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A Diagnostic Odyssey: Non-Hodgkin Lymphoma Masquerading as Recurrent Typhoid Fever with Concurrent Nephrolithiasis.

Abstract

This case documents the clinical journey of a 38-year-old non-smoking, non-alcoholic male who underwent 14 months of repeated misdiagnosis before being correctly identified as a case of Stage IIIB [1] Diffuse Large B-Cell Lymphoma (DLBCL), the most aggressive subtype of Non-Hodgkin Lymphoma. Over this period, the patient was treated three times for typhoid fever — twice on serology alone and once on confirmed blood culture — and was simultaneously managed for bilateral nephrolithiasis and recurrent urinary tract infections.

The case carries a unique complication: a 6.8 cm para-aortic lymph node mass was causing extrinsic compression of the right ureter, producing hydronephrosis that was attributed entirely to kidney stones. Lithotripsy had been scheduled before a contrast-enhanced MRI revealed the true obstructive lesion. A further unusual dimension is that the lymphoma-induced immunosuppression was itself responsible for recurrent Salmonella infections — the disease was, in effect, manufacturing its own disguise.

The patient is currently on active treatment: induction chemotherapy with R-CHOP-15 every 15 days for 6 months, followed by maintenance Rituximab once a month for 4 months. This report covers the diagnostic journey, the confirmed diagnosis, the treatment protocol, cycle-by-cycle clinical progress, adverse effects, and the surveillance plan.

Patient Profile

Parameter	Details
Age and Sex	38 years, Male

Occupation

Senior Software Engineer — sedentary desk role, high occupational stress

Marital Status

Married, two children aged 9 and 6

Smoking

Non-smoker (lifelong)

Alcohol

Non-alcoholic (lifelong abstainer)

Diet

Predominantly vegetarian; no red meat

Family History

No haematological malignancy; father — hypertension; mother — type 2 diabetes

Body Weight at First Presentation

72 kg (BMI 25.3 kg/m²; height 169 cm)

Body Weight at Diagnosis

54 kg (BMI 18.9 kg/m²) — 18 kg total loss over 14 months

Body Weight at Chemotherapy Month 4

61 kg — partial recovery on nutritional rehabilitation

Comorbidities

None documented prior to this illness

Presenting Symptoms and Clinical History

The patient first sought medical attention 14 months before the confirmed diagnosis. His complaints at initial presentation were non-specific and, individually, appeared consistent with common conditions prevalent in the region. Over the subsequent months, each new development was interpreted within the framework of the existing diagnosis rather than prompting a reassessment.

The following table traces the clinical journey month by month.

Period	Symptoms and Events	Working Diagnosis at the Time
Months 1 to 2	Low-grade fever between 99 and 100.6°F, fatigue, appetite loss, mild right flank discomfort	Viral fever; possible early urinary tract infection
Month 3	Fever rising to 103°F; Widal test O antigen 1:160; treated with ciprofloxacin for 14 days	Typhoid Fever — Episode One
Months 4 to 5	Temporary symptomatic relief but persistent fatigue; 5 kg weight loss; intermittent night sweats begin	Post-typhoid asthenia; nutritional deficiency
Month 6	Fever returns; Widal O antigen 1:320; blood culture isolates Salmonella typhi; right flank pain worsens; ultrasound shows bilateral renal calculi (right 9 mm, left 5 mm) and mild right hydronephrosis	Recurrent Typhoid and Nephrolithiasis — Episode Two
Months 7 to 8	Treated with IV ceftriaxone for typhoid and tamsulosin for stone passage; fever never fully clears; 9 kg total weight loss; urine culture grows E. coli; haematuria noted	Complicated urinary tract infection with nephrolithiasis and recurrent typhoid
Months 9 to 10	Third febrile episode; Widal positive again; referred to urology; urologist attributes worsening hydronephrosis to 9 mm calculus; lithotripsy formally scheduled	Obstructive nephropathy from stone disease; lithotripsy planned
Months 11 to 12		

Patient notices bilateral neck swellings; drenching night sweats; 15 kg total weight loss; breathlessness on exertion; small right pleural effusion on chest X-ray; lithotripsy deferred for reassessment

Lymphoma queried for the first time; urgent haematology referral made

Month 13

LDH 1,040 IU/L; CRP 138 mg/L; ESR 94 mm/hr; beta-2 microglobulin 5.2 mg/L; CT abdomen reveals para-aortic mass 6.8 cm compressing right ureter; mediastinal widening seen

Aggressive lymphoma strongly suspected

Month 14

Contrast MRI confirms bulky retroperitoneal and mediastinal lymphadenopathy; PET-CT shows FDG-avid nodes with SUV max 19.4; excisional biopsy of left cervical node confirms DLBCL Non-GCB; treatment initiated

Confirmed Diagnosis: Stage IIIB DLBCL Non-Hodgkin Lymphoma

Physical Examination at Oncology Referral

The following findings were recorded at the time of haematology and oncology referral in Month 13.

