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CASE REPORT

CD4+ chronic lymphocytic leukemia in an 86-year-old male veteran: A case report

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Abstract

CD4+ chronic lymphocytic leukemia (CLL) represents an extremely rare example of phenotypic aberrancy within CLL. We present a case of an 86-year-old male veteran with a history of multiple comorbidities who was incidentally diagnosed with CD4+ CLL during a routine peripheral blood workup. This case highlights the diagnostic challenges and characteristic features of CD4+ CLL, including flow cytometric analysis, molecular, and fluorescence in situ hybridization findings. The patient was classified as asymptomatic CLL Rai stage 0, warranting regular monitoring without a need for treatment intervention.

KEYWORDS
chronic lymphocytic leukemia, immunophenotype, mutations

1 | INTRODUCTION

Chronic lymphocytic leukemia (CLL) is a hematologic malignancy that is characterized by the proliferation of clonal mature B lymphocytes in peripheral blood and bone marrow, with the involvement of lymph nodes, spleen, and other lymphoid tissues [1]. CLL/small lymphocytic lymphoma (SLL) is a low-grade malignancy of small mature B-lymphocytes that makes up about 25% of all leukemia cases in the United States [2, 3]. For a diagnosis of CLL, monotypic B-cell count must reach ≥ 5000 cell/uL and remain sustained for at least 3 months with characteristic morphologic and immunophenotypic features including negative expression of markers such as CD3, CD10, or CD34 [4]. From an immunophenotypic perspective, CLL can have a classic phenotype: CD5+, CD10−, CD19+, CD23+, CD200+, dim expression of CD20 and CD79a, with (−/weak) FMC7 (part of CD20 molecule) expression, or a non-classic/atypical phenotype if there is abnormal expression pattern of any of the previously mentioned markers. On flow cytometric analysis, CLL cells usually demonstrate dim surface immunoglobulin–light chain expression [5]. Clinically, CLL/SLL often takes an indolent course with a wide range of clinical outcomes that range from stable disease to long-term overall survival (OS), requiring little to no treatment (watchful waiting). However, in some instances, the disease may take a more accelerated or aggressive clinical course that requires more treatment and intervention.

There have been reports of B-cell lymphomas including variants of the CLL/SLL disease, characterized by aberrant immunophenotypic marker expression. One noteworthy, rare variant is CD4+ CLL, which is characterized by the co-expression of typical B cell markers such as CD19 and CD20 alongside one of the T-cell markers (CD4) [6–11]. This variant is extremely rare and accounts for less than 1% of all cases reported thus far [5]. CD4 is a T-cell-associated marker expressed by many T-cell lymphoproliferative disorders and is also expressed by monocyte macrophages and dendritic cells. Due to its rarity and distinct features, the clinical behavior and management of CD4+ B-cell CLL remain undefined.

In this case report, we describe the diagnostic workup, clinicopathological findings, and management approach of an 86-year-old male veteran diagnosed with CD4+ CLL. We describe the unique characteristics of CD4+ CLL compared to cases with classic CLL/SLL phenotype. This case report aims to increase awareness and contribute to the current, albeit limited, literature on this exceedingly rare phenotypic variant of CLL.

2 | CASE PRESENTATION

An 86-year-old male veteran with a medical history of coronary arteriosclerosis, hyperlipidemia, gastroesophageal reflux disease, hypothyroidism, and diverticulosis presented to the Louis Stokes VAMC clinic for a routine blood workup. The patient was asymptomatic, aside from occasional night sweats, and physical examination was unremarkable. Laboratory investigations revealed a persistently elevated white blood cell (WBC) count of $24.2 \times 10^3/\mu$L (reference range: $4.0\text{-}11.0 \times 10^3/\mu$L), with hemoglobin (Hb) level of 14.1 g/dL, hematocrit of 44.8%, red blood cell (RBC) count of $4.93 \times 10^6/\mu$L, and platelet count of $168 \times 10^3/\mu$L. In addition, the patient's absolute lymphocyte count was notably elevated at 20.2×10^9/L.

