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Case Report
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A Rare Constellation of Multiple Allergies, Chronic Urticaria, Micronutrient Deficiencies, and Multisystem Manifestations of Colorectal Motility Disorder in a Teenage Girl

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Abstract

Multisystem presentations in adolescents often lead clinicians down fragmented diagnostic pathways. Chronic urticaria, multiple allergies, and micronutrient deficiencies are typically approached through immunological or nutritional frameworks, while gastrointestinal dysmotility is often siloed within gastroenterology. This case highlights the diagnostic complexity and systemic interplay between immune dysregulation, nutritional compromise, and colorectal motility disorder in a teenage girl whose symptoms spanned dermatological, gastrointestinal, autonomic, and neurocognitive domains. The case underscores the importance of holistic, cross-specialty evaluation in adolescents with chronic, unexplained multisystem symptoms. This case highlights a rarely documented pathophysiological axis linking gastrointestinal dysmotility, small intestinal bacterial overgrowth, increased intestinal permeability, mast-cell activation, and systemic allergic manifestations. The patient's multisystem symptoms—including fatigue, dizziness, bloating, and early satiety—were ultimately traced to a self-reinforcing loop involving immune dysregulation and nutritional compromise. Her recovery was achieved through coordinated multidisciplinary care.

Keywords: Multiple Allergies; Chronic Spontaneous Urticaria; Micronutrient Deficiencies; Colorectal Motility Disorder; Pediatric Dysmotility; Delayed Colonic Transit; Anorectal Dysfunction; Mast-Cell–Mediated Disease; Immune–Gut Axis; Adolescent Multisystem Disease; Functional Gastrointestinal Disorder; Nutritional Malabsorption; Iron Deficiency; Vitamin D Deficiency; Zinc Deficiency; Multidisciplinary Management; Complex Chronic Illness in Adolescence; Gastrointestinal Dysmotility; Atopy and Dysmotility Overlap; Rare Pediatric Case

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Introduction

Adolescents presenting with chronic urticaria, multiple allergies, and gastrointestinal symptoms often undergo fragmented evaluations across dermatology, immunology, and gastroenterology. However, emerging evidence suggests that these domains may be interconnected through shared pathophysiological mechanisms involving mast-cell activation, intestinal permeability, and neuromuscular dysfunction [1-6].

Colorectal motility disorders in paediatric populations are frequently under-recognised, yet they can produce profound systemic effects—ranging from nutritional compromise to immune dysregulation [6, 7, 8]. Ileocecal valve dysfunction and delayed colonic transit may predispose to small intestinal bacterial overgrowth (SIBO), ileal inflammation, and increased intestinal permeability ("leaky gut"), amplifying antigen exposure and mast-cell activation [9–10]. This cascade can manifest as chronic urticaria, histamine intolerance, and multisystem symptoms including fatigue, dizziness, and early satiety [2, 3, 11, 12, 13].

Micronutrient deficiencies—particularly iron, vitamin D, and zinc—may further impair epithelial integrity and neuromuscular coordination, perpetuating the cycle of dysmotility and immune activation [5, 9, 11]. In this case, we describe a rare and diagnostically challenging constellation of chronic urticaria, multiple allergies, micronutrient deficiencies, and multisystem manifestations

of colorectal motility disorder in a teenage girl, highlighting the importance of integrated, multidisciplinary assessment.

Case Presentation

A 13-year-old girl presented with chronic spontaneous urticaria, multiple food and environmental allergies, fatigue, abdominal pain, bloating, early satiety, and alternating constipation with overflow diarrhea. She was born at term following an uneventful pregnancy, normal antenatal scans, and an uncomplicated vaginal delivery. She passed urine and meconium promptly after birth.

Intermittent symptoms began at 3 years of age, including constipation, abdominal pain, bloating, recurrent vulvovaginitis, urinary tract infections, histamine intolerance, bronchospasms, urticaria, and multiple allergies. These episodes were initially infrequent but became more frequent and severe after the age of 10 years, particularly following 2.5 years of prolonged homeopathic treatment for chronic urticaria and recurrent urinary tract infections. Her parents, frustrated by conflicting diagnoses and intermittent steroid prescriptions from multiple specialists, sought alternative therapies. During this period, she received several courses of antibiotics, analgesics, antispasmodics, antihistamines, and muscle relaxants, all providing only temporary relief.

At around this time, she developed abdominal pain following the passage of hard, sausage-like stools, lasting several minutes and persisting for 3–4 months. One morning, she experienced acute urethral pain without urinary urgency, frequency, hematuria, or pyuria. Urinalysis, microscopy, and culture were normal. She subsequently developed urethro-vaginal reflux (vaginal voiding), reporting that urine appeared to flow retrograde from the urethra into the vagina after micturition. This was associated with recurrent vulvovaginitis characterised by inflammation, redness, oedema, swelling, and tenderness of the vulva and vagina. Pain was sometimes triggered after leaving the washroom, on standing, or when water touched the vulva during washing. She also experienced intermittent severe perineal pain centred over the perineal body, vulva, and vagina.

Symptom improvement with avoidance of suspected food allergens, and recurrence upon re-exposure, raised the possibility of allergic urethritis. However, immunologists considered isolated allergic reactions confined to the vulva, vagina, and perineum unlikely in the absence of systemic manifestations. Pain episodes typically began during or after micturition and lasted 1–2 hours, with symptom-free intervals in between. Occasionally, pain occurred independently of micturition.