System

Findings

General

Cachectic and chronically ill appearance; BMI 19.1; pallor; mild icterus

Vitals

Temperature 99.6°F; heart rate 108 bpm; BP 106/68 mmHg; SpO2 96% on room air; respiratory rate 18/min

Lymph Nodes

Bilateral cervical chains: multiple firm, discrete, non-tender nodes up to 3.1 cm on left and

2.4 cm on right; bilateral axillary and inguinal lymphadenopathy also present

Abdomen

Splenomegaly with spleen palpable 4 cm below costal margin; no hepatomegaly; right lumbar tenderness on deep palpation; no ascites

Chest

Decreased breath sounds and dullness to percussion at right base — consistent with small pleural effusion

Cardiovascular

Tachycardia; no murmurs; no raised jugular venous pressure

Musculoskeletal

Generalised muscle wasting; no bone tenderness on palpation

Skin

No purpura, petechiae, or rash; no oral candidiasis

Investigations

Tests Performed Before Lymphoma Was Suspected

During the first 12 months, all investigations were directed toward confirming and treating the established diagnoses of typhoid, kidney stones, and urinary tract infection. The results below illustrate how each finding, while genuine, served to deflect attention from the underlying malignancy.

Investigation

Result

How It Contributed to Misdiagnosis

Widal Test — three occasions

O antigen rising from 1:160 to 1:320 across episodes

Each positive result reinforced the typhoid diagnosis; rising titres were attributed to re-infection rather than lymphoma-driven immunosuppression

Blood Culture (Month 6)

Salmonella typhi isolated from peripheral blood

Genuine bacteraemia confirmed; no clinician considered why a healthy adult had recurrent intracellular infections

Urine Culture (Month 9)

E. coli more than 100,000 CFU/mL

Genuine urinary tract infection; attributed to urinary stasis from stone obstruction rather than lymph node compression

Ultrasound Abdomen (Month 6)

Bilateral renal calculi; right hydronephrosis

Stones provided a visible structural explanation for flank pain and obstructive features; retroperitoneal nodes not visualised on ultrasound

Complete Blood Count (Month 9)

Haemoglobin 10.4 g/dL; WBC 7,100; Platelets 340,000; mild eosinophilia

Anaemia attributed to chronic infection; no cytopenias to prompt haematological review

Erythrocyte Sedimentation Rate (Month 9)

82 mm/hr

Attributed to ongoing sepsis

Kidney Function Test (Month 8)

Creatinine 1.4 mg/dL

Attributed to dehydration and obstructive stone disease

Chest X-Ray (Month 10)

Small right pleural effusion

Reported as reactive effusion secondary to infection; mediastinal widening not flagged at this stage

Investigations After Lymphoma Was Suspected

Once haematology was involved, a structured workup was initiated. The results below led

to the confirmed diagnosis.

Investigation	Result	Clinical Significance
LDH	1,040 IU/L — markedly elevated	Key indicator of aggressive lymphoma with high cell turnover
Beta-2 Microglobulin	5.2 mg/L — elevated	Adverse prognostic marker in DLBCL; correlates with tumour burden
Uric Acid	9.2 mg/dL	Elevated tumour cell turnover; pre-treatment tumour lysis risk identified
CRP and ESR	CRP 138 mg/L; ESR 94 mm/hr	Marked systemic inflammatory burden
CECT Abdomen and Pelvis	Para-aortic mass 6.8 cm encasing right ureter; bilateral hydronephrosis; mesenteric lymphadenopathy	Critical finding: obstruction confirmed as arising from lymph node mass, not from stone alone
Contrast-Enhanced MRI	Bulky retroperitoneal and pelvic lymphadenopathy; splenic involvement; right ureteric displacement by nodal mass	Definitive anatomical mapping; confirmed extrinsic compression of ureter by lymphoma
PET-CT Whole Body	FDG-avid nodes in bilateral cervical, mediastinal, para-aortic, and pelvic regions; SUV max	

