AA Cases (n = 19)	P-value
Gender, n female/total (%)	60/141 (43)	51/122 (42)	9/19 (47)	0.6
Origin, n Switzerland (%)	80/141 (57)	69/122 (57)	11/8 (58)	0.9
Age, median years (IQR)	8 (2–12.8)	7.5 (3–12.8)	9.4 (2.8–13.6)	0.6
Comorbidities (%)	16/118 (14)	12/99 (12)	4/19 (21)	0.3
Duration of symptoms, median days (IQR)	3 (1–4) (n = 132)	3 (1–5) (n = 115)	3 (1–3) (n = 17)	0.3
Maximum temperature (SD)	39.2 (0.6) (n = 91)	39.2 (0.6) (n = 79)	39.2 (0.6) (n = 12)	0.6
Abdominal pain, n/total (%)	101/132 (77)	87/114 (76)	14/18 (78)	>0.99
Localised pain, n/total (%)	29/133 (22)	20/115 (17)	9/18 (50)	0.002
Tenderness, n/total (%)	2/129 (2)	0/111	2/18 (11)	0.02
Rebound pain, n/total (%)	5/112 (4)	3/94 (3)	2/18 (11)	0.2
Diarrhoea, n/total (%)	131/140 (94)	117/121 (97)	14/19 (74)	0.002
Vomiting, n/total (%)	59/118 (50)	52/104 (50)	7/14 (50)	>0.99
Fever, n/total (%)	111/132 (84)	97/114 (85)	14/18 (78)	0.5
Loss of appetite, n/total (%)	62/72 (86)	53/63 (84)	9/9 (100)	0.3
Diminished general wellbeing, n/total (%)	16/126 (13)	8/108 (7)	8/18 (44)	<0.001
Dehydration, n/total (%)	12/141 (9)	10/122 (8)	2/19 (11)	0.7

IQR: interquartile range; SD: standard deviation

Clinical diagnosis of CSE patients

The primary diagnosis was known for 132 patients. One hundred and three patients had a clinical diagnosis of gastroenteritis. This diagnosis was less common among AA cases (3/19, 16%) in comparison to non-AA cases (100/113, 89%; p <0.001). Nine patients were suspected to have a digestive surgical pathology (four cases of appendicitis, one case of Meckel's disease, four intussusceptions). Three patients were suspected to have a non-surgical digestive pathology (one bacterial translocation, one milk protein allergy, one case of inflammatory bowel disease). Finally, 17 patients received another main diagnosis (three fevers of unknown origin, one febrile seizure, one testicular mass, two cases of febrile neutropenia, one case of otitis media, one case of pneumothorax, two cases of pyelonephritis, one case of rhombencephalitis, one case of haemolytic uremic syndrome, one suspicion of osteomyelitis, one bladder exstrophy, one case of alcohol abuse, and one case of aphthous stomatitis). Of these 17 patients, 8 had a secondary diagnosis of gastroenteritis.

Microbiological results of CSE patients

The majority of *Campylobacter* isolates were *C. jejuni* (123, 87%), followed by *C. coli* (17, 12%) and *C. concisus* (1, 1%). There was only one infection with *C. coli* among the AA cases. All other infections with *C. coli* and *C. concisus* were in the non-AA cases group.

Twenty-three patients had a rapid stool antigen test for adenovirus and rotavirus. Two patients were found to be positive for adenovirus, and one of them also had a positive test for rotavirus. There were eight other patients (three AA cases and five controls) with documentation of other pathogens: one positive urine culture for *E. coli* (AA case), one positive nasopharyngeal PCR for picornavirus (AA case), one case with positive antigen and toxin tests for *Clostridioides difficile* (control), three positive stool cultures for *Aeromonas* spp. (control), one positive stool culture for *Salmonella enterica* serovar Enteritidis (control), and one positive urine antigen test for *Streptococcus pneumoniae* (AA case).