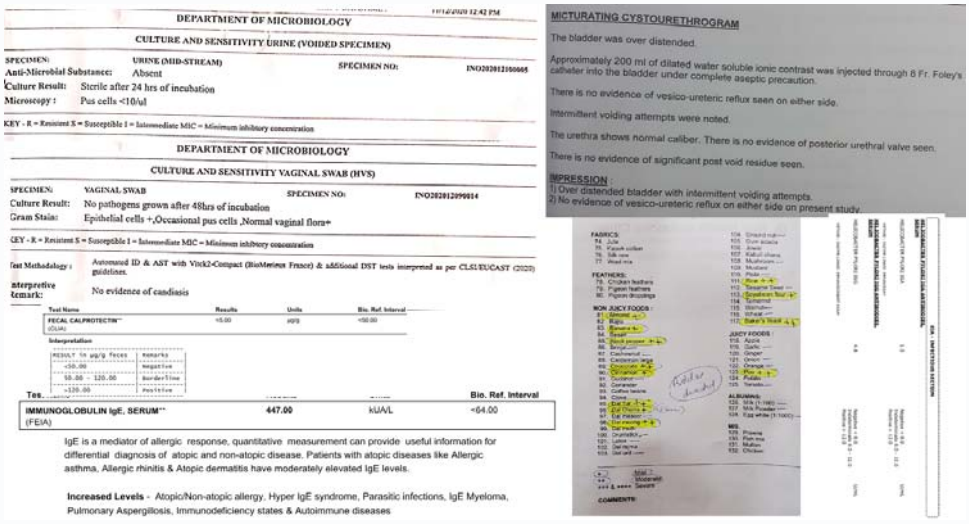
Her symptoms significantly affected school attendance, physical activity, and psychosocial wellbeing. There was no history of eating disorder, intentional dietary restriction, or psychosocial stressors. Family history was notable for atopy and autoimmune thyroid disease.

Despite comprehensive health insurance, the patient had been evaluated by numerous clinicians—including general practice, pediatrics, allergy, dermatology, immunology, rheumatology, respiratory medicine, urology, gynecology, gastroenterology, psychology, naturopathy, ayurveda, and homeopathy—and had undergone extensive investigations. A remote consultation with the paediatric urology team at Great Ormond Street Hospital resulted in a diagnosis of bowel–bladder dysfunction, with no indication for invasive urodynamic studies. Intensive biofeedback therapy, dietary modification, and lifestyle changes were recommended. A separate American consultation suggested possible interstitial cystitis and advised cystoscopy and biopsy.

Laboratory investigations showed hemoglobin of 10.4 g/dL, consistent with anemia of chronic disease. ESR, CRP, biochemical profile, coeliac serology (tTG), HLA-B27 PCR, rheumatoid factor, ANA, PTH, and thyroid function tests were all normal. Immunoglobulin screening was normal except for elevated IgE at 447 IU/mL (normal <64), confirming atopy without evidence of systemic mast-cell disease. She had deficiencies in iron, calcium, magnesium, vitamin D, and zinc, along with hair thinning and impaired concentration. Urinalysis and culture, stool calprotectin and culture, and vaginal smear and culture were all normal (Figure 1).

Extensive serum allergy testing demonstrated sensitisation to multiple inhalant, contact, food, and drug allergens (Figure 2), with summary and quantitative reports (Figures 3 and 4).

Ultrasound of the abdomen and pelvis was normal except for rectosigmoid and colonic stool and gas loading (Figure 5). Abdominal and pelvic radiographs confirmed rectosigmoid and colonic fecal and gas loading (Figure 6). Micturating cystourethrogram (MCUG)


DEPARTMENT OF MICROBIOLOGY

CULTURE AND SENSITIVITY URINE (VOIDED SPECIMEN)

SPECIMEN: URINE (MID-STREAM) SPECIMEN NO: INO20201290005

Anti-Microbial Substance: Absent

Culture Result: Sterile after 24 hrs of incubation

Microscopy : Pus cells <10/ul

KEY - R = Resistant S = Susceptible I = Intermediate MIC = Minimum inhibitory concentration

DEPARTMENT OF MICROBIOLOGY

CULTURE AND SENSITIVITY VAGINAL SWAB (HVS)

SPECIMEN: VAGINAL SWAB SPECIMEN NO: INO20201290014

Culture Result: No pathogens grown after 48hrs of incubation

Gram Stain: Epithelial cells +,Occasional pus cells ,Normal vaginal flora+

KEY - R = Resistant S = Susceptible I = Intermediate MIC = Minimum inhibitory concentration

Test Methodology : Automated ID & AST with Vitek2-Compact (BioMerieux France) & additional DST tests interpreted as per CLSI/EUCAST (2020) guidelines.

Interpretive Remark: No evidence of candiasis

Test Name	Results	Units	Bio. Ref. Interval
FECAL CALPROTECTIN (CLIA)	<5.00	µg/g	<50.00

Interpretation

RESULT in µg/g feces	remarks
<50.00	negative
50.00 - 120.00	borderline
>120.00	positive

Tes.			Bio. Ref. Interval
IMMUNOGLOBULIN IgE, SERUM** (FEIA)	447.00	kUA/L	<64.00

IgE is a mediator of allergic response, quantitative measurement can provide useful information for differential diagnosis of atopic and non-atopic disease. Patients with atopic diseases like Allergic asthma, Allergic rhinitis & Atopic dermatitis have moderately elevated IgE levels.

Increased Levels - Atopic/Non-atopic allergy, Hyper IgE syndrome, Parasitic infections, IgE Myeloma, Pulmonary Aspergillosis, Immunodeficiency states & Autoimmune diseases

MICTURATING CYSTOURETHROGRAM

The bladder was over distended.

Approximately 200 ml of diluted water soluble ionic contrast was injected through 8 Fr. Foley's catheter into the bladder under complete aseptic precaution.

There is no evidence of vesico-ureteric reflux seen on either side.

Intermittent voiding attempts were noted.

The urethra shows normal caliber. There is no evidence of posterior urethral valve seen.

There is no evidence of significant post void residue seen.

IMPRESSION :

1) Over distended bladder with intermittent voiding attempts.

2) No evidence of vesico-ureteric reflux on either side on present study.

Figure 1: Lab reports-urine, vaginal swab, MCUG, IgE, allergy test results.