Peripheral blood smear submitted for pathologist review showed an increased number of atypical medium-sized lymphocytes with round to slightly irregular nuclear contours and mature chromatin. Contrary to the classic morphology of CLL in the peripheral blood where cells have condensed "cracked/soccer-ball-like" chromatin and a scant amount of cytoplasm, many cells showed nuclear grooves, small nucleoli, and scant-to-moderate amounts of cytoplasm. Additionally, some cells had a monocytoid appearance with flower-like nuclear morphology, reminiscent of T-cell prolymphocytic leukemia (T-PLL) (Figure 1).

Flow cytometric analysis of peripheral blood lymphocytes was performed according to the standard clinical protocol. The peripheral blood specimen (WBC = 24.7 × 10^3 diluted to WBC = 12.35×10^3) was washed with a wash buffer (1:50 v/v 2% heat-inactivated fetal calf serum in phosphate-buffered saline [FCS/PBS]) three times to remove endogenous plasma proteins. The cells were incubated in 3 sperate tubes [ClearLLab 10C **B** Cell Tube (PN B96805): Kappa-FITC/Lambda-PE/CD10-ECD/CD5-PC5.5/CD200-PC7/CD34-APC/CD38-AA700/CD20-AA750/CD19-PB/CD45-KrO; ClearLLab 10C **T** Cell Tube (PN B96806): TCR$\gamma\delta$-FITC/CD4-PE/CD2-ECD/CD56-PC5.5/CD5-PC7/CD34-APC/CD7-AA700/CD8-AA750/CD3-PB/CD45-KrO; and a Third tube consisting of Beckman coulter CD4-PE/CD19-ECD/CD20-APC/CD3-PB/CD45-KrO). After a 15 min incubation, red cells were lysed with IOTest 3 Lysing Solution (1:9 v/v IOTest 3 Lysing Solution, PN A07799/DI Water+.25% IOTest 3 Fixative Solution, PN A07800). Samples were resuspended in PBS and then analyzed on a Beckman Coulter Navios 10-color flow cytometer instrument by collecting nearly 50,000 ungated list mode events. The List Mode Data files were analyzed using Kaluza C Software, selecting an appropriate lymphocyte gate on the combination of forward and side scatter, and analyzing cells with the most appropriate lymphocyte gate.

As shown in Figure 2, Flow dot plots reveal that the majority of cells in the lymphosum gate were of the B-cell lineage with a small T-cell population detected. B-cells were surface Kappa–light chain restricted with a non-classic CLL phenotype. The neoplastic cells showed an aberrant CD4, moderate CD20, and dim CD200 expression [MdFI is 2,515 for CD19 and 1,946 for CD200]. CD23 is not used in our panels, however presence of CD200 expression favors CLL [additional CD19/CD200 dot blot is added to Figure 2]. Additionally, the cells exhibited dim CD38 expression, and were negative for CD2, CD5, CD8, CD10, and CD34.

Further cytogenetic analysis was conducted using fluorescence in situ hybridization (FISH) with DNA probes specific for CLL. The FISH analysis demonstrated trisomy 12 in 61.5%, explaining the monocytoid appearance of CLL cells observed on peripheral blood smear [12] and a 13q deletion in 71.5% of nuclei from the patient's cultured cells. No aberrations involving ATM/11q, TP53/17p13, or CCND1::IGH—t(11::14) were detected. NGS study was also conducted which included IGHV mutation status. The NGS study revealed a *MYD88* p.L265P mutation with a variant allele frequency (VAF) of 32%. No *TP53* or *NOTCH1* mutations were detected. Of note, variants of uncertain significance (VUS) involving *ATM* (p.M2531T) with a VAF of 41%, *JARID2* (p.R733L), *RICTOR* (p.R907C), and *TSC2* (p.E92V) were detected. The mutational status of the IGHV gene region was also assessed and showed an unproductive V-D-J rearrangement and was therefore of indeterminate prognostic significance according to the European Research Initiative on CLL (ERIC) guidelines [13].

Based on the overall morphologic and immunophenotypic findings, the patient was diagnosed with asymptomatic CD4+ CLL, staged clinically as Rai stage 0. Given the asymptomatic nature of the disease and the absence of indications for immediate treatment, a watch-and-wait approach with regular monitoring was implemented.

