VEBINARS- CASE CAPSULE 01

CC_01 Watershed infarcts in Patient with Aortic dissection- for weekly discussion

Sunday, 22 March 2020

Value & Evidence



Figure legend (1a-h) - Initial DWI and ADC (1a and b) show scattered bilateral juxta cortical acute infarcts. MRA (1c and d) show eccentric thickening and intimal flaps in aortic arch and carotids. Follow up MRI shows progressive infarcts (1e) while USG Doppler (1f) and CT Angiogram (1g and h) confirmed type A aortic dissection extending into bilateral carotid arteries.

CC_01: Summary

Middle aged male who presented with abrupt onset of jaw pain and decreased consciousness followed by seizures. Initial MRI with MRA (Fig 1a-d) showed scattered bilateral juxta cortical infarcts with eccentric wall thickening of aortic arch and left common carotid as well as suspicious intimal flap within right common carotid artery. Subsequent color doppler USG and CT Angiogram confirmed type A aortic dissection extending into bilateral carotid vessels. Patient worsened clinically within next few days and follow up MRI showed severe progressive bilateral infarcts (Fig e-h).

WATERSHED INFARCTS-TYPES

CC_01 Watershed infarcts

Last Meadow Concept of Brain Ischemia

https://www.ahajournals.org/doi/10.1161/STROKEAHA.115.010976

Learning points / Summary

- Two types of watershed infarctions can be distinguished:
- 1. Cortical watershed (CWS) between the cortical territories of the anterior, middle, and posterior cerebral artery, and
- 2. **Internal watershed** (IWS) in the white matter along and slightly above the lateral ventricle, between the deep and the superficial systems of the anterior and middle cerebral arteries.
- 3. **Triple watershed** triangular border zone between the cortical branches of all 3 brain arteries in region of inferior parietal lobule (temporo-parietal angular gyrus)
- · Lesser known watershed regions
- 4. In the depth of the brain, between putamen and insula at the border of the deep and superficial branches of the middle cerebral artery, and at the terminal end of perforators arteries which do not make anastomotic contact with other territories
- 5. Hippocampal formation, between end arterial branches of the anterior choroidal and posterior cerebral arteries
- 6. Cerebellum borders between the supply territories of the anterior inferior, superior, and posterior inferior cerebellar arteries
- 7. Spinal cord midthoracic level between the supply territories of the segmental artery VI which originates from vertebral artery, and the segmental artery IX which is a branch of the aorta

Various levels of the medullar—pontine—mesencephalic axis after vertebral artery occlusion (inconsistent and lesser pathophysiological importance)

WATERSHED INFARCTS-PATHOPHYSIOLOGY

CC 01 Watershed infarcts

Last Meadow Concept of Brain Ischemia https://www.ahajournals.org/doi/10.1161/STROKEAHA.115.010976

Learning points / Summary

- Most of the CBF studies agree that the IWS is more sensitive to hemodynamic failure then CWS, and IWS infarcts—especially the rosary-like pattern— are related to hemodynamic impairment
- · Hemodynamic impairment

stage I: decline of perfusion pressure and autoregulatory vasodilatation; stage II: decrease of CBF and increase of oxygen extraction fraction

- Patients with IWS infarcts had higher degrees of occlusive vascular disease and showed more severe early and late clinical course than those with CWS infarcts
- For infarcts within the CWS, hemodynamic mechanisms are more frequent in the anterior than in the posterior watershed.
- Embolism coexists with hypoperfusion, and that stagnant flow increases the risk of embolic events, facilitating the development of infarcts in the CWS

Ischemic stroke & Acute aortic dissection: Clinical recommendations for urgent diagnosis

https://www.ncbi.nlm.nih.gov/pubmed/29948797

Summary

- Approximately 1.7% of suspected stroke patients have acute aortic dissection (Stanford type A)
- It can be suspected based on the presence of chest or back pain and blood pressure laterality
 - Look for upper mediastinal widening on CXR
- Imaging study of arch (CTA including the common carotid arteries) and measure the D-dimer level
 - Avoid thrombolysis

https://www.ncbi.nlm.nih.gov/pubmed/29948797

www.ncbi.nlm.nih.gov (https://www.ncbi.nlm.nih.gov/pubmed/29948797)
Acute ischemic stroke as a complication of Stanford type A acute aortic dissection: a review and proposed clinical recommendations for urgent diagn... - PubMed - NCBI
Gen Thorac Cardiovasc Surg. 2018 Aug;66(8):439-445. doi: 10.1007/s11748-018-0956-4.
Epub 2018 Jun 13. Review