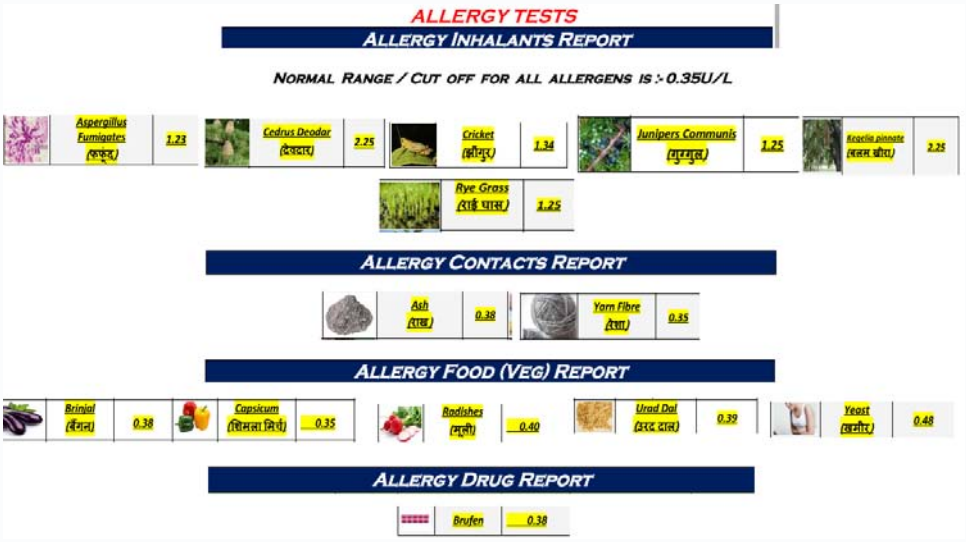


Figure 2: Multiallergen tests.

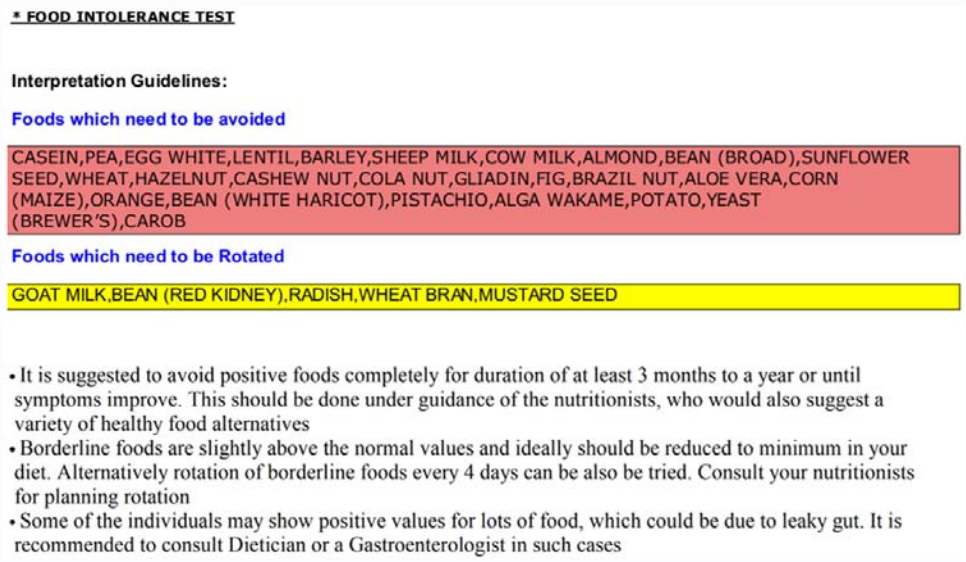


Figure 3: Food intolerance test results.

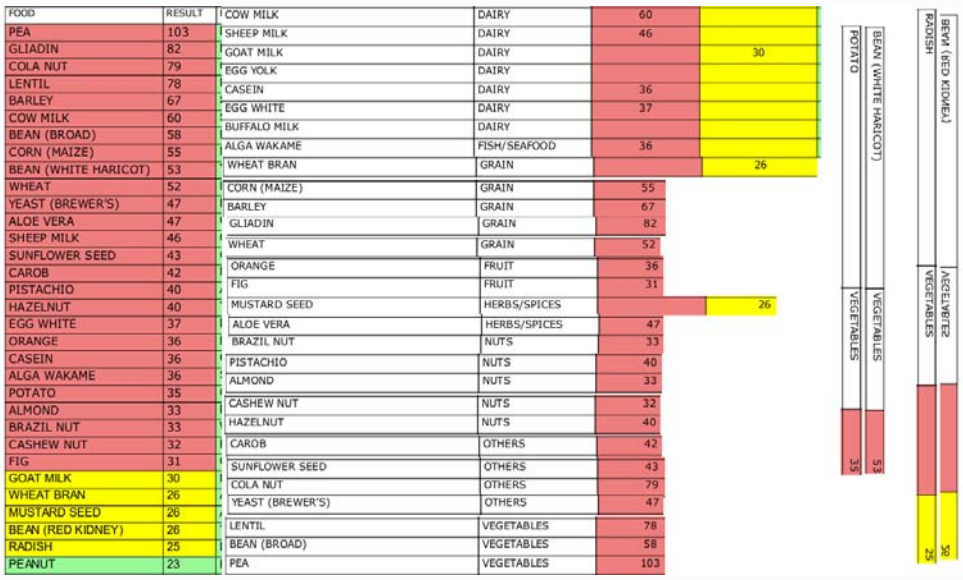
was normal (Figure 7). Cystoscopy and biopsies were normal. Colonoscopy with superficial mucosal biopsies showed non-specific inflammation secondary to fecal retention. Upper gastrointestinal endoscopy and biopsies were normal, and Helicobacter pylori testing was negative. MRI of the abdomen and pelvis showed bilateral sacroiliac joint inflammation, which resolved with treatment, and confirmed rectosigmoid and colonic loading.

Differential diagnoses included mast-cell activation syndrome, coeliac disease, inflammatory bowel disease, eating-disorder–related malnutrition, connective-tissue disorder with dysmotility, primary colorectal motility disorder, and chronic idiopathic constipation with secondary nutritional compromise.