Radiological investigation of CSE patients (AA cases)

Eighteen patients underwent abdominal radiological investigation. Nine received only an ultrasound, two underwent only computed tomography (CT), and seven underwent ultrasonography and CT. In three patients, radiology focused only on the kidneys (two patients with ultrasound only) or on potential metastasis of a testis tumour (one patient who underwent ultrasonography and CT). Four of the remaining 15 patients (27%) had normal results on ultrasonography (no CT performed). Examinations of 11 patients yielded pathological findings: free abdominal fluid (n = 4), adenitis (n = 7), and ileitis and/or colitis (n = 8). Mixed findings were present in six patients. No patient had only free abdominal fluid, while two patients had only adenitis and three patients had only ileitis and/or colitis. There was one patient with a normal ultrasound and a pathological CT scan (colitis). All CT scans were abnormal.

Management of CSE patients

Almost all AA cases were hospitalised (18/19, 95%), compared to only 22% (26/119) of non-AA cases (p <0.001). AA cases were also more likely to be prescribed an antibiotic, with 53% (10/19) of them receiving a prescription for antibiotics compared to only 22% (26/120) of non-AA cases (p = 0.004). Most treated patients were prescribed a macrolide (16/36 patients, 44%). Eight patients (22%) received a prescription for quinolone and 12 (33%) received a prescription for another treatment (beta-lactam, aminoglycoside, metronidazole, cotrimoxazole, or a combination of these). There were no cases that required surgical intervention. Among the hospitalised patients, there were no statistically significant differences in the degree of fluid support and pain management provided. However, the only three patients who required morphine were in the AA cases group.

Comparison of hospitalised CSE patients with age- and gender-matched controls (supplementary table)

Between June 1, 2008 and May 31, 2016, we could identify 2,818 hospital stays for a primary diagnosis of gastroenteritis (Figure 1). Forty-four patients were selected on the basis of having the best age and sex match with the 44 CSE patients who were hospitalised. A microbiological evaluation was carried out in 10/44 patients (23%), and this revealed an aetiology in only four patients (three cases with positive rotavirus stool antigen and one case with positive adenovirus stool antigen).

Median age and gender ratio were similar between the hospitalised CSE patients (8.7 years, IQR 2.4-14.8 and 19/44 females, 43%) and the matched controls (8.9 years, IQR 2.5–13.9 and 19/44 females, 43%). The proportion of patients with co-morbidities was also comparable between the two groups (13/44, 30% for CSE patients vs 15/42, 36% for controls).

Clinical presentation was different between the two groups. The matched controls had less localised abdominal pain than the CSEpatients (5/43, 12% vs 13/43, 30%; p = 0.03). They also presented to the emergency department earlier (median duration of symptoms of 1 day, IQR 0–2 vs 3 days, IQR 1–4; p = 0.002). Finally, the matched controls were more frequently dehydrated (30/42, 71% vs 8/44,18%; p <0.001) and had inflammatory diarrhoea less frequently (3/24,13% vs 19/30, 63%; p = 0.001).

Only five control patients (5/44, 11%) fulfilled our criteria for AA cases, compared to 18 CSE patients (18/44, 41%; p = 0.002). Among those five control patients, two had a surgical consultation and three had a radiological investigation. One patient had abdominal radiography (small intestine dilatation) and the other two had ultrasonography (one had a heterogeneity of the renal cortex with no indirect findings of appendicitis but the appendix was not visualised, and the other had normal findings in the ultrasound without any free liquid in the abdomen). Moreover, only 7% of the control patients (3/44) required pain treatment, compared to 95% (21/22) of the CSE patients (p <0.001).

In a conditional Poisson regression model with grouping on the matched sets, *Campylobacter* spp. infection was positively correlated with being seen by a surgeon and/or with a prescription for a radiological examination (risk ra-

tio 3.6, 95% CI 1.33–9.7; p = 0.01). After adjusting for the absence of diarrhoea or poor general wellbeing, our results did not change (risk ratio 3.2, 95% CI 1.2–8.7; p = 0.02 for both analyses). However, after adjustment for the presence of localised pain, the measured result was lower and no longer statistically significant (risk ratio 2.5, 95% CI 0.9–7.4; p = 0.1).

Discussion

It is well known that abdominal pain in cases of CSE can easily lead to an incorrect clinical evaluation. The pain is typically reported as periumbilical and colicky, with diminished intensity after defecation. This reported sensation is quite different from the lower-right, continual, increasing pain of appendicitis; nonetheless, appendicitis may be incorrectly included in the differential diagnosis [14]. Previous authors have concluded that the severity of the pain is the main explanation for a misleading diagnosis in CSE, but they also cite other factors that may hamper diagnosis: the presence of abdominal pain prior to the onset of diarrhoea, abdominal tenderness most commonly in the lower quadrants, and the absence of diarrhoea [12, 15].

