

Nilotinib-Associated Demyelinating Disease

Casey Judge DO

Lehigh Valley Health Network, Casey.Judge@lvhn.org

Negar Moheb MD

Lehigh Valley Health Network, Negar.Moheb@lvhn.org

Christopher Melinosky MD

Lehigh Valley Health Network, Christopher.Melinosky@lvhn.org

Follow this and additional works at: <https://scholarlyworks.lvhn.org/medicine>

Part of the [Internal Medicine Commons](#), and the [Neurology Commons](#)

Published In/Presented At

Judge, C. Moheb, N. Melinosky, C. (2019, May 6). *Nilotinib-Associated Demyelinating Disease*. Poster Presented at: American Academy of Neurology (ANA) Annual Conference, Philadelphia, PA.

This Poster is brought to you for free and open access by LVHN Scholarly Works. It has been accepted for inclusion in LVHN Scholarly Works by an authorized administrator. For more information, please contact LibraryServices@lvhn.org.

Nilotinib-Associated Demyelinating Disease

Casey J Judge, DO¹, Negar Moheb, MD², Christopher Melinosky, MD¹

¹ Department of Neurology, Lehigh Valley Health Network, Allentown, PA ² Department of Medicine, Lehigh Valley Health Network, Allentown, PA

INTRODUCTION

Tyrosine kinase inhibitors (TKIs) have revolutionized oncology, allowing for targeted, non-toxic chemotherapy. However, these medications may actually have unanticipated side effects. TKIs, such as nilotinib, have been associated with autoimmune associated side-effects, including reports of peripheral and central nervous system demyelination^{1,2,3,4}.

CASE DESCRIPTION

A 62-year-old woman with medical history significant for well-controlled chronic myeloid leukemia (CML) on maintenance nilotinib for 5 years presented with a ten day history of descending weakness and sensory loss, followed by dyspnea requiring emergent intubation. Neurologic exam revealed quadriparesis and areflexia with a seemingly intact though limited sensory exam.