The parents discovered our published work on colorectal secretomotility disorders and allergic urethritis and contacted us seeking diagnostic clarification and management guidance. On examination, the patient (height 5 ft 5 in, weight 41 kg) had palpable fecalomas in the left lower quadrant, with rectosigmoid and left colonic fecal loading. Colonic transit studies and anorectal manometry demonstrated markedly delayed colonic transit and anorectal dyssynergia, with impaired rectoanal coordination and reduced propulsive force. Contrast enema showed a transition zone at the rectosigmoid junction and severe ileocecal reflux, despite previously normal endoscopic and radiological findings, consistent with colorectal motility disorder (Figure 8). Dietetic assessment confirmed adequate caloric intake but poor micronutrient absorption. Dermatology review confirmed chronic spontaneous urticaria. Immunology review supported mast-cell–mediated hypersensitivity without systemic mastocytosis.

Management and Outcome

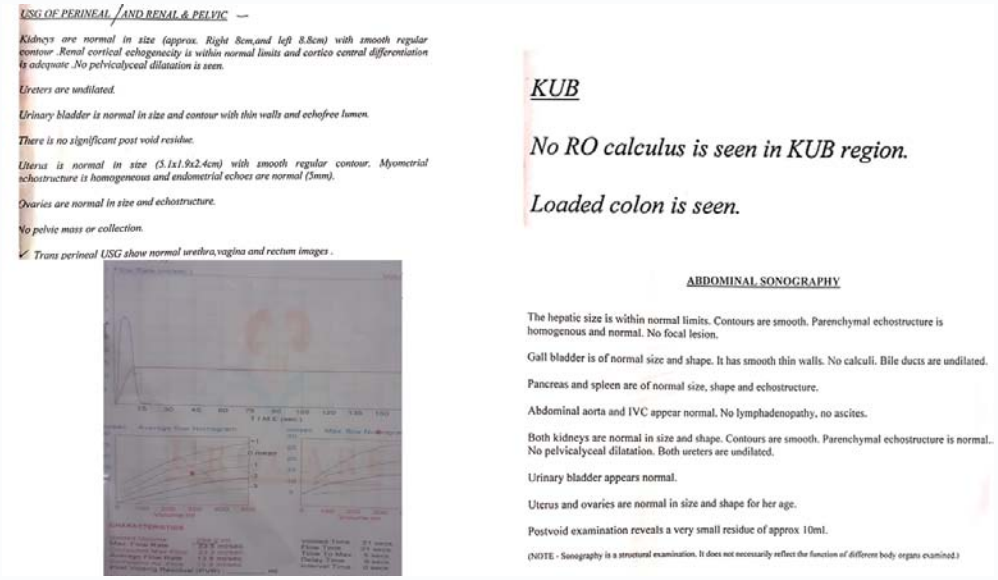
Management options included either continued conservative therapy or diagnostic and therapeutic transanal modified extended anorectal myomectomy with endosurgical procedures. After detailed counselling, the patient and her parents opted for conservative



FOOD	RESULT
PEA	103
GLIADIN	82
COLA NUT	79
LENTIL	78
BARLEY	67
COW MILK	60
BEAN (BROAD)	58
CORN (MAIZE)	55
BEAN (WHITE HARICOT)	53
WHEAT	52
YEAST (BREWER'S)	47
ALOE VERA	47
SHEEP MILK	46
SUNFLOWER SEED	43
CAROB	42
PISTACHIO	40
HAZELNUT	40
EGG WHITE	37
ORANGE	36
CASEIN	36
ALGA WAKAME	36
POTATO	35
ALMOND	33
BRAZIL NUT	33
CASHEW NUT	32
FIG	31
GOAT MILK	30
WHEAT BRAN	26
MUSTARD SEED	26
BEAN (RED KIDNEY)	26
RADISH	25
PEANUT	23

COW MILK	DAIRY	60
SHEEP MILK	DAIRY	46
GOAT MILK	DAIRY	30
EGG YOLK	DAIRY	
CASEIN	DAIRY	36
EGG WHITE	DAIRY	37
BUFFALO MILK	DAIRY	
ALGA WAKAME	FISH/SEAFOOD	36
WHEAT BRAN	GRAIN	26
CORN (MAIZE)	GRAIN	55
BARLEY	GRAIN	67
GLIADIN	GRAIN	82
WHEAT	GRAIN	52
ORANGE	FRUIT	36
FIG	FRUIT	31
MUSTARD SEED	HERBS/SPICES	26
ALOE VERA	HERBS/SPICES	47
BRAZIL NUT	NUTS	33
PISTACHIO	NUTS	40
ALMOND	NUTS	33
CASHEW NUT	NUTS	32
HAZELNUT	NUTS	40
CAROB	OTHERS	42
SUNFLOWER SEED	OTHERS	43
COLA NUT	OTHERS	79
YEAST (BREWER'S)	OTHERS	47
LENTIL	VEGETABLES	78
BEAN (BROAD)	VEGETABLES	58
PEA	VEGETABLES	103

Figure 4: Allergy quantitative tests.



USG OF PERINEAL AND RENAL & PELVIC

Kidneys are normal in size (approx. Right 8cm,and left 8.8cm) with smooth regular contour .Renal cortical echogenecity is within normal limits and cortico central differentiation is adequate .No pelvicalyceal dilatation is seen.

Ureters are undilated.

Urinary bladder is normal in size and contour with thin walls and echofree lumen.

There is no significant post void residue.

Uterus is normal in size (5.1x1.9x2.4cm) with smooth regular contour. Myometrial echostructure is homogeneous and endometrial echoes are normal (5mm).

Ovaries are normal in size and echostructure.

No pelvic mass or collection.

Trans perineal USG show normal urethra,vagina and rectum images .

KUB

No RO calculus is seen in KUB region.

Loaded colon is seen.

