

A rare bladder presentation with refractory disease at end of treatment
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CLINICAL PRESENTATION

19-year-old male • ECOG 2/4 • No comorbidities

November 2025

- Lower abdominal pain
- One week of haematuria and dysuria

20 Nov 2025 — CT angiogram

- Anterolateral bladder-wall thickening

PATHOLOGY &

Integrated diagnosis: ALK-positive anaplastic large cell lymphoma (systemic, CD30+).

- ALK: positive
- CD30: positive
- CD3: patchy positive

Morphology: large pleomorphic cells with horseshoe / kidney-shaped “hallmark” nuclei

BACKGROUND

- Distinct mature CD30+ T-cell non-Hodgkin lymphoma.
- Most cases carry t(2;5)(p23;q35) with NPM–ALK fusion and constitutive ALK signalling.
- ALK-positive disease predominates in children and young adults and is more often seen in males.
- ALK positivity is a favourable prognostic factor versus ALK-negative ALCL.
- Extranodal disease is common; the urinary

FRONTLINE THERAPY

Brentuximab vedotin + CHP

BV 1.8 mg/kg + cyclophosphamide + doxorubicin + prednisone.

Anti-CD30 antibody–drug conjugate delivering MMAE directly to CD30-positive tumour cells; replaces vincristine in the CHOP backbone.

ECHELON-2: median PFS 48.2 vs 20.8 months (HR 0.71).

TREATMENT COURSE

Dec 2025 — HRCT: bilateral pleural effusions.

Dec 2025–Apr 2026 — BV + CHP; six cycles planned. Last cycle 21 Apr 2026.

During therapy — large left pleural effusion required chest-tube drainage; left lung abscess managed in parallel.

27 Feb 2026 — Interim PET/CT: presacral node SUVmax 2.1; focal C6/C7 uptake; bladder residue non-avid.

INTERIM PET/CT — 27 FEB 2026

PRESACRAL NODE

Solitary FDG-avid node, SUVmax 2.1 → viable disease.

C6/C7

Focal FDG uptake, SUVmax 3.4 → marrow involvement queried.

BLADDER

Anterolateral thickening metabolically inactive

END-OF-TREATMENT — 19 MAY 2026

2026 response with new pelvic nodal disease

- New right common iliac and bilateral pelvic side-wall nodes (SUVmax 2.3; Deauville 4).
- Bladder thickening (39 × 16 mm) is now non-avid — morphologic residue only.
- Presacral node stable (SUVmax 1.7).
- C6/C7 uptake resolved.
- Lung abscess resolved; chest tube remained in situ.

CLINICAL INTERPRETATION

The index bladder lesion became metabolically inactive, while new FDG-avid pelvic nodes appeared.

This local-response / nodal-progression discordance is the central discussion point of the case.

SALVAGE STRATEGY

Crizotinib for refractory ALK-positive ALCL

- Reported ORR ~80–90% in relapsed/refractory ALK-positive ALCL after chemotherapy failure.
- Directly inhibits NPM–ALK, the oncogenic driver.
- COG and AcSé–crizotinib data support activity in young patients (median age ~19).

Alternatives raised: alectinib, brigatinib, lorlatinib, BV re-treatment, or salvage chemotherapy

MONITORING & RESISTANCE

- Optimal duration of ALK inhibition remains uncertain.
- NPM–ALK PCR may have a role in monitoring.
- Resistance mutations such as ALK I1171N may influence subsequent therapy.
- Role of consolidative autologous or allogeneic transplant after response.

KEY LEARNING POINTS

- Bladder-wall thickening can represent lymphoma and mimic urothelial malignancy.
- CD30 and ALK expression with an appropriate T-cell / epithelial panel is central to diagnosis.
- The bladder lesion showed metabolic response despite new pelvic nodal disease after BV–CHP.
- Young patients with ALK-positive ALCL have targeted and transplant-based salvage considerations.

FOR PANEL DISCUSSION

Q1. How should a non-avid bladder residue with new FDG-avid pelvic nodes be interpreted?

Q2. What is the optimal salvage after BV–CHP: crizotinib, next-generation ALK inhibition, BV re-treatment, or salvage chemotherapy?

Q3. Should response to ALK inhibition be consolidated with autologous or allogeneic transplant?

Q4. What is the optimal duration of ALK inhibition and role of NPM–ALK PCR monitoring?

Q5. In LMIC settings with constrained access to next-generation inhibitors and transplant, what salvage pathway is pragmatic and evidence-aligned?

SELECTED REFERENCES FROM SOURCE PRESENTATION

- Horwitz et al. ECHELON-2. Lancet 2019; 393:229–240; 5-year update, Annals of Oncology 2022.
- COG and AcSé–crizotinib data in relapsed/refractory ALK-positive ALCL.
- Case imaging, histopathology, PET/CT and treatment details are derived from the submitted case presentation.