19.4; no marrow uptake

Stage IIIB confirmed; highly aggressive metabolic activity

Excisional Biopsy — Left Cervical Node

1 Diffuse Large B-Cell Lymphoma, Non-GCB subtype; Ki-67 proliferation index 88%

Final histological diagnosis; highly proliferative aggressive tumour

Immunohistochemistry Panel

CD20 positive, CD79a positive, BCL2 positive, BCL6 positive, MUM1 positive, CD10 negative, MYC positive at 35%

Activated B-Cell or Non-GCB subtype confirmed — known to carry inferior prognosis

FISH Analysis

BCL2 rearrangement positive; MYC rearrangement negative

Single-hit lymphoma — not double-hit; modifies prognosis and treatment considerations

Bone Marrow Biopsy

No lymphoma infiltration

Stage III rather than Stage IV confirmed

Echocardiogram Pre-Chemotherapy

LVEF 64%; structurally normal

Baseline cardiac function documented before doxorubicin administration

The Unique Complication

Lymphoma-Driven Ureteric Obstruction Mistaken for Stone Disease

From Month 6 onward, progressive right-sided hydronephrosis and a rising creatinine were attributed entirely to an obstructing 9 mm right renal calculus. This attribution was supported by an ultrasound, a urology opinion, and the patient's own colicky flank pain. Extracorporeal shock wave lithotripsy was formally scheduled.

The CECT obtained at Month 13 overturned this assumption completely. A 6.8 cm para-aortic lymph node mass was identified encasing and displacing the right ureter at the level of L3 to L4. The renal calculus was real but was an incidental finding — it was not the

primary cause of obstruction. The lymphoma had been compressing the kidney silently for months while every clinician's attention was directed at the stone.

Had lithotripsy proceeded without this imaging, the ureteric obstruction would have persisted unchanged. Renal function would have continued to deteriorate. The lymphoma would have remained undetected for further months, with the realistic possibility of progression to Stage IV disease with bone marrow involvement.

Lymphoma-Facilitated Recurrent Typhoid Fever

The three episodes of Salmonella typhi infection in this patient were not independent events caused by repeated environmental exposure or vaccine failure. DLBCL produces significant impairment of T-cell and B-cell mediated immunity. This functional immunosuppression reduces the host's capacity to clear intracellular pathogens — a category that includes Salmonella typhi.

In effect, the lymphoma was creating the conditions for recurrent typhoid. Each confirmed typhoid episode became a diagnostic destination in itself, consuming clinical attention and delaying recognition of the underlying malignancy. The combination of two masking mechanisms — mimicking stone disease through ureteric compression, and enabling recurrent typhoid through immunosuppression — made this one of the more complex diagnostic presentations in haematological oncology.

Final Diagnosis

Parameter	Details
Primary Diagnosis	1 Diffuse Large B-Cell Lymphoma, Activated B-Cell or Non-GCB Subtype
Stage	Stage IIIB — lymphoma present on both sides of the diaphragm with all three B symptoms confirmed

B Symptoms

Persistent fever exceeding 38°C for more than one month; drenching night sweats; weight loss of 18 kg representing 25% of body weight

International Prognostic Index Score

3 out of 5 — High-Intermediate Risk

Ki-67 Proliferation Index

88% — highly aggressive tumour biology

Concurrent Diagnoses

Bilateral nephrolithiasis (incidental, not the primary obstructing lesion); resolved E. coli urinary tract infection; lymphoma-driven right ureteric obstruction; small right pleural effusion of lymphomatous origin

Duration from First Symptom to Diagnosis

14 months

Pre-Chemotherapy Interventions

Before initiating systemic chemotherapy, the multidisciplinary team — comprising oncology, nephrology, urology, dietetics, and psychiatry — carried out the following preparatory measures.

- Right ureteric DJ stenting was performed to relieve the lymphoma-mediated obstructive uropathy and protect residual renal function before the introduction of nephrotoxic chemotherapy agents.
- Allopurinol 300 mg daily and aggressive intravenous hydration were commenced for tumour lysis syndrome prophylaxis, given the high Ki-67 index and bulky disease presenting significant TLS risk.
- Nutritional rehabilitation was initiated under dietitian supervision with high-calorie oral supplementation three times daily. The patient had lost 25% of his body weight and required nutritional restoration before and during treatment.

