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

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
1Department of Neurology, Lehigh Valley Health Network, Allentown, PA 2Department 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 demyelination1,2,3,4.

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 quadriplegia 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

Primarily concerned for Guillain–Barre Syndrome (GBS), 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.

REFERENCES

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.