ABDOMINAL SONOGRAPHY

The hepatic size is within normal limits. Contours are smooth. Parenchymal echostructure is homogenous and normal. No focal lesion.

Gall bladder is of normal size and shape. It has smooth thin walls. No calculi. Bile ducts are undilated.

Pancreas and spleen are of normal size, shape and echostructure.

Abdominal aorta and IVC appear normal. No lymphadenopathy, no ascites.

Both kidneys are normal in size and shape. Contours are smooth. Parenchymal echostructure is normal.. No pelvicalyceal dilatation. Both ureters are undilated.

Urinary bladder appears normal.

Uterus and ovaries are normal in size and shape for her age.

Postvoid examination reveals a very small residue of approx 10ml.

(NOTE - Sonography is a structural examination. It does not necessarily reflect the function of different body organs examined.)

Figure 5: Imaging reports.

management initially, with the understanding that surgical intervention could be pursued at a later stage if required.

A comprehensive, multidisciplinary plan was implemented. This included dietary and lifestyle modification, intensive biofeedback therapy, pelvic floor physiotherapy, and a structured bowel regimen tailored to address delayed colonic transit and anorectal dyssynergia. Antihistamines were gradually tapered and discontinued over the following weeks. Micronutrient deficiencies were corrected through targeted supplementation of iron, vitamin D, calcium, magnesium, and zinc. Holobiotic therapy (pre-, pro-, and post-biotics) was introduced to support gut microbial balance and intestinal barrier function.

Dietetic review confirmed adequate caloric intake but poor micronutrient absorption, guiding ongoing nutritional support. Dermatology review reaffirmed the diagnosis of chronic spontaneous urticaria, while immunology assessment supported mast-cell–mediated hypersensitivity without evidence of systemic mastocytosis. Regular follow-up ensured coordinated care across gastroenterology, immunology, dermatology, dietetics, and physiotherapy.

Over a 48-month period, the patient experienced gradual and sustained improvement in gastrointestinal function, allergic symptoms, nutritional status, and overall quality of life. All symptoms—including abdominal pain, bloating, early satiety, constipation, overflow diarrhea, vulvovaginal discomfort, and urticaria—resolved completely. Her school attendance normalised, physical activity improved, and cognitive symptoms such as impaired concentration resolved. She successfully completed her secondary school examinations (GCSE) and is now preparing for her A-Level examinations.

With long-term stability achieved, the patient and her family

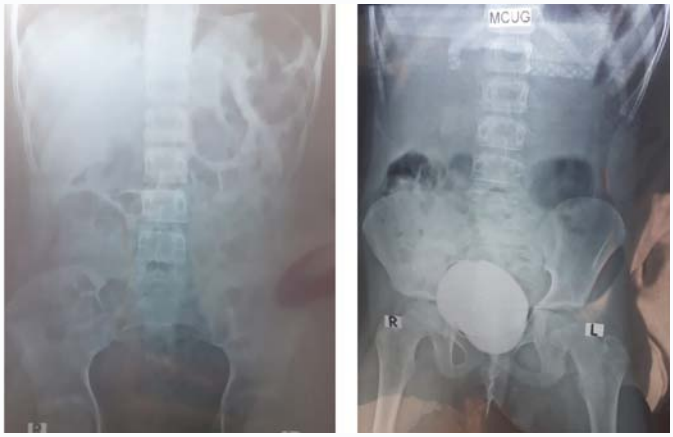


Figure 6: Abdominal radiograph with left colon fecal loading, excessive gas and colonic distention and a normal MCUG.

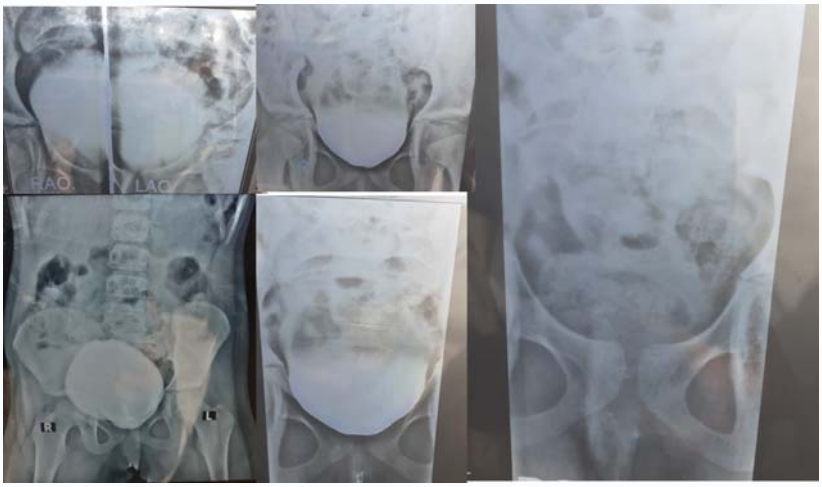


Figure 7: Normal MCUG with physiological urethra-vaginal reflux/vaginal voiding and Pelvic radiograph showing rectosigmoid loading.

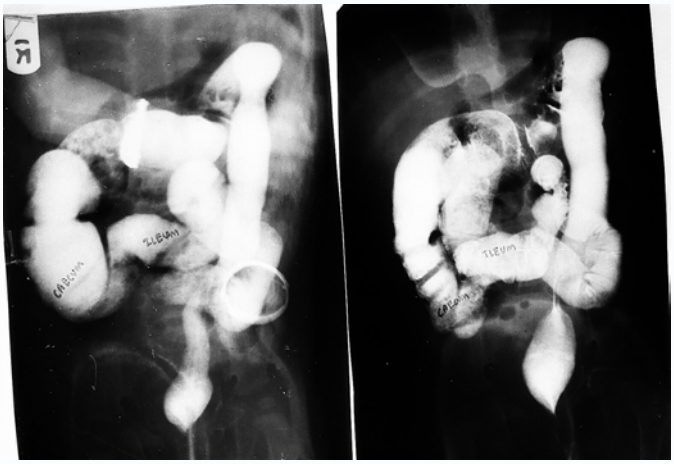


Figure 8: Contrast enema – Note transition zone at rectosigmoid junction with tapering, massive colonic distension and redundancy with gross ileocecal reflux into the terminal ileum.

have expressed interest in undergoing modified extended transanal anorectal myomectomy and endosurgical procedures prior to her transition to university, aiming to ensure durable bowel function and minimise the risk of symptom recurrence while living away from home.

