Abstract:
Vincristine is an antitubulin vinca alkaloid used in combination with other agents in the treatment of leukemia and other malignancies. A dose-limiting complication of vinca alkaloids is neurotoxicity. Vincristine is the oldest and most neurotoxic agent in this group. This is a case of a 65 year old white male with bilateral optic atrophy secondary to Vincristine therapy for Hodgkins disease during the 1970s. This presentation should make physicians aware of the ocular risk with vincristine therapy.

35 word abstract:
Vincristine is a vinca alkaloid known for its neurotoxicity. This is a case of a 65 year old white male with bilateral optic atrophy secondary to Vincristine therapy for Hodgkins disease during the 1970s.

I. Case History
- Patient demographics
  65 yowm
- Chief complaint
  Patient feels he has decreased VA at distance for past few years
- Ocular, medical history
  (+) h/o smoking 2 packs/day for about 2 years in early 1970's
  (-) h/o EtOH, other substance abuse
  (+) multiple episodes of trauma in service to the head with loss of consciousness
  (-) exinuation/major surgeries/treatment for "bad blood" per patient
  (+) h/o Hodgkins disease s/p chemotherapy in 1970's - pt reports was on Vinblastine for about 1 month - lead to bilateral graying in vision few days after initiation of therapy and then vinblastine was changed to vincristine.

PTSD, hyperlipidemia, impotence

-Medications
Ketoconazole 2% shampoo, propranolol hcl 10mg tab bid prn, Urea 40% cream, simvastatin 80mg tab qhs, Docusate NA 100mg 1 cap PO qd, Vardenafil HCL 20mg, gabapentin 600mg 1 tab PO tid, Trazodone HCL 100mg 1-2 tab PO for sleep, Bupropion HCL 150mg 1 tab bid

-Other salient information
None

II. Pertinent findings
-Clinical
Objective
Habitual correction
  OD -3.00 -0.75 x074
  OS -2.50 -0.50 x119 add +2.50
VA Snellen Habitual Correction
  OD 20/ 25 PH 20/2
  OS 20/ 25-2 PH 20/ NI
Pupil Assessment
  Equal size Normal
  Round Normal
  Reactive to Light Normal
  Afferent Pupillary Defect (APD) Normal
Anterior chamber depth estimation:
  Shadow test
  Deep OU? Yes

EOM = full and smooth    Fields = FTFC OD/OS
Color vision: 14/14 OD, OS

MVA     Sphere  Cylinder  Axis  Bifocal
       OD  -3.00 - 1.00 x 074  +2.50
       OS  -2.50 - 0.75 x 119  +2.50

Distance VA
20/25-2    PPA: 20/25
20/30+2    PPA: 20/30

SLIT LAMP EXAM: Tonopen OD 13 OS 15 Aug 24,2011@08:25
Conjunctiva(-)
Cornea(-)
Anterior Chamber Deep & Quiet
Lids/Lashes(-)
Iris(-)
Lens(+) trace NS OU with few central cortical vacuoles OS

FUNDUS Mydriacyl 1% and Neosynephrine 2.5% @ 8:26am OU
C/D OD: 0.45/0.45, OS: 0.65/0.45; 2+ diffuse pallor OU
Macula clear OU
Vitreous: PVD OU; (-) shaffer's sign OU
Vessels 2/3 a/v OU
Periphery no holes/tears OU

OCT: RNFL analysis: optic disc cube 200x200
OD: signal strength 8/10; avg thickness 50; red superior, temporal, inferior; yellow nasal
OS: signal strength 8/10; avg thickness 48; red superior, temporal, inferior; yellow nasal

