



JSMCR-25-44

# Purulent Peritonitis Masked by Disease-Modifying Therapy: A Case Report

**Brock P Shipp<sup>1\*</sup>, Stephanie Gonzalez<sup>1</sup>, Lindsay Chapman<sup>1</sup> and Ricardo Mohammad<sup>2</sup>**<sup>1</sup>Department of Osteopathic Medicine, Alabama College of Osteopathic Medicine, Dothan, USA<sup>2</sup>Department of Osteopathic Medicine, Santa Rosa Medical Center, Florida, USA**\*Corresponding author:** Brock P Shipp, Department of Osteopathic Medicine, Alabama College of Osteopathic Medicine, Dothan, USA, E-mail: [shippb@acom.edu](mailto:shippb@acom.edu)**Received date:** August 22, 2025; **Accepted Date:** August 25, 2025; **Published date:** September 22, 2025**Citation:** Shipp BP, Gonzalez S, Chapman L, Mohammad R (2025) Purulent Peritonitis Masked by Disease-Modifying Therapy: A Case Report. *J Surg Med Case Rep* Vol.2 No.4: 044.

## Abstract

We report the case of a 70-year-old woman with relapsing-remitting multiple sclerosis on dimethyl fumarate therapy who presented with acute abdominal pain. Despite severe infection, her initial laboratory tests were misleading due to treatment-related lymphocytopenia. Imaging and surgical exploration revealed purulent peritonitis with a pelvic abscess. This case emphasizes the importance of early imaging and prompt intervention in immunocompromised patients with atypical abdominal presentations.

**Keyword:** Multiple sclerosis; Purulent peritonitis; Disease-modifying therapy; Dimethyl fumarate; Lymphocytopenia

## Introduction

Inflammation of the peritoneum, otherwise known as peritonitis, can be divided into three subgroups: primary, secondary and tertiary peritonitis [1]. Primary peritonitis refers to an acute inflammation within the peritoneal cavity, marked by infection of ascitic fluid in the absence of a clear intra-abdominal cause [2]. Secondary peritonitis results from inflammation or visceral perforation; this can be classified as acute peritonitis by perforation, postoperative peritonitis or post-traumatic peritonitis<sup>3</sup>. Tertiary peritonitis is chronic or recurrent peritonitis that persists after treatment of primary or secondary peritonitis [3]. Secondary peritonitis is observed in 1% of hospital admissions and is the second leading cause of sepsis among intensive care unit patients worldwide [1]. There should be a high degree of clinical suspicion for bacterial peritonitis in patients presenting with abdominal pain or tenderness, fever or altered mental status [4]. Early identification of peritonitis is critical due to its ability to cause sepsis and multiorgan failure [5].

Multiple Sclerosis (MS) is an autoimmune disorder characterized by chronic inflammation of the Central Nervous System (CNS). It is driven by autoreactive lymphocytes that penetrate the blood-brain barrier, leading to inflammation within the CNS. The disease is marked by chronic demyelinating lesions and an immune response directed against myelin with marked effects to the brain, spinal cord and optic nerves [6]. The American Gastroenterological Association (AGA) highlights that immunocompromised patients can

present with milder signs and symptoms, necessitating a low threshold for imaging to diagnose and rule out complications [7].

Common MS treatments, particularly Disease-Modifying Therapies (DMTs), can influence immune responses and potentially mask infections, even when laboratory findings appear normal [8]. Among these drugs is Dimethyl Fumarate (DMF), used for Relapsing Remitting Multiple Sclerosis (RR-MS). Although its mechanism of action is not fully understood, studies show a dose-dependent effect of DMF on T-cell counts which occurs through the induction of apoptosis and decreased proliferation of CD4<sup>+</sup> and CD8<sup>+</sup> T-cells [9]. When looking at the intestinal barrier and Gut-Associated Lymphoid Tissue (GALT) we must consider the role of lymphocytes and the effect of lymphocytopenia. When bacteria breach the gastrointestinal epithelial layer, the body triggers an adaptive immune response by activating CD4<sup>+</sup> T-cells and inducing cytokine release; therefore, lymphocytopenia could impair the intestinal barrier and facilitate the transit of bacteria from the intestines into the peritoneal cavity [10].

Here, we report the case of an atypical presentation of purulent peritonitis and abdominal abscess in a 70-year-old female with RR-MS undergoing therapy with DMF.