Discussion

This case illustrates a rare but clinically important convergence of gastrointestinal dysmotility, immune dysregulation, micronutrient deficiency, and multisystem symptomatology in an adolescent.

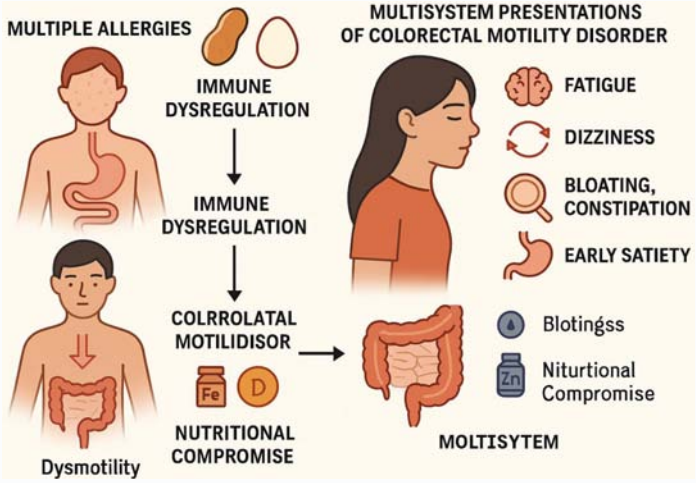


Figure 9: Graphical Abstract: Multisystem Interplay in a Teenage Girl with Colorectal Motility Disorder, Allergies, and Micronutrient Deficiencies.
A conceptual schematic illustrating the interconnected pathophysiology of chronic urticaria, multiple allergies, micronutrient deficiencies, and colorectal motility disorder. Arrows depict bidirectional relationships between immune dysregulation, nutritional compromise, and gastrointestinal dysmotility. Multisystem symptoms—including fatigue, dizziness, bloating, and early satiety—are shown as downstream effects of this interplay.

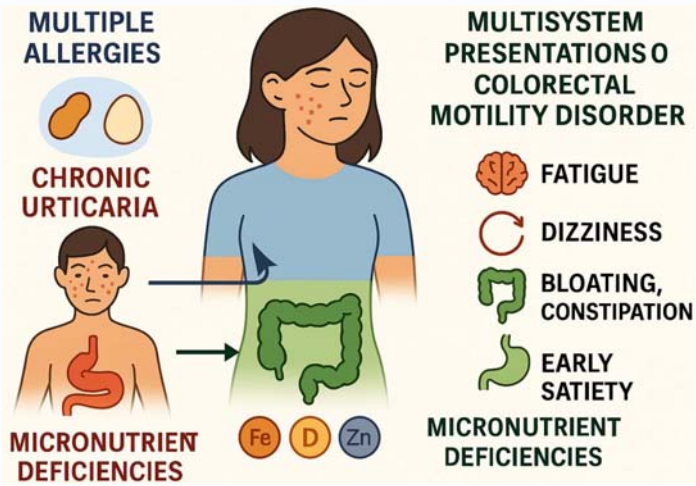


Figure 10: Color-Coded Organ-System Map: Multisystem Manifestations in a Teenage Girl.
Infographic showing organ-system involvement in a teenage girl with chronic urticaria, multiple allergies, micronutrient deficiencies, and colorectal motility disorder. Each system is color-coded: red for immune/skin (urticaria), blue for allergies, orange for gastrointestinal tract (dysmotility), and green for micronutrient compromise. Icons highlight systemic symptoms including fatigue, dizziness, bloating, and early satiety.

Although each component—chronic urticaria, multiple allergies, micronutrient deficiencies, and constipation—can occur independently, their simultaneous presence suggests a unifying pathophysiological axis rather than isolated disorders [2–5, 6].

Several Mechanisms may Explain this Constellation

Colorectal Dysmotility as the Upstream Driver

Delayed colonic transit and anorectal dyssynergia can significantly alter intraluminal pressure dynamics and impair the competence of the ileocecal valve [6–8, 11]. In adolescents, this dysfunction may be subtle and easily overlooked, yet it can create conditions that favor retrograde flow of colonic contents into the terminal ileum. This "ileocecal reflux" phenomenon is well recognised in motility disorders and provides a plausible mechanical substrate for downstream complications [7, 8].

Mechanistic anchor: Colorectal motility disorder → altered transit + hypotensive ileocecal valve → ileocecal reflux + stasis.

Ileocecal Reflux, SIBO, and Ileal Inflammation

Retrograde movement of colonic bacteria into the small intestine, combined with prolonged small bowel transit, predisposes to small intestinal bacterial overgrowth (SIBO) [8–10]. SIBO is increasingly recognised in paediatric dysmotility and can produce abdominal pain, bloating, early satiety, and nutritional compromise [2, 6, 13]. Ileocecal valve dysfunction (hypotensive valve, impaired barrier) is associated with prolonged small bowel transit and higher risk of SIBO [7, 8].

In this patient, the clinical picture was consistent with SIBO-related dysbiosis and terminal ileal irritation, analogous to "backwash" phenomena described in inflammatory bowel disease, though here driven by motility rather than mucosal pathology [14].