This observational study evaluated the distinctive characteristics of patients with CSE presenting as a potential abdominal surgical emergency. The results presented above show that these patients have distinctive features of abdominal pain that could evoke peritoneal irritation. They were also less likely to have diarrhoea and presented more frequently with a decreased general sense of wellbeing. Consequently, hospitalisations and antibiotic prescriptions were more frequent.

Fortunately, none of our patients required surgical intervention. The results of the radiological investigations excluded appendicitis or any other surgical pathology and revealed other findings, mainly mesenteric adenitis and/or ileocolitis.

Comparison with gastroenteritis of other or unknown aetiologies provides additional information. Although microbiological documentation was scarce in the control group, we can assume that most patients had a viral gastroenteritis. Patients with an aetiology other than *Campylobacter* spp. were more often dehydrated and had less localised abdominal pain. They were mainly hospitalised because they were in need of rehydration. Furthermore, based on the results of our multivariate analyses, we can conclude that it is the *Campylobacter* spp. infection itself, with its localised accompanying pain, that triggers the prescription of a surgical consultation or a radiological examination. The intensity of the pain due to enteritis caused by *Campylobacter* spp., attested by a very extensive use of painkillers, in combination with the localised nature of pain, is certainly a convincing explanation.

Our study has some limitations. First, we used a retrospective design, and our population included only 141 CSE patients. The matched hospitalised population was also of limited size, with only 88 patients in total. Larger future studies are needed to confirm our results and prove the external validity of our findings. Second, some patients did not receive a diagnosis of gastroenteritis, while additional pathogens were identified in others. We cannot exclude the possibility that these patients were colonised by *Campylobacter* spp. only. Third, our control group may not represent certain pathogens, such as those that can generate localised pain, very well. For example, *Yersinia* spp. enteritis is known to mimic acute appendicitis [19]. However, this aetiology is very rare and its absence is therefore unlikely to have biased our results. The specificity of the manifestations observed in our patients infected with *Campylobacter* spp. cannot, therefore, be fully certified. Finally, microbiological documentation was incomplete in our control group. However, we can assume that most of them had a viral gastroenteritis, as observed in case series of hospitalised children [20, 21]. In a prospective survey in Paris, 69.3% of 552 children hospitalised for gastroenteritis had a viral infection (54.5% rotavirus and 12.2% norovirus), while only 3.6% had a single bacterial infection and 1.3% had a viral and bacterial co-infection [21].

In conclusion, we identified a subset of patients with *Campylobacter* spp. gastroenteritis presenting as an acute abdominal emergency. The presentation of these patients was characterised mainly by the nature of the associated abdominal pain. Also, fewer of them had diarrhoea. Knowledge of this particular picture will allow clinicians to evoke this aetiology in the differential diagnosis of localised pain. Ultrasound and/or CT will then be useful tools to identify signs of *Campylobacter* spp. infection, such as mesenteric adenitis and/or enterocolitis, and to differentiate them from appendicitis.

Conflicts of interest
The authors declare no conflicts of interest.