## Key Clinical Message

Dimethyl fumarate-induced lymphocytopenia may



obscure typical laboratory signs of severe intra-abdominal infection. Multiple sclerosis patients on disease-modifying therapy can present atypically despite severe pathology. Maintain a low threshold for early imaging and surgical evaluation when acute abdominal symptoms arise to prevent delayed diagnosis and sepsis.

## Case Presentation

A 70-year-old caucasian female with a past medical history of RR-MS and hypertension presented to the emergency department for an acute onset of abdominal pain that began one day prior to admission. The patient reported a sudden popping sensation at the onset of her pain, which progressively intensified over the following 24 hours, accompanied by chills and nausea. On examination, the patient was alert without altered

mental status and in no acute distress. She was febrile, with a temperature of 101.2°F, normotensive with a blood pressure of 113/73 mmHg, tachycardic with a heart rate of 102 beats per minute, a decreased respiratory rate of 8 breaths per minute and hypoxic with an oxygen saturation of 89% on room air. Evaluation of the abdomen revealed a soft and nondistended abdomen with rebound tenderness, no guarding and normal bowel sounds. Initial laboratory studies on admission demonstrated a White Blood Cell (WBC) count of 7.8 cells/μL (reference range: 4.5-11.0 cells/μL) with a neutrophil predominance of 91.6% (reference range: 40-60%) and a lymphocytopenia of 4.8% (reference range: 20-40%).

Over the next few days, the patient developed leukocytosis with a peak WBC count of 17.6 cells/μL on day 6 (**Table 1**).

**Table 1:** Complete blood count trends over hospital course.

Complete Blood Count								
Test	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Units	Reference Range
WBC	7.8	9	11.7	12.5	11.6	17.6	cells/μL	4.5-11.0
RBC	5.52	4.69	3.94	4	3.86	4.21	mm <sup>3</sup>	4.2-5.4
Hemoglobin	16.7	14.2	12.1	11.9	11.8	12.8	g/dL	12.0-15.5
Hematocrit	50.2	42.1	36.1	36.5	34.9	42.1	%	36-46
MCV	91	90	92	91	90	90	fL	80-100
MCH	30.2	30.2	30.6	29.8	30.5	30.3	pg/mL	27-33
MCHC	33.2	33.7	33.4	32.7	33.7	33.7	g/dL	32-36
Platelet Count	261	226	170	190	203	243	mm <sup>3</sup>	150-450
Neutrophils %	91.6	89.4	95.1	-	-	-	%	40-60
Lymphocytes %	4.8	5.8	2.7	-	-	-	%	20-40
Monocytes %	2.7	4.3	2.1	-	-	-	%	2-8
Eosinophils %	0.2	0	0	-	-	-	%	1-4
Basophils %	0.7	0.5	0.1	-	-	-	%	0.5-1

**\*Note:** μL: microliters; g: grams; dL: deciliters; fL: femtoliters; pg: picogram; mm: millimeters.

Initial serum chemistry and lactic acid level were within normal limits. However, progressive hypoalbuminemia and electrolyte fluctuations were

observed over the hospital course indicating systemic illness and metabolic stress (**Table 2**).

**Table 2:** Comprehensive metabolic panel trends over hospital course.

Comprehensive Metabolic Panel								
Test	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Units	Reference Range
Sodium	141	139	142	142	142	138	mEq/L	135-145
Potassium	3.2	3.5	3.8	3.7	3.4	3.3	mEq/L	3.5-5.0
Chloride	101	105	108	107	106	100	mEq/L	98-107
CO <sub>2</sub>	29	27.8	25.8	28.4	28.1	21.4	mEq/L	22-29
BUN	23	22	28	27	20	10	mg/dL	7-20
Creatinine	0.88	0.84	0.79	0.63	0.62	0.6	mg/dL	0.7-1.3
eGFR	71	-	-	-	-	-		