Barrier Dysfunction and the "Leaky Gut" State

SIBO and ileal inflammation can disrupt epithelial tight junctions,

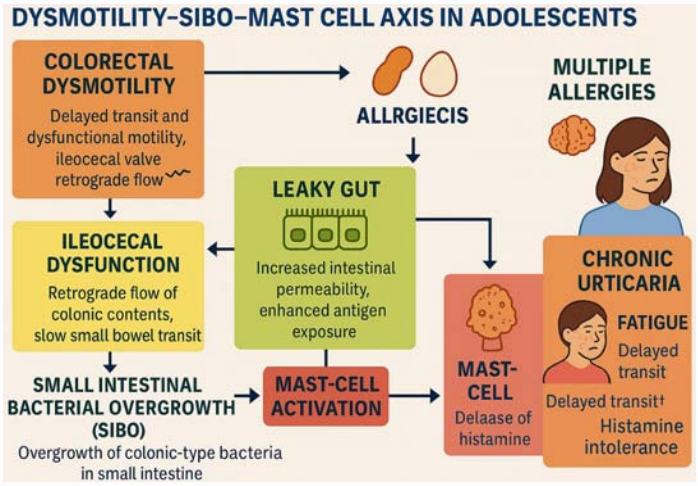


Figure 11: Dysmotility–SIBO–Mast Cell Axis in Adolescents: A Systems-Based Teaching Schematic.
A color-coded infographic illustrating the stepwise pathophysiological progression from colorectal dysmotility to multisystem manifestations in adolescents. Orange section: delayed colonic transit and ileocecal valve dysfunction lead to retrograde flow. Yellow section: ileocecal reflux and slow small bowel transit promote small intestinal bacterial overgrowth (SIBO). Green section: SIBO and ileal inflammation contribute to increased intestinal permeability ("leaky gut"), enhancing antigen exposure. Red section: mast-cell activation triggers histamine release, leading to chronic urticaria, multiple allergies, and histamine intolerance. Green icons highlight systemic symptoms including fatigue, dizziness, bloating, and early satiety. Arrows depict feedback loops and inter-system amplification.

increasing intestinal permeability [9, 10]. This "leaky gut" state enhances exposure of the mucosal immune system to dietary antigens, bacterial metabolites, and inflammatory mediators. In genetically or immunologically predisposed individuals, this can amplify systemic immune activation and contribute to mast-cell hyper-responsiveness [2, 3, 12, 13, 15].

Mast-Cell Activation, Histamine Intolerance, and Allergic Phenotypes

Mast cells play a central role in both gastrointestinal and cutaneous immune responses [3, 12, 13, 16]. Increased antigenic load from a permeable gut can trigger mast-cell degranulation, releasing histamine and other mediators that manifest as chronic urticaria, flushing, pruritus, and food-related reactions [2, 3, 12]. Mast cells influence enteric nerve signaling and may contribute to delayed transit and visceral hypersensitivity [13].

In this patient, the coexistence of chronic urticaria, multiple allergies, and gastrointestinal symptoms suggests a mast-cell activation phenotype, potentially exacerbated by impaired histamine degradation and increased luminal histamine from dysbiosis [2, 12, 13].

Micronutrient Deficiencies as Both Cause and Consequence

Iron, vitamin D, and zinc deficiencies are common in adolescents but take on added significance in the context of dysmotility and immune activation [5, 11].

- Iron and zinc are essential for epithelial repair and neuromuscular coordination [11].
- Vitamin D modulates immune tolerance and mast-cell stability [5].

Deficiencies may therefore perpetuate both dysmotility and immune dysregulation, creating a self-reinforcing cycle [5, 9, 11]. Micronutrient deficiencies may impair neuromuscular coordination of the colon, while dysmotility reduces nutrient absorption, creating a self-perpetuating cycle [6, 11, 17].

A Multisystem Clinical Phenotype

The patient's fatigue, dizziness, cognitive slowing, and gastrointestinal symptoms reflect the systemic consequences of this interconnected axis [2–5, 9, 12]. Adolescents are particularly vulnerable to multisystem manifestations due to rapid growth, hormonal changes, and heightened nutritional demands [4, 5].

Importance of a Multidisciplinary Approach

This case underscores the limitations of single-specialty evaluation. Gastrointestinal dysmotility, allergic disease, and micronutrient deficiency are often managed in isolation, yet in complex cases they may represent different expressions of a shared underlying mechanism [2–5, 6–9, 18]. Early involvement of gastroenterology, immunology, dermatology, dietetics, and physiotherapy was essential in achieving clinical improvement.

Clinical Implications

This case highlights the need for clinicians to consider:

- Dysmotility as a potential driver of immune and nutritional abnormalities [6–8, 11].
- SIBO and ileocecal dysfunction in adolescents with unexplained multisystem symptoms [8–10].
- Mast-cell activation as a downstream effect of gut barrier dysfunction [2, 3, 9, 12, 13].
- The value of integrated care pathways in complex paediatric presentations [2–5, 6–9, 19].

Immune–Gut Axis Dysregulation

Chronic allergic inflammation may alter gut permeability and neuromuscular function, contributing to dysmotility [2–5, 9, 12,13].

Adolescent Vulnerability

Rapid growth, hormonal changes, and psychosocial stressors may amplify multisystem presentations [4, 5, 14, 20].