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References

1. Kaakoush NO, Castaño-Rodríguez N, Mitchell HM, Man SM. Global Epidemiology of Campylobacter Infection. Clin Microbiol Rev. 2015 Jul;28(3):687–720. http://dx.doi.org/10.1128/CMR.00006-15. PubMed. 1098-6618
2. Maladies infectieuses à déclaration obligatoire - Nombre de cas hebdomadaires. [Available from: https://www.bag.admin.ch/bag/fr/home/service/zahlen-fakten/zahlen-zu-infektionskrankheiten/meldepflichtige-infektionskrankheiten---woechentliche-fallzahlen.html
3. Jiménez M, Soler P, Venanzi JD, Canté P, Varela C, Martinez Navarro JF. An outbreak of Campylobacter jejuni enteritis in a school of Madrid, Spain. Euro Surveill. 2005 Apr;10(4):9–10. http://dx.doi.org/10.2807/esm.10.04.00533-en. PubMed. 1560-7917
4. Tompkins DS, Hudson MJ, Smith HR, Eglin RP, Wheeler JG, Brett MM, et al. A study of infectious intestinal disease in England: microbiological findings in cases and controls. Commun Dis Public Health. 1999 Jun;2(2):108–13. PubMed. 1462-1843
5. Calva JJ, Ruiz-Palacios GM, Lopez-Vidal AB, Ramos A, Bojalil R. Cohort study of intestinal infection with campylobacter in Mexican children. Lancet. 1988 Mar;1(8584):503–6. http://dx.doi.org/10.1016/S0140-6736(88)91297-4. PubMed. 0140-6736
6. Blaser MJ, Wells JG, Feldman RA, Pollard RA, Allen JR. Campylobacter enteritis in the United States. A multicenter study. Ann Intern Med. 1983 Mar;98(3):360–5. http://dx.doi.org/10.7326/0003-4819-98-3-360. PubMed. 0003-4819
7. Hay DJ, Ganguli LA. Campylobacter enteritis presenting as an acute abdomen. Postgrad Med J. 1980 Mar;56(653):205–6. http://dx.doi.org/10.1136/pgmj.56.653.205. PubMed. 0032-5473
8. Moore MH, Davidson JR. Campylobacter enteritis in the acute surgical ward. N Z Med J. 1984 Apr;97(753):219–20. PubMed. 0028-8446
9. Pai CH, Sorger S, Lackman L, Sinai RE, Marks MI. Campylobacter gastroenteritis in children. J Pediatr. 1979 Apr;94(4):589–91. http://dx.doi.org/10.1016/S0022-3476(79)80016-5. PubMed. 0022-3476
10. Pearson AD, Suckling WG, Ricciardi ID, Knill M, Ware E. Campylobacter-associated diarrhoea in Southampton. BMJ. 1977 Oct;2(6092):955–6. http://dx.doi.org/10.1136/bmj.2.6092.955-b. PubMed. 0007-1447

11. Pitkänen T, Pönkä A, Pettersson T, Kosunen TU. Campylobacter enteritis in 188 hospitalized patients. Arch Intern Med. 1983 Feb;143(2):215–9. http://dx.doi.org/10.1001/archinte.1983.00350020033007. PubMed. 0003-9926
12. Pönkä A, Pitkänen T, Kosunen TU. Campylobacter enteritis mimicking acute abdominal emergency. Acta Chir Scand. 1981;147(8):663–6. PubMed. 0001-5482
13. Tanner EI, Bullin CH. Campylobacter enteritis. BMJ. 1977 Aug;2(6086):579. http://dx.doi.org/10.1136/bmj.2.6086.579-a. PubMed. 0007-1447
14. Lehours P, Aladjidi N, Sarlangue J, Mégraud F. [Campylobacter infections in children]. Arch Pediatr. 2012 Jun;19(6):629–34. http://dx.doi.org/10.1016/j.arcped.2012.03.017. PubMed. 0929-693X
15. Vaidya GN, Sharma A, Khorasani-Zadeh A, John S. Enterocolitis without diarrhoea in an adult patient: a clinical dilemma. BMJ Case Rep. 2014;2014.
16. Puylaert JB, Lalisang RI, van der Werf SD, Doornbos L. Campylobacter ileocolitis mimicking acute appendicitis: differentiation with graded-compression US. Radiology. 1988 Mar;166(3):737–40. http://dx.doi.org/10.1148/radiology.166.3.3277242. PubMed. 0033-8419
17. Lamps LW. Infectious causes of appendicitis [ix-x.]. Infect Dis Clin North Am. 2010 Dec;24(4):995–1018. http://dx.doi.org/10.1016/j.idc.2010.07.012. PubMed. 1557-9824
18. Cummings P, McKnight B. Analysis of Matched Cohort Data. Stata J. 2004;4(3):274–81. http://dx.doi.org/10.1177/1536867X0400400305. 1536-867X
19. Lai CH, Lin JN, Chen YH, Chang LL, Huang WY, Ku HP, et al. The first imported human case of Yersinia pseudotuberculosis serotype O1 septicemia presents with acute appendicitis-like syndrome in Taiwan. J Formos Med Assoc. 2014 Sep;113(9):656–9. http://dx.doi.org/10.1016/j.jfma.2011.06.020. PubMed. 0929-6646
20. Friesema IH, de Boer RF, Duizer E, Kortbeek LM, Notermans DW, Norbruis OF, et al. Etiology of acute gastroenteritis in children requiring hospitalization in the Netherlands. Eur J Clin Microbiol Infect Dis. 2012 Apr;31(4):405–15. http://dx.doi.org/10.1007/s10096-011-1320-0. PubMed. 1435-4373
21. Lorrot M, Bon F, El Hajje MJ, Aho S, Wolfer M, Giraudon H, et al. Epidemiology and clinical features of gastroenteritis in hospitalised children: prospective survey during a 2-year period in a Parisian hospital, France. Eur J Clin Microbiol Infect Dis. 2011 Mar;30(3):361–8. http://dx.doi.org/10.1007/s10096-010-1094-9. PubMed. 1435-4373

