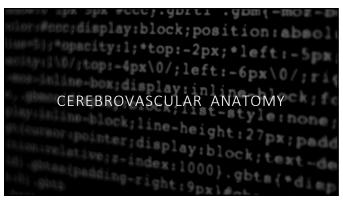


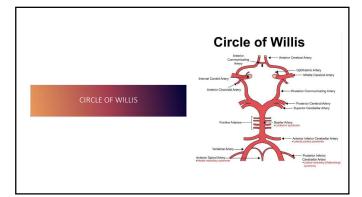


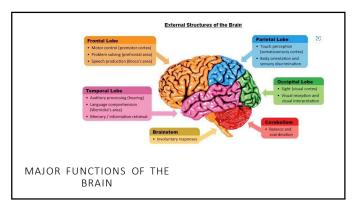
AGENDA

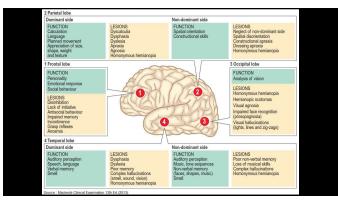
- INTRODUCTION TO
 CEREBROVA SCULAR ANATOMY
- 2. STEPS THROUGH STROKE STOP AND
- 3. RISK FACTORS FOR WOMEN
- 4. PREGNANCY AND POST-PARTUM
- 5. RUN THROUGH ACTUAL CASES

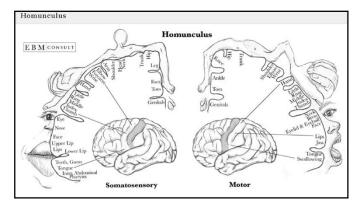
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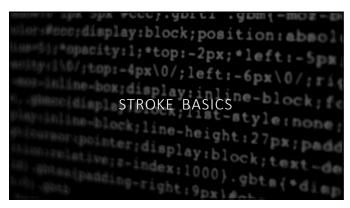


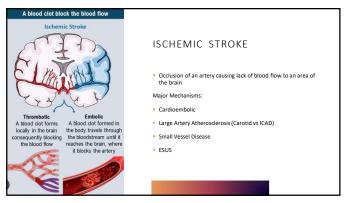


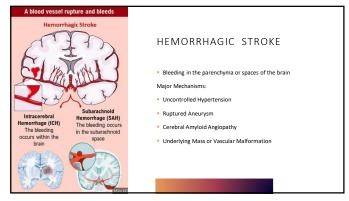


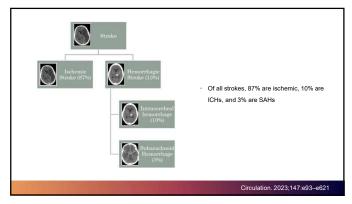




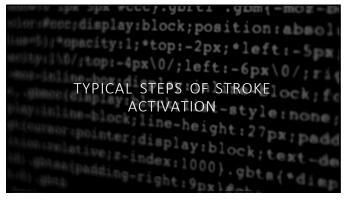








CEREBRAL VENOUS SINUS THROMBOSIS Women are at risk of suffering this rare form of stroke → CVST Thrombosis of the sinuses that drain the brain Major Mechanism: Hypercoagulability – inherited or situational Coagulopathy – APA, FVL, MTHFR Pregnancy, OCP, Trauma, Infection Can cause ischemic or hemorrhagic strokes



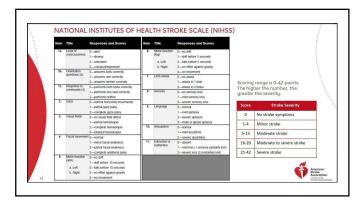
STROKE STOP

- Stroke page activated
- NIH Stroke Scale Performed at Stroke Stop
- Try to get patient to CT scanner in 5 minutes
- CTH w/o + CTA + CTP
- Decide quickly if patient is a tPA candidate or Mechanical Thrombectomy Candidate
- They go back to the ER room to either get tPA or for further evaluation
- Or they go to the angio suite for thrombectomy

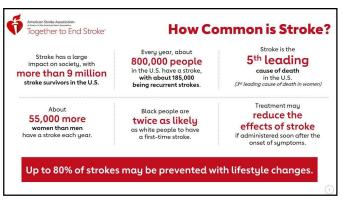


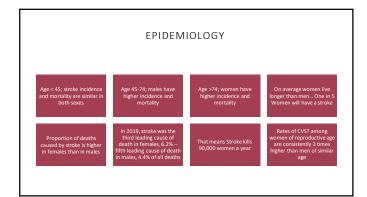
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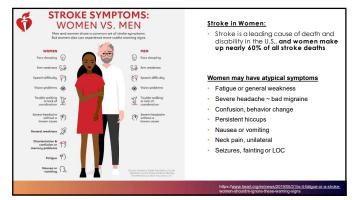
NIH STROKE SCALE