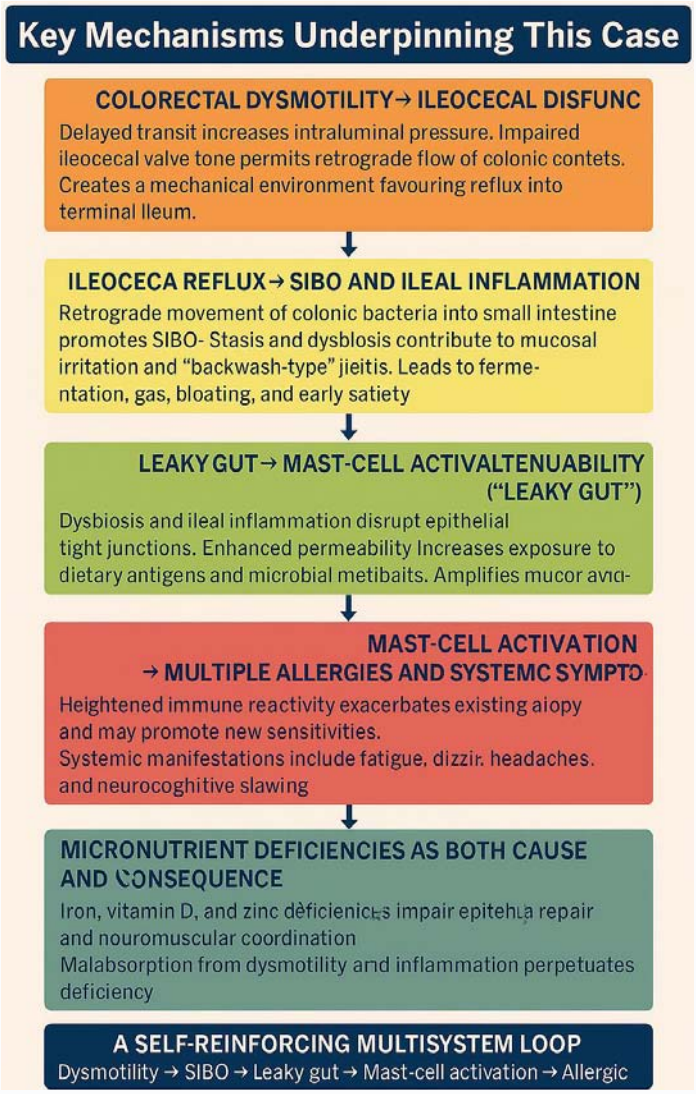


Figure 12: Key Mechanisms: Dysmotility–SIBO–Mast Cell Axis in Adolescents.
A color-coded flowchart illustrating the interconnected pathophysiological cascade in a teenage girl with multisystem symptoms. Orange box: colorectal dysmotility and ileocecal valve dysfunction lead to retrograde flow. Yellow box: ileocecal reflux promotes small intestinal bacterial overgrowth (SIBO) and ileal inflammation. Green box: SIBO and mucosal irritation increase intestinal permeability ("leaky gut"), enhancing antigen exposure. Red boxes: mast-cell activation and histamine intolerance drive chronic urticaria, multiple allergies, and systemic symptoms. Teal box: micronutrient deficiencies (iron, vitamin D, zinc) impair epithelial repair and neuromuscular coordination. Blue box: the cycle reinforces itself through a feedback loop linking dysmotility, immune activation, and nutritional compromise.

This case emphasises the need for holistic, multidisciplinary assessment when adolescents present with chronic multisystem symptoms that do not fit neatly into a single specialty. In our patient, we hypothesise that colorectal motility disorder led to altered ileocecal dynamics, favoring ileocecal reflux and small intestinal bacterial overgrowth [6–8]. SIBO and terminal ileal inflammation likely contributed to increased intestinal permeability [9, 10], amplifying exposure to luminal antigens and bacterial products. This "leaky gut" state may have intensified pre-existing atopy, driving mast-cell activation, chronic urticaria, and a histamine-intolerance phenotype [2, 3, 12, 13], while dysmotility and inflammation impaired micronutrient absorption [5, 11]. The result was a self-reinforcing loop linking dysmotility, SIBO, leaky gut, immune dysregulation, and multisystem symptoms.

Learning Points/Take-Home Messages

- **Colorectal motility disorders in adolescents can present with multisystem symptoms,** including allergic, dermatological, urological, nutritional, and neurocognitive manifestations, leading to diagnostic delay if evaluated in isolation.
- **Ileocecal valve dysfunction and delayed colonic transit may predispose to ileocecal reflux, SIBO, and ileal inflammation,** creating a cascade of gastrointestinal and immune dysregulation.
- **Increased intestinal permeability ("leaky gut") can amplify mast-cell activation**, contributing to chronic urticaria, histamine intolerance, and multiple food and environmental allergies.
- **Micronutrient deficiencies (iron, vitamin D, zinc, magnesium, calcium) may be both a consequence of dysmotility and a driver of impaired epithelial repair**,

immune imbalance, and neuromuscular dysfunction.

- **Recurrent vulvovaginitis, urethral pain, and urethro-vaginal reflux** can be secondary to severe constipation and pelvic floor dysfunction, and may mimic primary urological or gynecological pathology.
- **A multidisciplinary approach** is essential, integrating gastroenterology, immunology, dermatology, dietetics, physiotherapy, and psychology to address the interconnected mechanisms driving symptoms.
- **Conservative management**—including bowel retraining, biofeedback, micronutrient repletion, holobiotics, and lifestyle modification—can lead to complete symptom resolution, even in complex multisystem presentations.
- **Early recognition of the dysmotility**–SIBO–mast-cell axis may prevent years of fragmented care, unnecessary investigations, and inappropriate treatments.

Conclusion

This case demonstrates an unusual and diagnostically challenging convergence of multiple allergies, chronic spontaneous urticaria, micronutrient deficiencies, and significant colorectal motility disorder in an adolescent. The patient's multisystem symptoms initially appeared unrelated, yet ultimately reflected a shared pathophysiological interplay between immune dysregulation, impaired gastrointestinal motility, and secondary nutritional compromise. Her journey underscores the limitations of single-specialty assessment in complex paediatric presentations and highlights the value of early, coordinated, multidisciplinary care. Recognising these interconnected mechanisms can prevent delayed diagnosis, reduce unnecessary investigations, and improve long-term outcomes for young patients with similarly complex, overlapping conditions. This case demonstrates an unusual convergence of multiple allergies, chronic urticaria, micronutrient deficiencies, and colorectal motility disorder in a teenage girl. The multisystem nature of her presentation underscores the importance of early, integrated, multidisciplinary evaluation. Recognising the interconnected mechanisms between immune dysregulation, nutritional compromise, and gastrointestinal motility can prevent diagnostic delay and improve outcomes. This case highlights the complex interplay between immune dysregulation, micronutrient deficiency, and colorectal motility disorder in adolescence. Early recognition of multisystem patterns and coordinated multidisciplinary care are essential to avoid fragmented management and improve outcomes.

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