Supplementary material

Table S1:
Comparison of hospitalised CSE patients with matched controls.

Characteristic	All subjects (n = 88)	Campylobacter (n = 44)	Matched controls (n = 44)	P-value
Gender, n female/total (%)	38/88 (43)	19/44 (43)	19/44 (43)	1
Age, median years (IQR)	8.8 (2.5-14.2)	8.7 (2.4-14.8)	8.9 (2.5-13.9)	1
Comorbidities (%)	28/86 (33)	13/44 (30)	15/42 (36)	0.5
Duration of symptoms, median days (IQR)	1 (0–4) (n = 83)	3 (1–4) (n = 39)	1 (0–2) (n = 44)	0.002
Maximum temperature (IQR)	39 (38.7–39.6) (n = 52)	39.3 (39–39.7) (n = 27)	39 (38.5–39.5) (n = 25)	0.2
Abdominal pain, n/total (%)	55/85 (65)	27/41 (66)	28/44 (64)	0.8
Localized pain, n/total (%)	18/86 (21)	13/43 (30)	5/43 (12)	0.03
Tenderness, n/total (%)	3/86 (4)	2/42 (5)	1/43(2)	0.6
Rebound pain, n/total (%)	3/79 (4)	3/37 (8)	0/42 (0)	0.1
Diarrhoea, n/total (%)	72/84 (86)	36/40 (90)	36/44 (82)	0.4
Vomiting, n/total (%)	55/77 (71)	15/33 (45)	40/44 (91)	1
Fever, n/total (%)	57/81 (70)	32/39 (82)	25/42 (60)	0.03
Loss of appetite, n/total (%)	31/35 (89)	17/17 (100)	14/18 (78)	0.1
Diminished general wellbeing, n/total (%)	29/82 (35)	13/40 (33)	16/42 (38)	0.6
Dehydration, n/total (%)	38/86 (44)	8/44 (18)	30/42 (71)	<0.001
Type of diarrhoea, n inflammatory/total (%)	22/54 (41)	19/30 (63)	3/24 (13)	0.001
Lack of appetite, n/total (%)	31/35 (89)	17/17 (100)	14/18 (78)	0.1
Gastroenteritis diagnosis, n/total (%)	64/88 (72)	20/44 (45)	44/44 (100)	<0.001
No diarrhoea, n/total (%)	12/88 (14)	4/44 (9)	8/44 (18)	0.35
Acute abdomen, n/total (%)	23/88 (26)	18/44 (41)	5/44 (11)	0.002
Surgical consultation, n/total (%)	16/88 (18)	14/44 (32)	2/42 (5)	0.002
Radiological investigation, n/total (%)	20/88 (23)	17/44 (39)	3/44 (7)	0.001
NGT, n/total (%)	10/88 (11)	4/44 (9)	6/44 (14)	0.7
IV infusion, n/total (%)	46/88 (52)	24/44 (55)	22/44 (50)	0.7
Antibiotics, n/total (%)	24/88 (27)	21/44 (48)	3/44 (7)	<0.001
Pain treatment, n/total (%)	44/66 (67)	21/22 (95)	23/44 (52)	<0.001
Morphine, n/total (%)	4/53 (8)	3/9 (33)	1/44 (2)	0.01

IQR: interquartile rang; IV: intravenous, NGT: nasogastric tube