3 | DISCUSSION

CD4+ CLL is an exceedingly rare phenotypic variant of CLL, accounting for less than 1% of all CLL cases in the literature. Similarly, CD8+ CLL is another rare subtype that has also been reported in only a handful of cases in the literature [7, 8, 14], see also Table 1. The presence of CD4 on neoplastic B cells is atypical, as it is usually associated with T-cell lymphoproliferative and/or monocytic disorders. Diagnostic analysis of a case like this often involves comprehensive evaluation through flow cytometry, which reveals the aberrant co-expression of T-cell markers such as CD4 or CD8 along with typical B-cell markers such as CD19, CD20, CD22, or CD79a. This case highlights the significance of comprehensive flow cytometric, FISH, and NGS analysis when working up cases of CLL, especially when there is/are unusual marker(s) expression. The FISH analysis demonstrated a trisomy 12 and a 13q deletion, which are recurrent genetic abnormalities in CLL. Trisomy 12 is detected in roughly 20% of CLL patients and generally indicates an intermediate to a less favorable prognosis. Deletion of 13q is a more common cytogenetic aberration seen in 50–60% of CLL patients and is associated with a favorable prognosis [15, 16], though the predicted

TABLE 1 Summary of T-cell-associated markers aberrantly expressed on some B-cell leukemias and lymphomas.

Authors	Marker	# Cases	Diagnosis	Outcome
Jani et al. [11]	CD4	1	Chronic lymphocytic leukemia (CLL)	The patient was clinically stable at the time of reporting; no treatment was given.
Kaleem et al. [6]	CD4 & CD7	1	Diffuse large B-cell lymphoma	The patient was clinically stable and was lost on follow-up after 13 months.
	CD8	2	CLL	One patient was lost on follow-up after 6 years with persistent disease. The other patient was lost on follow-up after 2.5 years, with no evidence of aggressive disease.
	CD2	2	CLL/SLL	Both patients were clinically stable at the time of reporting; no treatment was given. Follow-up continued for 3 and 8 years, respectively.
Parisi-Duchêne et al. [10]	CD8	5	CLL	Two out of five patients died (1 and 6 years after diagnosis) one of which had breast cancer. Follow-up data was not provided for the remaining patients.
Kern et al. [25]	CD8	61	CLL	Clinical follow-up data was only available for 12 out of the 61 patients. Only six required therapy. Notably, the CD8+ patients had **higher ZAP-70** expression and less favorable outcomes (shorter median time-to-treatment) than CD8− patients (12.0 months vs. 77.1 months, respectively).
Islam et al. [26]	CD8	1	CLL	The patient required no treatment for the first 6 years, but the disease **progressed** and fludarabine phosphate therapy was given. He developed severe myelosuppression during his course of treatment and eventually died from recurrent respiratory tract infections.
Hanza et al. [27]	CD8	1	CLL	The patient was clinically stable at the time of reporting; no treatment was given.
Jain et al. [28]	CD8	1	CLL	The patient was clinically stable at the time of reporting and was followed up for 9 months; no treatment was given.
Carulli et al. [7]	CD8	11	CLL/SLL	Seven patients were clinically stable and required no treatment. The remaining four patients had **progressive** disease and required chemotherapy, two of which showed **ZAP-70** and **CD38** expression.
		1	Marginal zone lymphoma	The patient was clinically stable at the time of reporting; no treatment was given.
		1	Lymphoplasmacytic lymphoma	The patient was clinically stable at the time of reporting; no treatment was given.
Li et al. [14]	CD8	1	CLL	The patient was clinically stable at the time of reporting and was followed up for 17 years; no treatment was given. Throughout this period the patient gradually developed bilateral axillary, cervical, supraclavicular, hilar, and retroperitoneal lymphadenopathy.
Espinosa et al. [8]	CD8	1	CLL	The patient was treated and within two weeks of treatment, there was a marked reduction in leukemic cells.
Schroers et al. [29]	CD8	1	CLL	The patient was clinically stable at the time of reporting; no treatment was given.
Venugopalan et al. [30]	CD7	2	CLL	The clinical outcomes of these patients were not reported.