Glucose	150	127	133	78	72	72	mg/dL	70-100
Calcium	9.3	8.1	7.7	8.7	7.8	7.8	mg/dL	8.6-10.2
Albumin	3.8	-	2.1	2.1	2.1	2	g/dL	3.5-5.0
Alkaline Phosphatase	107	-	59	69	76	80	IU/L	30-120
ALT	14	-	17	20	20	20	IU/L	0-35
AST	16	-	23	23	26	21	IU/L	0-35
Bilirubin total	0.7	-	0.9	0.8	0.8	1	mg/dL	0.1-1.2
Protein total	7.6	-	4.7	6.1	5.2	4.9	g/dL	6.0-8.3
Lipase	27	-	9	-	-	-	mEq/L	135-145
Lactic Acid	3.8	3.5	3.8	3.7	3.4	3.3	mEq/L	3.5-5.0

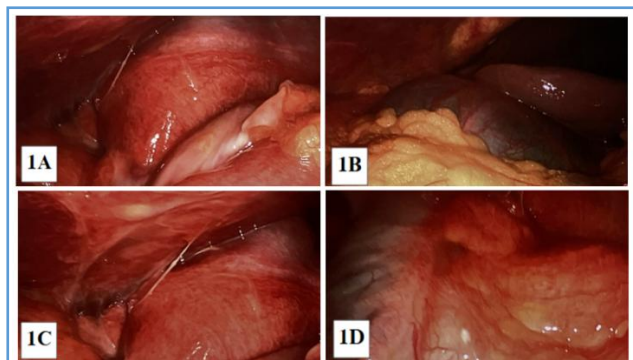
\***Note:** mEq: milliequivalents; L: liters; dL: deciliter; IU: international unit; mg: milligrams; g: grams.

Computed Tomography (CT) imaging was performed to assess the patient further and revealed a small pneumoperitoneum without a definitive identification of the perforation site. The patient's pain was well-controlled with hydromorphone 0.5 mg, however, over the next 24 hours she experienced clinical deterioration prompting surgical consultation. Upon surgical evaluation it was determined that the patient's worsening abdominal pain and peritoneal signs, including guarding, indicated the patient had developed an acute abdomen requiring emergent surgical intervention.

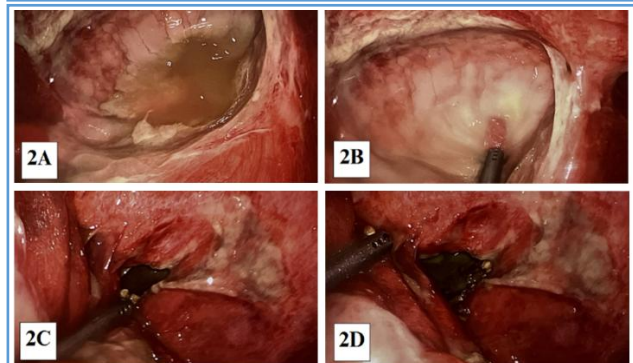
### Differential diagnosis

- Gastrointestinal perforation
- Gangrenous cholecystitis
- Spontaneous bacterial peritonitis
- Tubo ovarian abscess
- Phlegmonous appendicitis
- Treatment and Outcome

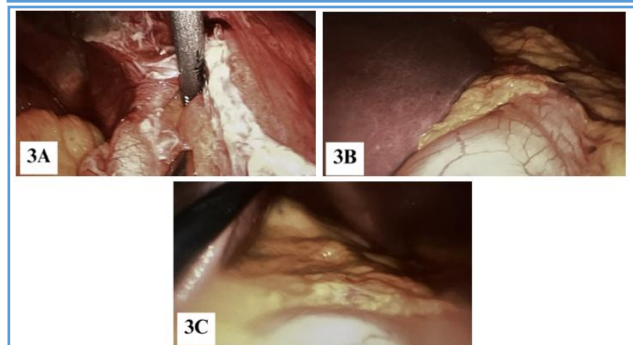
On day 2 of hospitalization the patient underwent a diagnostic laparoscopy for further evaluation. The gallbladder, although distended, did not demonstrate any evidence of necrosis or perforation. Perihepatic fluid was present as well as exudate throughout the mid abdomen. No obvious perforation or inflammation was seen in the stomach, duodenum or sigmoid colon and no fluid collection or necrosis was noted. Phlegmonous changes were observed along the appendix and small intestine without obvious purulent pockets. Significant inflammation, exudate and inflammatory reaction were observed throughout the paracolic gutter on the right with extension to the pelvis anterior to the uterus (**Figures 1, 2, 3**). A pocket demonstrating gangrenous changes to the anterior abdominal wall was identified, consistent with an abscess cavity (**Figure 4**).