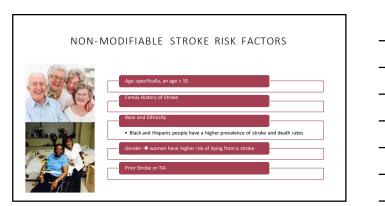












MODIFIABLE RISK FACTORS

Medical Risk Factors:

- Hypertension
 - Stroke risk increases as your baseline blood pressure increases
 - Blood pressure > 120/80 is abnormal
- Diabetes Mellitus doubles your risk of stroke
- Cholesterol build up increases risk of stroke
- Carotid Disease can cause circulation problems and increase risk of stroke
- Atrial Fibrillation increases strokerisk up to 5 times

Lifestyle Risk Factors:

- Tobacco use and Vaping
 - Current smokers have 2-4 times the stroke risk
- Heavy Alcohol Use
- Weight and Obesity increase risk of stroke and cardiovascular events
- Physical Activity
- Sedentary lifestyle increases strokerisk
- Diet
 - High salt and added sugar intake increases
 risk

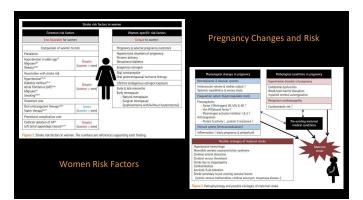
22

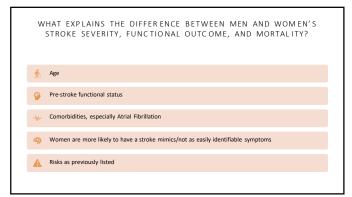
WOMEN SPECIFIC STROKE RISK FACTORS

- Age at onset of menarche
 - Early Onset < 13
 - Late Onset > 16
- Oral contraceptive use
 - ~2.5-fold increased risk of ischemic stroke
 - ~1.4-fold increased risk of hemorrhagic stroke
- Menopause
 - Early menopause has increased risk; whether natural or surgically caused (oophorectomy)
- Hormone replacement therapy
- Lifetime endogenous estrogen exposure
- Pregnancy*
- Hypertensive disorders of Pregnancy
- Pre-term Delivery
- Gestational Diabetes
- Puerperium (6-8 weeks)
- Migraine with Aura

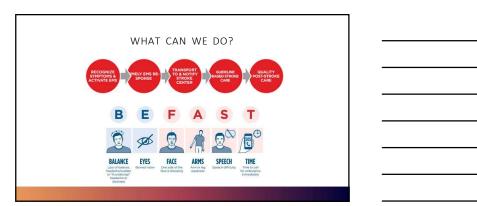
Journal of Stroke 2023;25(1):2-15

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PRESENTATION

- LKW around 3 a.m. on 6/20/24
- Acted weird around 3 a.m., then went back to sleep
- Woke up and suddenly lost consciousness around 12 p.m. and became unresponsive → EMS called and taken to local ER
- Intubated and sedated due to lack of airway protection
- In OSH ED found to have Bilateral PE's and started on Heparin gtt

PMHx: 56-year-old female

- On oral contraceptives
- No other medical history otherwise healthy

amily Hx:

Grandparent with lymphoma, no other history known

Social Hx:

Never Smoker

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WHAT WORK UPDO YOU WANT TO DO NEXT?

OMAHA CAMPUS

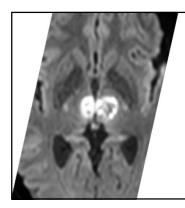
(CTA OF THE HEAD AND NECK)	
31	
WHAT SYMPTOMS DO YOU EXPECT WITH BASILAR	-
OCCLUSION?	
KEAR NEY CAM PUS	
32	
BROUGHT HER TO UNMC FOR MECHANICAL	
THROMBECTOMY (ANGIOGRAM)	
33	



HOSPITAL COURSE CONTINUED

- ADMITTED TO THE ICU POST THROMBECTOMY, TICI 3
- STARTED ON ASPIRIN 81 MG AND HIGH INTENSITY STATIN (ATORVASTATIN 80 MG) AFTER MT (HD#2)
- FOUND TO HAVE HYPODENSITIES OF LIVER/KIDNEY + SEVERAL DVTS
- HD#2 WENT FOR THROMBECTOMY OF BILATERAL PE'S
- LATER THAT EVENING STARTED ON CUSTOM HEPARIN GTT