Note: The majority of the cases have been diagnosed as CLL/SLL with an indolent course that requires no treatment, similar to the classic CLL and other CD4-expressing CLL reported in the literature. The majority of the patients with aberrant CD8 expression show stable clinical course, however, some patients tend to show disease progression, have a higher % of ZAP-70 expression, and a less favorable outcome [shorter median time-to-treatment (TTT)] than CD8-negative CLL patients. This aberrant marker expression does not quietly correlate with IGHV gene mutation status.

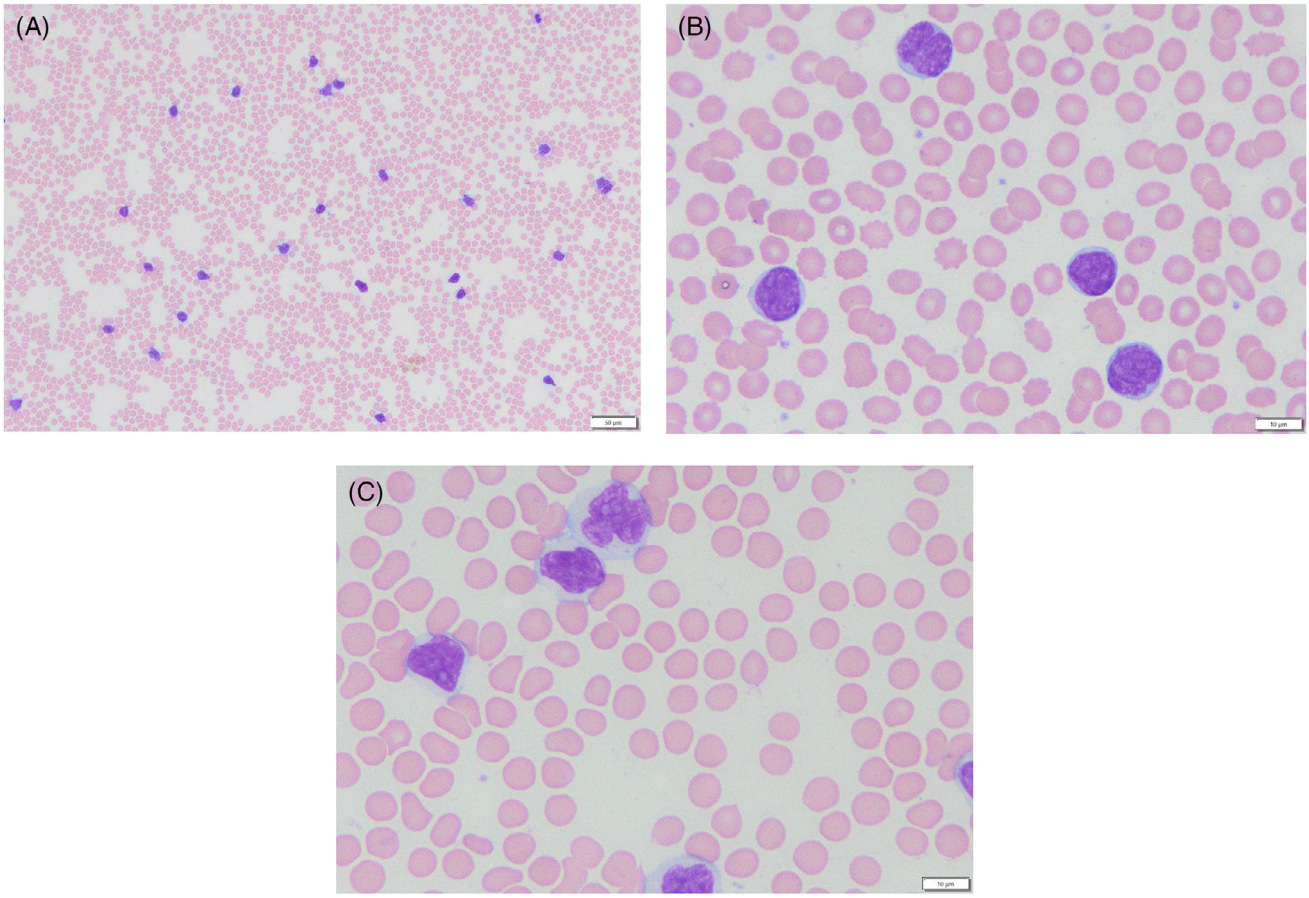

FIGURE 1 Peripheral blood smear images: Atypical lymphocytes with nuclear grooves and monocytoid features. (A) 20X magnification; (B, C) 100X.

benefit in OS may be attenuated by the presence of > 60% of cells having the 13q deletion in this patient [17]. The absence of ATM/11q, TP53/17p13, or CCND1::IGH—t(11::14) in this case also suggests a relatively favorable prognosis [18].