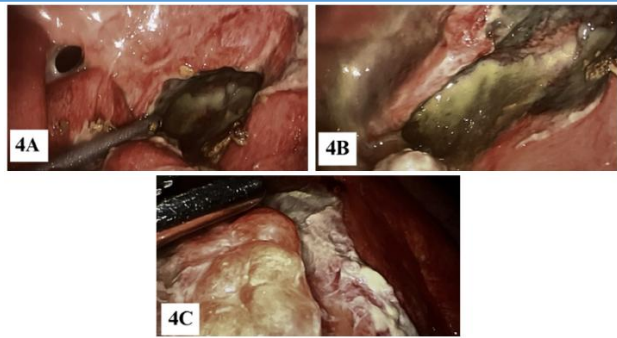
**Figure 1:** Laparoscopic images reveal widespread erythema on the peritoneal surface.



**Figure 2:** Laparoscopic images detail multiple extensive purulent inflammatory lesions with yellow-white nodules on the peritoneal surface.



**Figure 3:** Extensive inflammation, fluid pooling, and yellow-white exudate found on peritoneal laparoscopy.



**Figure 4:** A pocket demonstrating gangrenous changes to the anterior abdominal wall was identified, consistent with an abscess cavity.

A large pelvic abscess was encountered and entered, revealing stool and debris, which were suctioned. Culture and sensitivity samples were obtained from the purulent collection in the right paracolic gutter. Following thorough inspection of the small intestine, sigmoid colon, stomach, gallbladder and right colon, the peritoneal cavity was irrigated with normal saline and suctioned. Two 19 French JP drains were placed: One in the pelvic abscess cavity and the other in the hepatorenal fossa, extending toward the gallbladder. The exact etiology remains uncertain; however, it is suspected that a perforation may have occurred and subsequently sealed. Common sites of perforation did not demonstrate active inflammation or visible perforations.

Following surgical intervention on day 2, continuous infusion of lactated ringers was administered and the patient was treated by ID with Piperacillin/Tazobactam 3.375g, 100 mL per hour piggyback Q6 hrs. On day 6 vancomycin 1250 mg 166.67 mg per hr IV piggyback Q12 hrs was added due to increasing WBC count and elevated temperature the night prior. Patient was continued on DMF therapy once she was able to tolerate oral intake and Lovenox 40 mg was given for venous thromboembolism prophylaxis. Upon symptomatic improvement and lab normalization, the patient was subsequently discharged to outpatient skilled rehabilitation.

## Result and Discussion

Purulent peritonitis is a severe and potentially life-threatening intra-abdominal infection, commonly resulting from gastrointestinal perforation or the development of an abscess. It often involves polymicrobial flora, such as *Escherichia coli* and *Streptococcus anginosus* [5]. The incidence of purulent peritonitis in patients with RR-MS on DMT like DMF is a rare occurrence and diagnostically complex due to immunosuppression. This case involving a 70-year-old female with RR-MS illustrates an unusual presentation of purulent peritonitis characterized by acute

abdominal pain, fever and rebound tenderness without guarding, alongside an initially normal WBC count but notable lymphocytopenia. Due to the mechanism of action of DMF, a patient may present with lymphocytopenia, which can reduce the body's ability to mount an effective immune response to infections. This reduction in lymphocyte counts can lead to serious infections, including opportunistic infections, which can sometimes occur without a corresponding increase in standard laboratory infection markers. DMF reduces CD4<sup>+</sup> and CD8<sup>+</sup> T-cell counts *via* apoptosis, impairing gut-associated lymphoid tissue and facilitating bacterial translocation from the gut lumen into the peritoneal cavity precipitating infection [9,10]. The lymphocytopenia present in this case masked early inflammatory markers, necessitating reliance on clinical signs and imaging. CT revealing a pneumoperitoneum together with signs of an acute abdomen prompted emergent exploratory diagnostic laparoscopy on day 2. Surgical intervention identified a pelvic abscess and gangrenous abdominal wall changes, reinforcing the value of early imaging and thorough physical exams in immunocompromised patients.