- Pre-chemotherapy vaccinations were administered — Pneumococcal (PCV13), Influenza, and Meningococcal — as immunosuppressive therapy would preclude effective vaccination response afterward.
- Baseline echocardiogram confirmed LVEF of 64%, which was required before commencing doxorubicin, an anthracycline with cumulative cardiotoxic potential.
- Sperm banking was arranged given the patient's age, family planning considerations, and the gonadotoxic risk of cyclophosphamide.
- Dental clearance was obtained to reduce the risk of odontogenic infection during expected periods of neutropenia.
- The patient and his family received detailed counselling by the psycho-oncology team regarding the diagnosis, treatment duration of 10 months, likely side effects, occupational leave requirements, and financial planning.

Treatment Protocol

Treatment consists of two phases. The first is an induction phase of R-CHOP-15 chemotherapy administered every 15 days for 6 months, totalling 12 cycles. The second is a maintenance phase of Rituximab monotherapy administered once a month for 4 months. The decision to use R-CHOP-15, the dose-dense 15-day interval variant rather than the conventional 21-day R-CHOP-21 schedule, was based on three factors. First, the Non-GCB subtype of DLBCL is known to carry inferior outcomes with standard R-CHOP-21. Second, the Ki-67 proliferation index of 88% indicated rapid inter-cycle tumour regrowth. Third, the IPI score of 3 placed the patient in the high-intermediate risk group. Dose-dense scheduling with mandatory G-CSF support was adopted to counter these adverse biological features.

Induction Phase — R-CHOP-15 Drug Regimen

Drug

Class

Dose and Route

Day Given

Mechanism of Action

Rituximab

Anti-CD20 Monoclonal Antibody

375 mg/m² IV infusion

Day 1

Binds CD20 on malignant B-cells; induces apoptosis and antibody-dependent cellular cytotoxicity

Cyclophosphamide

Alkylating Agent

750 mg/m² IV

Day 1

Cross-links DNA strands and prevents tumour cell replication

Doxorubicin (Hydroxydaunorubicin)

Anthracycline

50 mg/m² IV push

Day 1

Intercalates DNA and inhibits topoisomerase II; generates free radicals causing cell death

Vincristine (Oncovin)

Vinca Alkaloid

1.4 mg/m² IV (maximum 2 mg)

Day 1

Disrupts microtubule assembly and arrests mitosis at the metaphase stage

Prednisolone

Corticosteroid

100 mg oral

Days 1 to 5

Synergistic anti-tumour effect; reduces inflammation and supports tolerability of cytotoxics

Supportive and Prophylactic Medications

Medication

Purpose

G-CSF (Filgrastim or Pegfilgrastim)

Administered Day 3 of each cycle; mandatory with R-CHOP-15 to prevent febrile neutropenia at the shortened 15-day interval

Ondansetron and Dexamethasone pre-chemotherapy

Anti-emetic prophylaxis; essential for maintaining nutritional intake and patient comfort

Pantoprazole 40 mg once daily

Gastroprotection during high-dose prednisolone on Days 1 to 5 of each cycle

Co-trimoxazole (Septran DS) three times per week

Prophylaxis against Pneumocystis jirovecii pneumonia throughout the treatment period

Acyclovir 400 mg twice daily

Prophylaxis against herpes zoster reactivation, which is common during B-cell depleting therapy

Intrathecal Methotrexate at Cycles 1, 4, and 8

Central nervous system prophylaxis given Non-GCB subtype, IPI of 3, and elevated LDH — CNS relapse risk estimated at 10 to 15%

Blood glucose monitoring each cycle

Prednisolone on Days 1 to 5 causes transient hyperglycaemia requiring active monitoring and management

Cycle-by-Cycle Clinical Progress

The following table summarises the patient's clinical and laboratory response across the 12

cycles of induction chemotherapy, including side effects encountered and management steps taken.