CLINICAL COURSE AND RESULTS

Initial MRI C spine with questionable T2 abnormality at C2, attributed to motion

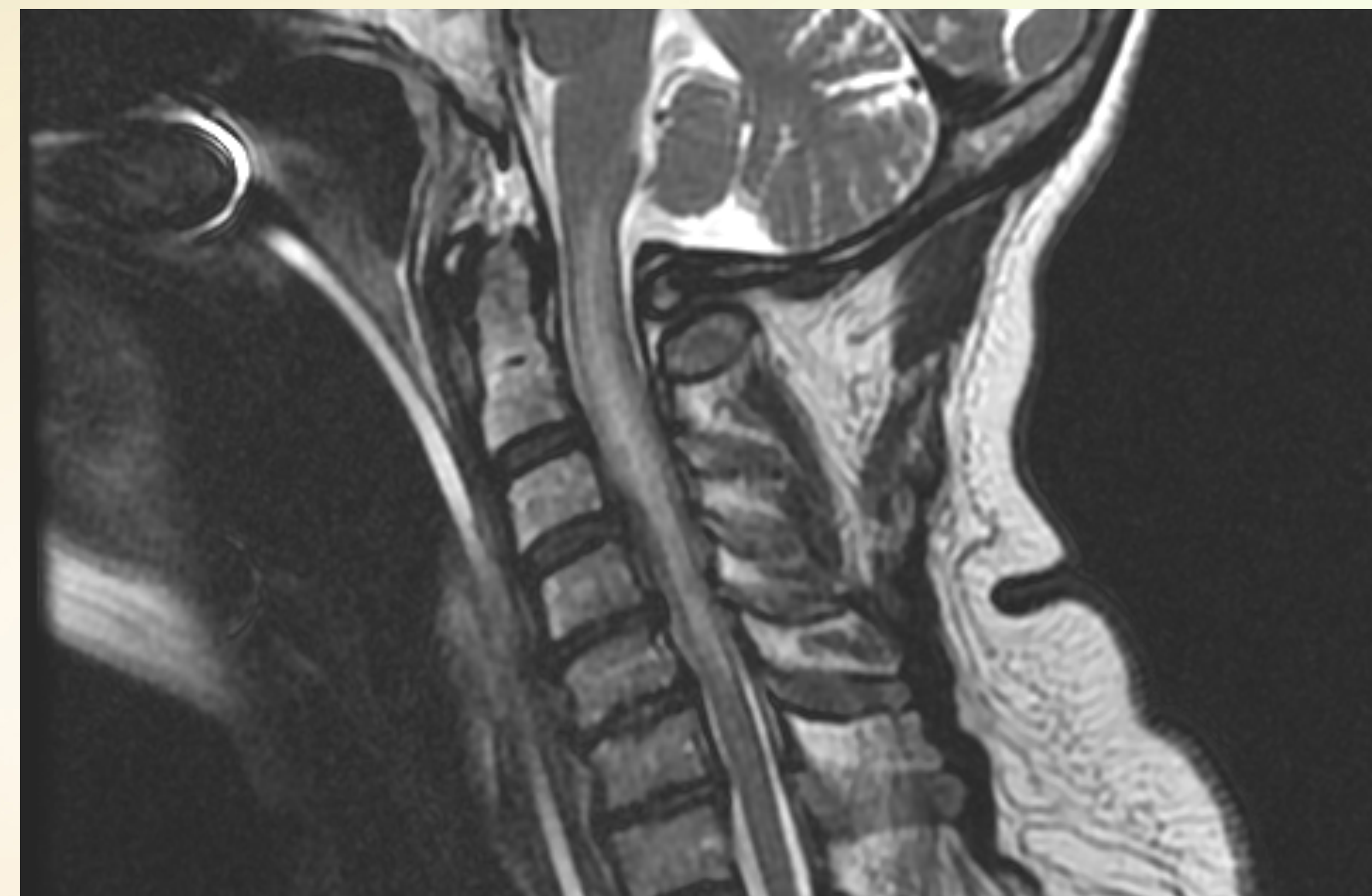
Lumbar puncture: protein 82mg/dL and 9 WBCs/uL

CLINICAL COURSE AND RESULTS (CONT.)

Primarily concerned for Guillain-Barre Syndrome (GBS), started on 2gm/kg intravenous immunoglobulin (IVIg)

Patient developed C2 sensory level. Repeat MRI C spine demonstrated T2 hyperintensity C2-C7

MRI brain, aquaporin-4 antibodies, MOG antibodies, and Mayo clinic cerebrospinal fluid paraneoplastic and autoimmune panels all negative.



Reviewed case reports of nilotinib-associated demyelination. With oncology guidance, nilotinib discontinued.

Patient required tracheostomy and percutaneous gastric feeding tube. Discharged with minimal improvement in upper extremities and ventilator dependence.

CONCLUSIONS

TKIs, including nilotinib, dasatinib, and imatinib may be associated with demyelinating disease of the central and peripheral nervous system.

Demyelination may develop at any time during the treatment course. Pathology and deficits may not be reversible.

The mechanism is postulated to be autoimmune mediated.

Early recognition of these potential severe side effects and discontinuation of therapy may improve outcomes.

It is unknown if immune-modulating therapy (IVIg or plasma exchange) has any impact on outcome.

REFERENCES

- Rekhi E, Pryce A, Sohal M, et al CNS Demyelination in Patients On nilotinib treatment for CML. *Neurol Neurosurg Psychiatry* 2016;87:e1.
- Mulherin B, Loconte NK, Hoen KD. Guillain-Barré syndrome after treatment with sunitinib malate. *Oncology (Williston Park)*. 2008 Jan;22(1):66-7, 70-1.
- Kavanagh S, Brill V, Lipton JH. Peripheral neuropathy associated with imatinib therapy for chronic myeloid leukemia. *Blood Res*. 2018;53(2):172-174.
- Ishida T, Akagawa N, Miyata T, Tominaga N, Iizuka T, Higashihara M, Suzuki T, Miyazaki K. Dasatinib associated reversible demyelinating peripheral polyneuropathy in a case of chronic myeloid leukemia. *Int J Hematol*. 2018 Mar;107(3):373-377