The NGS study revealed a relatively high VAF of *MYD88* p.L265P mutation, a variant considered to have disease relevance but whose prognostic significance remains unclear [19]. *MYD88* mutations have been reported in 4%–10% of CLL cases and these tend to have a more atypical immunophenotype compared to wild-type, and those with the L265P mutation at the *MYD88* gene also tend to be younger and display a variety of favorable prognostic factors, including mutated *IGHV* [20]. However, in this case, NGS detected an unproductive V-D-J rearrangement in the *IGHV* gene region is exceedingly rare (< 0.1% of all CLL). This mutation status is deemed to have indeterminate prognostic significance according to ERIC guidelines [13]. No *TP53* or *NOTCH1* mutations, both of which are associated with poor outcomes [21–23] were detected. The clinical significance of *JARID2*, *RICTOR*, and *TSC* VUS remains in need of further investigation.

As observed in our case, CD4+ CLL can present asymptomatically, similar to the classic CLL and other CD4-expressing CLL reported in the literature. The majority of the patients with aberrant CD8 expression show stable clinical course, however, some patients tend to show disease progression, have a higher % of ZAP-70 expression, and a less favorable outcome [shorter median time-to-treatment (TTT)] than CD8-negative CLL patients, (see Table 1). IgHV mutation status seems to be irrelevant to CD8 expression. Treatment decisions are usually based on the presence or absence of constitutional symptoms, rate of rise of lymphocytosis, significant liver, or spleen enlargement, and/or cytopenia. In our case, the patient was classified as Rai stage 0, asymptomatic CLL, which typically warrants no treatment with close observation instead [24]. The prognosis, clinical behavior, and management strategies for CD4+ CLL still remain poorly understood due to the limited number of cases reported in the literature and the rarity of this variant. Further reporting of cases and close clinical follow-up would be necessary to enhance the understanding of this uncommon variant.

4 | CONCLUSION

In conclusion, we present a rare case of CD4+ CLL in an 86-year-old male veteran with multiple comorbidities. The diagnostic evaluation included flow cytometry, and molecular and FISH analysis, confirming the presence of atypical CD4+ CLL. The patient was classified as asymptomatic CLL Rai stage 0, indicating a favorable prognosis and supporting a watch-and-wait approach with regular monitoring. Further research is necessary to enhance our understanding of the