Cycle

Timeline

Clinical Response

Key Laboratory Values

Side Effects and Management

Cycle 1

Week 1 to 2

Fever resolved within 5 days of Rituximab infusion; night sweats reduced

LDH 1,040 to 740; Haemoglobin 10.4; ANC nadir 1,200 on Day 8

Grade 2 nausea controlled with ondansetron; mild alopecia starts; low-grade tumour lysis — managed with IV fluids and allopurinol

Cycle 2

Week 3 to 4

B symptoms fully resolved; left cervical node reduced from 3.1 cm to 1.8 cm; patient able to tolerate solid meals

LDH 540; CRP 42; ANC nadir 900 — G-CSF given on schedule

Grade 3 neutropenia at nadir; febrile episode Day 10 — blood cultures negative — treated with IV piperacillin-tazobactam

Cycle 3

Week 5 to 6

Weight 58 kg, up from nadir 54 kg; appetite improving; patient begins part-time remote work

LDH 310; Beta-2 microglobulin 2.8; Creatinine 1.0 — DJ stent functioning well

Grade 1 peripheral neuropathy in fingertips from vincristine; constipation managed with lactulose; Grade 1 mucositis

Cycle 4

Week 7 to 8

DJ stent removed — right hydronephrosis resolved on repeat ultrasound as para-aortic nodes regress; creatinine normalising

ANC nadir 800 Day 8; Haemoglobin 11.2; Platelets 290,000

Vincristine neuropathy worsening — dose reduced by 25%; steroid-induced hyperglycaemia — metformin 500 mg twice daily started

Cycle 5 — Interim PET-CT

Week 9 to 10

Interim PET-CT after Cycle 4: Deauville Score 2 — near-complete metabolic response; FDG-avidity markedly reduced across all nodal stations

LDH 198; CRP 11 — near normal

Complete alopecia; patient using cap; fatigue improving; Grade 1 neuropathy stable following vincristine dose reduction

Cycle 6

Week 11 to 12

No palpable lymphadenopathy; spleen no longer palpable; pleural effusion resolved on chest X-ray; weight 61 kg

Haemoglobin 12.1; LDH 180; all nodes clinically undetectable

Mild myalgia; mood low — referred to psycho-oncology; sleep disturbance noted and addressed with sleep hygiene counselling

Cycles 7 to 12

Weeks 13 to 24

Continued progressive clinical improvement each cycle; patient tolerating bi-weekly schedule with adopted dose modifications; returned to full-time remote work by Cycle 9

Serial LDH normalising; CBC recovering appropriately between cycles with G-CSF support; weight reaching 63 kg

Cumulative vincristine neuropathy managed with dose cap at 2 mg; fatigue remains the

main complaint; no new or unexpected toxicities in later cycles

End-of-Induction Assessment

After completion of the 12th cycle of R-CHOP-15, a formal response assessment was performed using PET-CT, CT imaging, and laboratory studies.

Assessment

Finding

Interpretation

PET-CT End of Induction

Deauville Score 1 — no FDG-avid lesions; all previously involved nodal stations metabolically negative

Complete Metabolic Response confirmed

CT Abdomen and Pelvis

Para-aortic region clear; prior 6.8 cm mass fully resolved; right ureter unobstructed; no residual lymphadenopathy

Complete Radiological Response

LDH

172 IU/L — within normal range

Normalised

Beta-2 Microglobulin

1.9 mg/L — normal

Normalised

Complete Blood Count

Haemoglobin 12.9; WBC 5,600; ANC 3,200; Platelets 310,000

Bone marrow recovery confirmed

Creatinine

0.88 mg/dL

Renal function fully restored following relief of obstructive uropathy

Body Weight

63 kg — BMI 22.0

9 kg recovered from nadir; ongoing nutritional rehabilitation

Echocardiogram Post-Induction

LVEF 60% — minor reduction from baseline 64%

Within acceptable limit; anthracycline cumulative dose monitored; cardiology follow-up arranged

Patient-Reported Status

Fatigue 4 out of 10; Grade 1 neuropathy in fingertips persisting; mood improved; returned to full-time remote work

Functional recovery ongoing but incomplete at end of induction

Maintenance Phase — Rituximab Monotherapy

Following confirmed complete metabolic remission, the patient transitioned to the maintenance phase. Rituximab 375 mg/m² was administered intravenously once a month for four months. The rationale was to consolidate the deep remission achieved during induction, given the high-risk Non-GCB biology and the high proliferation index.