34



HOSPITAL COURSE CONTINUED

- UNDERWENT IVC FILTER PLACEMENT HD#3
- MRI BRAIN SHOWING BILATERAL THALAMIC, LEFT MIDBRAIN, LEFT PONS, MULTIFOCAL CEREBELLAR, OCCIPITAL, LEFT TEMPO-PARIETAL INFARCTS
- PFO PRESENT ON ECHOCARDIOGRAM

35

WHAT CAUSED THE STROKE? WHAT'S YOUR DIFFERENTIAL?

NORFOLK CAMPUS

WOULD	YOU	DO AN	YTHING
ELSE	FOR	WORK	UP?

SCOTTS BLUFF CAMPUS

37

HYPERCOAGULABILITY WORK UP

- Upon further questioning, found to have significant family history of factor V Leiden (suspected in 2 maternal aunts and her father); however, no other significant family history of VTE
 Tested for Paroxysmal nocturnal hemoglobinuria, Factor V Leiden, APA, Lupus anticoagulant, cardiolipin antibody, inflammatory markers, Beta-2-glycoprotein, etc.
- CT C/A/P

- Results:

- Results:

 Factor V Leiden Negative

 Antiphospholipid Antibody Testing Negative

 PNH testing negative

 CT C/A/P 5 Similar under distention versus wall thickening along the lateral cecal wall potentially neoplasm

 PET scan 3 Intense focal activity localizing to the lateral wall of the eccum which appears

Eventually....

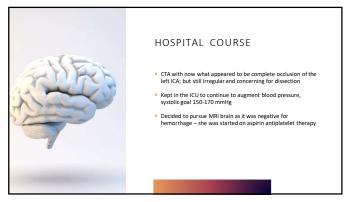
Found to have tubulovillous adenoma with high grade dysplasia + tubular adenomas → can eventually turn into high grade adenocarcinoma

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	PRESEN [*]	
• W	KW 1245 on 11/22/23 Vas at the airport when suddenly she had acute et inability speak + right arm weakness	PMHx: 29-year-old female No other medical history – healthy
	IIHSS originally 8 for aphasia and right sided	 Not on any medications, including OCP Family Hx:
• C part	TA Head and Neck without bleed; evidence of cially occlusive L ICA thrombus	 Significant for HTN, a few still births
		Social Hx: • Former Smoker
10		
10		
	(CTA HEAD ANI) NECK 11/22)
11		
	WOULD SHE QU	
	INTERVE	NTION?
	OM AHA	CA MPU S

	HOSPITAL COURSE
Ų,	Was given tPA and admitted to the ICU
9	Went from global aphasia, significant right sided weakness, lethargy to
(D)	Being awake, alert, with improved R side weakness, R arm 4/5, R leg 5/5. She was able to say some words (yes or no, one, two) but still was having difficulty following complex commands.
4	Unfortunately, had worsening of her exam again → but improved back to the prior exam with blood pressure augmentation



(MRI BRAIN)

SIGNIFICANT EVENT	
Overnight on HD#3 patient had worsening expressive aphasia Code Stroke Called Went to CT Scanner and this revealed	
46	
	1
(CTA 11/26)	
47	<u> </u>
WHAT WOULD YOU LIKE TO DO?	
KEAR NEY CA MPU S	

MEDICAL MANAGEMENT

- Started on heparin gtt without bolus
- Continued on aspirin antiplatelet therapy
- Continue blood pressure augmentation with same systolic goal of 150-170 mmHg
- Continue hourly neuro checks
- Discussed possibility of vascular intervention or even DSA, but neurosurgery very cautious due to her risk for vascular injury and dissection
- Again planned to hold off on any intervention at this time planning for medical management
- Planned for weaning of vasopressors, since further dissections occurred after admission while she had been augmented

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WORKUP/RESULTS

- Hypercoagulability Work Up: Negative
- SPEP, ANCA, Lyme, Cardiolipin, Lupus, Syphilis, Hepatitis, HIV, West Nile, VZV, Complement, RF, ANA → all within normal limits
- A1c, lipid panel, FA, B12, UA, UDS → WNL
- No family history of connective tissue disorders, no uterine rupture history with her personal history of deliveries of her children
- Continued to have intermittent worsening exams → found to have mostly occluded the vessels that were dissected
- Transitioned to DAPT and since then improved
- Discharged to AR