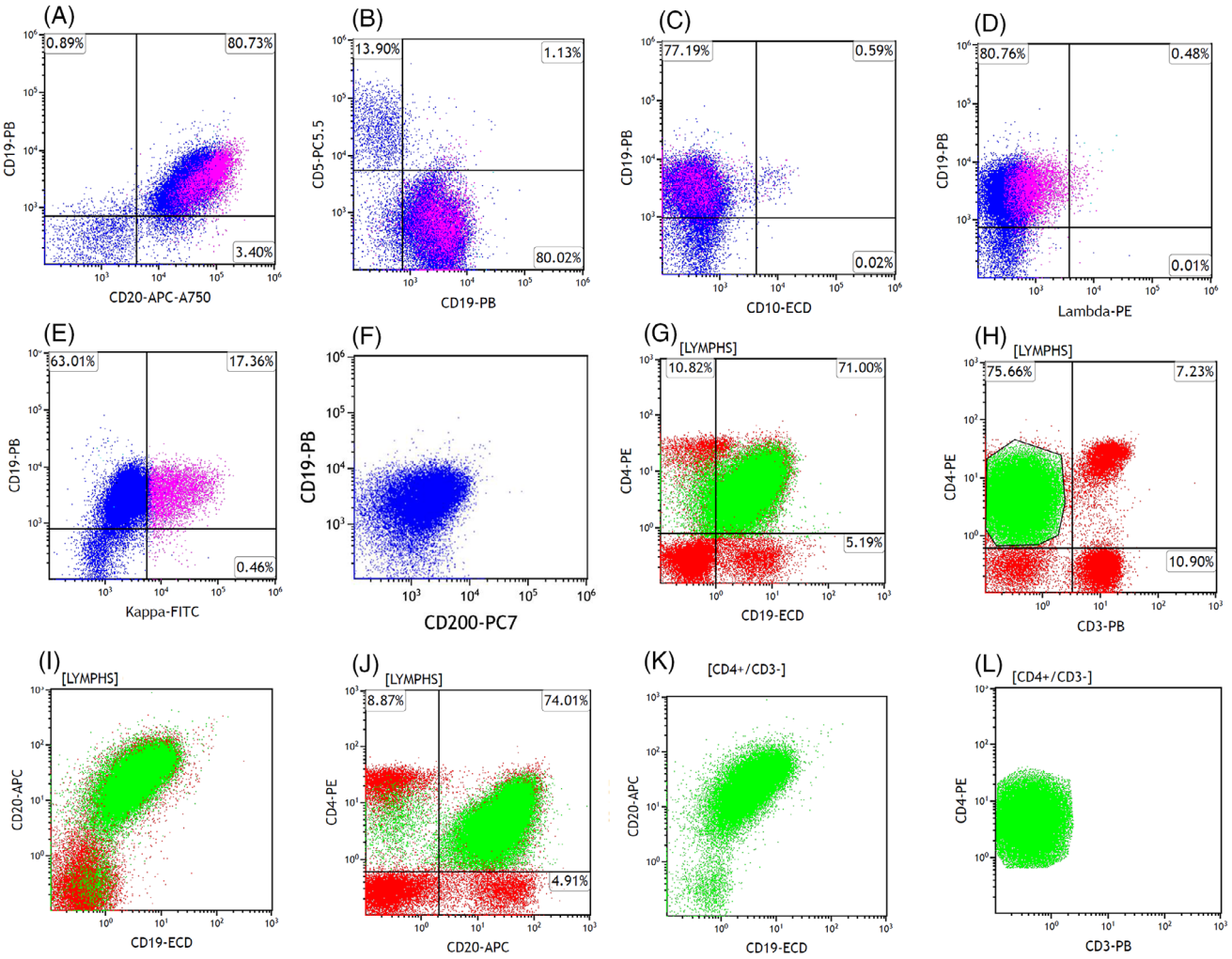

FIGURE 2 Flow cytometric characteristics of CD4+ CLL in peripheral blood specimen: Panels (A–F) total-lymphosum gating: show CD19+/CD20+ (A) CD5 negative (B), CD10 negative (C), with (dim)Kappa restriction (D, E) and CD200 expression (F), with similar MdFI to CD19 [2,515 for CD19 and 1946 for CD200, respectively]. Panels (G–J) total-lymphosum gating: show that CD19+ B-cell population identified in previous panels are CD4 positive (G), CD3 negative (H), and CD20+ (J). Panels (K, L) sabgating on "CD4+/CD3−" population: The population coexpresses B-cell specific markers "CD19 and CD20" (K) and is negative for T-cell specific marker "CD3".

clinical behavior and optimal management strategies for CD4+ CLL, given its rarity and distinct features reported herein.

AUTHOR CONTRIBUTIONS

Sara Abdelrahim, Glory H. Thai, and Hany Sakr wrote most of the manuscript. Juanita Burke wrote the flow cytometry section in the paper. Hany Sakr collected images and dot plots, the latter with the help of Juanita Burke. Chen Zhao, Timothy O'Brien, and Mohammad Q. Ansari read and revised the paper, and agreed with the final version of the paper. All authors read and approved the final manuscript.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

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DATA AVAILABILITY STATEMENT

The datasets generated are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

The authors have confirmed ethical approval statement is not needed for this submission.

PATIENT CONSENT STATEMENT

The patient gave verbal consent, as per treating clinician Dr. O'Brien.

CLINICAL TRIAL REGISTRATION

The authors have confirmed clinical trial registration is not needed for this submission.

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