Month

Dose

Clinical and Laboratory Status

Observations

Maintenance Month 1

375 mg/m² IV

No lymphadenopathy; LDH normal; weight 64.5 kg

Mild infusion-related chills on first maintenance infusion; managed with paracetamol pre-medication; resolved within 30 minutes

Maintenance Month 2

375 mg/m² IV

CBC stable; neuropathy improving — Grade 0 to 1

Well tolerated; fatigue now 2 out of 10; patient cycling regularly for physical rehabilitation

Maintenance Month 3

375 mg/m² IV

Surveillance CT: no new or recurrent lymphadenopathy; previously involved nodes remain resolved

Complete remission maintained; psychological status significantly improved; returned to social activities

Maintenance Month 4

375 mg/m² IV

End-of-treatment PET-CT: Deauville Score 1; full complete metabolic remission confirmed

Treatment completed; patient enters surveillance phase in complete remission after 10 months of total therapy

Adverse Effects and Management

The following table summarises all significant adverse effects encountered during the treatment period, their severity, the causative agent, and how each was managed.

Adverse Effect

Grade

Causative Agent

Management

Febrile Neutropenia — Cycle 2

3

Cyclophosphamide and Doxorubicin

IV piperacillin-tazobactam; G-CSF administered on accelerated schedule; blood cultures

negative; recovered by Day 14

Peripheral Neuropathy

1 to 2

Vincristine — cumulative

Vincristine dose reduced by 25% from Cycle 4 and capped at 2 mg per cycle; neuropathy stabilised at Grade 1; resolving during maintenance phase

Nausea and Vomiting

1 to 2

Multi-agent

Ondansetron and dexamethasone pre-chemotherapy; metoclopramide as rescue; adequately controlled throughout

Alopecia

3 — complete hair loss

Cyclophosphamide and Doxorubicin

Expected and anticipated; scalp cooling not feasible at the treatment centre; wig and head covering support arranged; hair regrowth commenced 6 weeks post-final cycle

Mucositis

1

Cyclophosphamide and intrathecal Methotrexate

Chlorhexidine mouthwash and sodium bicarbonate rinses; resolved spontaneously within each cycle

Steroid-Induced Hyperglycaemia

Moderate — recurrent each cycle

Prednisolone Days 1 to 5

Blood glucose monitoring before and after each cycle; metformin 500 mg twice daily added from Cycle 4; normalised between cycles

Fatigue

2 to 3 during induction

Multi-agent and disease burden

Structured rest periods; physiotherapy referral; nutritional optimisation; Grade 1 by end of treatment

Cardiac — LVEF reduction

1 — subclinical

Doxorubicin cumulative dose 360 mg/m²

Cardiology co-monitoring throughout; LVEF 60% at end of induction within acceptable limits; follow-up echocardiogram at 6 months post-treatment planned

Psychological Distress

Significant — not formally graded

Diagnosis, prolonged misdiagnosis, treatment burden

Psycho-oncology counselling from Cycle 6; family therapy; peer support group introduced; no pharmacological intervention required

Rituximab Infusion Reactions

1 — chills and flushing

Rituximab

Pre-medication with paracetamol and chlorphenamine; rate reduction on first infusion; subsequent infusions well tolerated

Discussion

Why Did the Diagnosis Take 14 Months

The delay in this case arose from a phenomenon well recognised in clinical medicine: anchoring bias. Once typhoid was diagnosed and treated — with genuine, if temporary, symptomatic relief — all subsequent symptoms were interpreted within that established framework. The concurrent discovery of renal calculi on ultrasound provided a visible structural explanation for flank pain, haematuria, and renal impairment. The two diagnoses reinforced each other, creating a self-sustaining diagnostic framework that excluded lymphoma despite escalating constitutional symptoms.

The fact that the Salmonella bacteraemia was real, culture-confirmed, and responded to antibiotics further entrenched the misdiagnosis. The treating clinicians were not wrong to treat typhoid — they were wrong to accept it as the complete explanation for a patient who was losing weight, sweating through the night, and deteriorating month after month despite appropriate antimicrobial therapy.

The absence of classical early lymphoma signs also contributed. Palpable lymphadenopathy did not become evident until Month 11 to 12. The para-aortic nodes — the primary disease site — are anatomically inaccessible to clinical examination and can only be identified through cross-sectional imaging. Without an early indication for CT or MRI, the disease grew silently for over a year.

The Role of Immunosuppression in Perpetuating Misdiagnosis

Non-Hodgkin Lymphoma, and DLBCL in particular, causes progressive impairment of both T-cell and B-cell mediated immune responses. In typhoid-endemic regions, this immunosuppression manifests as susceptibility to recurrent or persistent Salmonella infections that would ordinarily be cleared by an intact immune system. A clinician encountering a third episode of confirmed typhoid in an otherwise young and apparently healthy adult should consider an underlying immunocompromising condition — including haematological malignancy — before attributing recurrence to environmental re-exposure or inadequate treatment.