- In the end:
- had L ICA dissection → occlusion
- R ICA dissection, pseudoaneurysm, partial thrombus
- L V2 and V3 dissections; R V3 dissection
- Multifocal L ACA/MCA infarcts; R Corona Radiata + Multifocal Infarcts

Symptoms:

- · Partial Gerstman Syndrome
- Left Horner's Syndrome
- RUE Weakness + Apathy/Abulia

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WHAT TOP DIFFERENTIAL DO YOU THINK OF WITH NEW ONSET, SEVERE HEADACHE

NORFOLK CAMPUS

53

WHAT IMAGING WOULD YOU LIKE TO DO?

SCOTTS BLUFF CAMPUS

(MRV 3/15/21) HOSPITAL COURSE - After finding EXST—Insenferred to VIAMC - Southed two queen or influence and of Vindide prior to transfer Total branche of thromboa. Prigit saled transverse sizes, springer domin, and profession factors allowed transverse sizes, springer doministic sizes Total branche of the rocke by Care distance in the procession of the profession factors and the profession of the pr
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Started on heparin infusion and IV fluids prior to transfer Total burden of thrombus → right sided transverse sinus, sigmoid sinus, and proximal internal jugular veln No associated hemorrhage or stroke, no signs of hydrocephalus NIHSS was 0 Found to have low B12 and started on supplementation Transitioned to oral anticoagulation, before the contact and that included ANA, cardiolipin antibodies, lupus anticoagulant panel, beta-2 microglobulin, blood protein electrophoresis, ESR and Antithrombin activity were not suggestive of an acquired thrombophilla. Admitted for a total of 3 days and received heparin during this time, with improvement in headache Evaluated and okay'd to discharge home with supplementation Transitioned to oral anticoagulation with Eliquis
sinus, signoid sinus, and proximal internal jugular vein Admitted for a total of 3 days and received heparin during this time, with improvement in headache NIHSS was 0 Found to have low B12 and started on supplementation Transitioned to oral anticoagulation with Eliquis
hydrocephalus headache NIHSS was 0
supplementation • Transitioned to oral anticoagulation with Eliquis
i l
56
(REPEAT MRV 9/28/21)

CASE #4	
PRESENTATION * LKW 1230 on 8/6/24 when she started vomiting, experiencing diplopla, decreased facial sensation on the right side, and imbalance * Had been having a few days of neck pain and headache that started on 8/2 for which she laid in bed and had pain relief with Tylenol * Presented to OSH for the above symptom changes, and then increased lethargy * CTA Head and Neck with evidence of R Vertebral Dissection and Basilar Occlusion * Declined at OSH and intubated →> transferred to UNMC for MT * Never Smoker	
(DSA)	

WHAT ARE SYMPTOMS OF BASILAR OCCLUSION AGAIN?	
61	
IS ANY INTERVENTION INDICATED? KEAR NEY CAMPUS	
(DSA POST MT)	

HOCDITAL COURCE	
HOSPITAL COURSE	
 Underwent MT of basilar occlusion, TICl 3 and admitted to the ICU, remained intubated Started on Heparin gtt and ASA (for vertebral stent) 	
 Initially unable to move LUE, intermittently following commands Proceeded with MRI for quantification of stroke, and she was found to have bilateral cerebellar infarcts and right pontine infarct 	
Also had hydrocephalus developing (comparing CTH from day prior) Went for urgent suboccipital craniectomy and C1 laminectomy + EVD placement HD#2	
, , , , , , , , , , , , , , , , , , , ,	
64	
	1
(2.45) 55.4(1)	
(MRI BRAIN)	
	_
65	
HOSPITAL COURSE	
 Found to have R Frontal Tract Hemorrhage on follow up imaging 	
 Extubated on HD#4 Remained in ICU while EVD in place, but developed no signs of hydrocephalus 	
EVD removed on HD#6 Loaded with Plavix on HD#9, heparin infusion stopped HD#10	
 Stayed in ICU for several days, but deemed safe to transfer to the floor on HD#11 – off heparin and on DAPT 	
66	

MECHANISM

- Believed to have artery to artery embolism from her right vertebral dissection
- Unsure why she originally had a right vertebral dissection
- Pregnancy Testing was negative; A1c, lipid panel, echocardiogram, telemetry all within normal limits
- Fortunately, continues to improve mostly just suffering from axial and appendicular ataxia, but improving daily

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SOURCES

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