Differential diagnoses for this case included: SBP, diverticulitis with perforation and appendicitis. SBP was excluded due to the lack of cirrhosis or ascites. Clinical findings were more consistent with a perforated viscus, indicating that the peritonitis was likely secondary in origin. Prompt recognition and surgical management, alongside appropriate antimicrobial therapy, are essential to prevent the development of sepsis, as demonstrated in this case. ID initiated treatment with Piperacillin/Tazobactam and later added vancomycin. Corticosteroids were withheld, but the patient remained on DMF once she was able to tolerate oral intake.

Further research is needed on DMF's role in gastrointestinal infection risk and optimal management in MS patients.

## Conclusion

This case highlights an atypical presentation of purulent peritonitis in a 70-year-old female with RR-MS on DMT, marked by an initially normal WBC count with delayed leukocytosis and DMF-induced lymphocytopenia. The presence of clinical signs such as rebound tenderness and fever, in the absence of significant laboratory abnormalities, highlights the diagnostic challenges encountered in immunocompromised patients. CT imaging and prompt surgical intervention were crucial in identifying a pelvic abscess, gangrenous abdominal wall changes and purulent peritonitis likely secondary to a sealed perforation. Clinicians should maintain a low threshold



for advanced imaging and diagnostic laparoscopy in MS patients on DMTs who present with acute abdominal symptoms, as delayed diagnosis increases the risk of sepsis and multiorgan failure. This case also highlights the need for further evaluation of DMF and its potential role in the development of severe infections, particularly those involving the gastrointestinal tract. Future research should aim to elucidate the infection risks associated with DMT and inform evidence-based prophylactic strategies for immunocompromised populations.

## Acknowledgement

### Informed consent

Written informed consent was obtained from the patient for publication and the case has been reviewed and approved by the Professionalism and Ethics Committee at the Alabama College of Osteopathic Medicine.

### Author contributions

- Brock P Shipp: Conceptualization, data collection, manuscript drafting and critical revision.
- Stephanie Gonzalez: Data collection, literature review and manuscript drafting.
- Lindsay Chapman: Literature review, figure preparation and manuscript editing.
- Ricardo Mohammed: Surgical case management, data verification and critical revision of the manuscript.

All authors read and approved the final version of the manuscript.

## References

1. Ross JT, Matthay MA, Harris HW (2018) Secondary peritonitis: Principles of diagnosis and intervention. *BMJ* 361: k1407. [Crossref], [Google Scholar] [Indexed]
2. Barrés-Fernández A, Piolatti-Luna A, Bretón-Martínez JR, et al. (2021) Case report: Primary peritonitis as the onset of pediatric Ménétrier's disease. *Front Pediatr* 8: 589853. [Crossref], [Google Scholar] [Indexed]
3. Ordoñez CA, Puyana JC (2006) Management of peritonitis in the critically ill patient. *Surg Clin North Am* 86: 1323-1349. [Crossref], [Google Scholar] [Indexed]
4. MacIntosh T (2018) Emergency management of spontaneous bacterial peritonitis - a clinical review. *Cureus* 10: e2253. [Crossref], [Google Scholar] [Indexed]
5. Brown D, Vashisht R, Caballero AJA (2025) Septic peritonitis. *StatPearls Publishing*. [Google Scholar] [Indexed]
6. Yolanda IR and Ritarwan K (2022) Case report multiple sclerosis. *Open Access Maced J Med Sci* 10: 142-145. [Crossref], [Google Scholar] [Indexed]
7. Peery AF, Shaukat A, Strate LL (2021) AGA clinical practice update on medical management of colonic diverticulitis: Expert review. *Gastroenterology* 16: 906-911. [Crossref], [Google Scholar] [Indexed]
8. Mehta D, Miller C, Arnold DL, Bame E, Bar-Or A, et al. (2019) Effect of dimethyl fumarate on lymphocytes in RRMS: Implications for clinical practice. *Neurology* 92: e1724-e1738. [Crossref], [Google Scholar] [Indexed]
9. Mills EA, Ogrodnik MA, Plave A, Mao-Draayer Y (2018) Emerging understanding of the mechanism of action for dimethyl fumarate in the treatment of multiple sclerosis. *Front Neurol* 9: 5. [Crossref], [Google Scholar] [Indexed]
10. He Y, Huang X, Zhang J, et al. Decreased peripheral blood lymphocyte count predicts poor treatment response in peritoneal dialysis-associated peritonitis. *J Inflamm Res* 16: 5327-5338. [Crossref], [Google Scholar] [Indexed]