HVF 24-2
Reliable OU
OU: Enlarged blind spot

-Physical
Temp 97.8F
BP 127/77
Height 71 in
Weight 190lbs
BMI 26.67

-Laboratory studies

BLOOD    08/24/2011    Reference

  11:10    Units    Ranges
WBC        4.5 L    K/uL  4.87 - 9.47
RBC        4.75 M/uL  4.38 - 5.43
HGB        13.8 g/dL  13.11 - 16.01
HCT        40.9 %    40.53 - 48.45
MCV        86.1 fL   79 - 100
MCHC       29.1 pg   27.82 - 31.71
MCHC       33.7 H    g/dL  31.89 - 33.53
RDW        %    11.5 - 14.5
PLT        180 K/ul  150 - 400
MPV        9.5 L fL  10.03 - 11.7
NEU. %     50.4 L %  51.16 - 68.51
LYMPH %    42.6 H %  19.12 - 34.14
MONOS %    5.5 L %  6.78 - 13.7
EOS. %     1.1 %    1.04 - 4.56
BASO. %    0.4 %    .18 - .81
NEU. #     2.28 L K/uL  2.44 - 6.34
LYMPHS#    1.93 K/uL  1.19 - 2.5
MONO #     0.25 L K/uL  .5 - .91
EOS #      0.05 L K/uL  .07 - .31
BASO. #    0.02 K/uL  .01 - .06
PLT. EST   Ref: Normal
ATYPICA    %    0 - 7
NRBC       #/WBC's  0 - 1
RBC APP    ANISO 0 - 1+
POIKILO    0 - 1+
MICROCY    MACROCY
POLYCHR    HYPOCHR
GRAN %     %    42.2 - 75.2
GRAN #     x1000 1.4 - 6.5
PLT EST    Ref: NORMAL

ACE 51 U/L
Sed Rate 11 mm/hr
RPR nonreactive
Vitamin B-12 595 pg/mL
Folate 42.7 ng/mL
Lymes disease: pending
ANA: pending

-Radiology studies
Chest x-ray
Report: The heart, lungs, mediastinum, bony thorax and soft
tissues are negative.

Impression:
Normal Chest.

III. Differential diagnosis
-Primary/leading
toxic optic neuropathy
-Others
Glaucoma; after central retinal vein or artery occlusion; ischemic optic neuropathy; chronic optic neuritis;
chronic papilledema; compression of the optic nerve, chiasm, or tract by a tumor or aneurysm; traumatic
optic neuropathy; Syphilis; Leber optic atrophy; congenital amaurosis; lysosomal storage disease;
radiation neuropathy; congenital or hereditary optic atrophy.

IV. Diagnosis and discussion
ASSESSMENT:
1. Bilateral optic atrophy likely secondary to previous chemotherapy for Hodgkins disease in 1970s

2. Refractive error OU

PLAN:
1. Order optic atrophy workup to rule out other etiologies and follow-up accordingly with patient. Repeat VF 24-2.

2. New glasses ordered today.

-Elaborate on the condition
Patients taking or have taken vincristine because of a malignancy are associated with some variable of neurotoxicity. These toxicities are usually dose dependant and reversible upon cessation of the drug, which is not the case in this case. A patient recently presented with long standing decreased vision. Upon fudus examination bilateral optic atrophy was observed. Patient had a history of Hodgkins disease and was treated with vincristine in the 1970s. Appropriate testing was done to rule out other possible causes of optic atrophy and all testing came back unremarkable. We attributed this patient’s vision loss and optic atrophy to vincristine toxicity involving the optic nerves.

-Expound on unique features
Bilateral optic atrophy

V. Treatment, management
Patient will be monitored in 6 months with OCT and HVF 24-2 to monitor any possible progression in his condition. If no significant progression noted patient will be monitored yearly with OCT and HVF 24-2. Progression is unlikely due to the length of time that has passed since cessation of vincristine.

-Bibliography, literature review encouraged
1. Optic Atrophy Induced by Vincristine, Pediatrics 1982; 70;288, Shurin,MD, Rekate,MD, and Annable MD.

2. Retinal and Optic Nerve Atrophy Induced by Intravitreous Vincristine In the Primate; Ophth. Soc., vol ZXXIII, 1975; Green, MD.


5. Vincristine Sulfate as a Possible Cause of Optic Neuropathy; DOI 10.1002/phc.20638; Weisfeld-Adams, Dutton, Murphy.

VI. Conclusion
-Clinical pearls, take away points if indicated
Patients who have taken or are taking vincristine should be monitored for ocular side effects and vision loss with visual field and OCT. Signs of optic atrophy should be examined with investigative studies to rule out other causes of optic atrophy before vincristine toxicity is considered.