Choosing R-CHOP-15 Over Standard R-CHOP-21

The conventional first-line treatment for DLBCL is R-CHOP-21, administered once every 21 days for six cycles. In this patient, the oncology team chose the dose-dense R-CHOP-15 schedule — every 15 days for 12 cycles — based on three concurrent adverse features: Non-GCB subtype with known inferior outcomes on R-CHOP-21, a Ki-67 of 88% indicating rapid tumour regrowth between cycles, and IPI 3. The dose-dense approach aims to prevent inter-cycle tumour proliferation. Mandatory G-CSF support is a non-negotiable companion to this schedule to prevent the febrile neutropenia that would otherwise result from the shortened recovery interval.

Rationale for Maintenance Rituximab

Rituximab maintenance is an established standard in follicular lymphoma and is increasingly adopted in selected DLBCL patients following complete remission after induction. In this case, four monthly cycles were chosen to consolidate the deep complete metabolic response, given the high-risk Non-GCB biology, the elevated proliferation index, and the 14-month period during which the disease was advancing untreated. The maintenance phase was well tolerated and the patient remained in complete remission at its conclusion.

The Psychological Cost of Delayed Diagnosis

For a 38-year-old father of two young children, the emotional impact of spending 14 months being treated for the wrong conditions — losing nearly a quarter of his body weight, being told he needed kidney surgery, watching his health decline with no satisfying explanation — was profound. The eventual diagnosis of an aggressive lymphoma brought clarity but also grief, anger, and fear. The psycho-oncology team identified significant adjustment disorder with features of depression from mid-treatment onward. Early psychological integration from the point of diagnosis, rather than from the point of distress, is a lesson this case reinforces clearly.

Prognosis and Surveillance Plan

The patient completed 10 months of active treatment and achieved complete metabolic remission confirmed on end-of-treatment PET-CT. He enters a structured surveillance programme as follows.

Parameter	Detail
Estimated 5-Year Progression-Free Survival	55 to 65% based on IPI 3, Non-GCB DLBCL, and complete remission after dose-dense

induction

Estimated 5-Year Overall Survival

60 to 70%

Highest Risk Period for Relapse

First 24 months following completion of treatment; [1] majority of relapses in DLBCL occur within this window

Clinical Review Schedule

Every 3 months in Years 1 and 2; every 6 months in Years 3 to 5; annually thereafter

Surveillance Investigations Each Visit

Clinical examination, LDH, CBC

Imaging Schedule

CT chest, abdomen, and pelvis at 6, 12, and 24 months; PET-CT only if clinical relapse is suspected — not routine

Cardiac Monitoring

Echocardiogram at 6 months and 12 months post-treatment given cumulative anthracycline exposure

Renal Monitoring

Kidney function test every 6 months given prior obstructive uropathy history

Neuropathy Review

Neurology referral if vincristine-related neuropathy worsens; currently resolving toward baseline

Post-Treatment Vaccination

Pneumococcal booster and annual influenza vaccine recommended; B-cell immune reconstitution following Rituximab takes 6 to 12 months

Fertility

Sperm banking completed pre-chemotherapy; fertility specialist review at 12 months post-treatment if the patient wishes to plan conception

Conclusion

This case illustrates how three genuine, independently treatable conditions — recurrent Salmonella typhoid, bilateral nephrolithiasis, and urinary tract infection — converged to conceal a Stage IIIB Diffuse [1] Large B-Cell Lymphoma in a 38-year-old man for 14 months. Each diagnosis was correct. Each was treated appropriately. But none was ever examined in relation to the others, and the unifying diagnosis was consequently missed.

Two mechanisms made this masquerade particularly effective. The lymphoma compressed the right ureter through a 6.8 cm para-aortic nodal mass, producing hydronephrosis that appeared indistinguishable from stone obstruction. At the same time, lymphoma-driven immunosuppression permitted recurrent intracellular Salmonella infections that, on each occasion, provided a plausible and treatable explanation for the fever, weight loss, and systemic decline.

With the benefit of appropriate cross-sectional imaging and a willingness to revisit the diagnostic framework, the true diagnosis was eventually established. Prompt initiation of R-CHOP-15 chemotherapy followed by Rituximab maintenance produced a complete metabolic remission. The patient has recovered his weight, resumed employment, and re-engaged with family life.

Sources

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