Step 1: Paramedics Recommended: Check for the presence/absence of chest or back pain, radial arterial pulse laterality*, and presence/absence of changes in the level of consciousness in suspected stroke patients No aortic dissection suspected → routine medical care for suspected stroke Aortic dissection suspected - choose a facility able to diagnose and treat aortic dissection and stroke and provide information in advance Step 2: Initial-care physicians (stroke doctors, critical care physicians, and cardiologists, among others) Required: check for the presence/absence of chest or back pain, measure bilateral blood pressures simultaneously, and take chest X-ray images If aortic dissection is suspected: contrast chest CT + head CT (MRI/MRA) (cooperate with the radiology department) Step 3: Initial-care physicians (if aortic dissection cannot be ruled out*, cooperate with the radiology department) (1) Evaluate the common carotid arteries (ultrasonography, CTA, or MRA), required; (2) "head CT/CTA including the aortic arch" or "head MRI/MRA," recommended; (3) D-dimer measurement, recommended If aortic dissection is suspected, contrast chest CT (cooperate with the radiology department)** Step 4: Consult with a specialty practice department for thoracic aortic dissection (cardiovascular surgery or cardiology department) to decide on a treatment plan

Physicians must always assume the role of a monitor and suspect aortic dissection when treating stroke patients.

* When laterality is detected on pulse palpation, measure bilateral blood pressures; aortic dissection is suspected if the laterality is ≥20 mmHg or the right upper limb blood pressure is <110 mmHg. Even if the results of measurement performed in the ambulance met this criterion, blood

pressures must be re-evaluated upon arrival at a medical facility.

#If the presence/absence of chest or back pain cannot be confirmed due to consciousness disturbance or aphasia, the presence of changes in the level of consciousness and other changes in symptoms should be taken as an indication that aortic dissection cannot be ruled out.

**Not required if CTA including the aortic arch has been performed.

CC_01 - Watershed infarct Pathophysiology

https://www.ncbi.nlm.nih.gov/pubmed/28694111

Summary

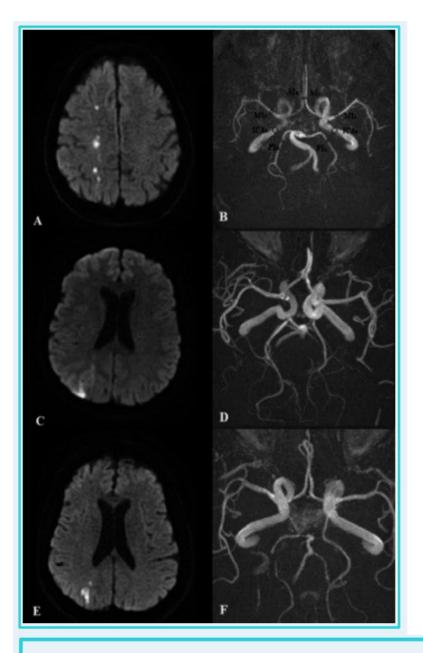
- Internal WI was associated with a statistically significant flow impairment along the carotid axis, both qualitatively and quantitatively
- Hemodynamic impairment is major trigger factor of deep Wls, often involves the centrum <u>semiovale</u> and presents with contralateral limb weakness secondary to high density of motor fibers
- CWIs, are more the product of a micro embolic phenomenon

These 2 phenomenon are probably linked: inflammation plaque leading to micro-occlusive distal events followed by hemodynamic impairment on the lumen narrowing

HEMODYNAMIC IMPAIRMENT-DEEP WATER SHED
MICROEMBOLIC PHENOMENON-CORTICAL WATERSHED

https://www.ncbi.nlm.nih.gov/pubmed/28694111

www.ncbi.nlm.nih.gov (https://www.ncbi.nlm.nih.gov/pubmed/28694111)
The Pathophysiology of Watershed Infarction: A Three-Dimensional Time-of-Flight Magnetic Resonance Angiography Study. - PubMed - NCBI
J Stroke Cerebrovasc Dis. 2017 Sep;26(9):1966-1973. doi: 10.1016/j.jstrokecerebrovasdis.2017.06.016. Epub 2017 Jul 8. Comparative Study

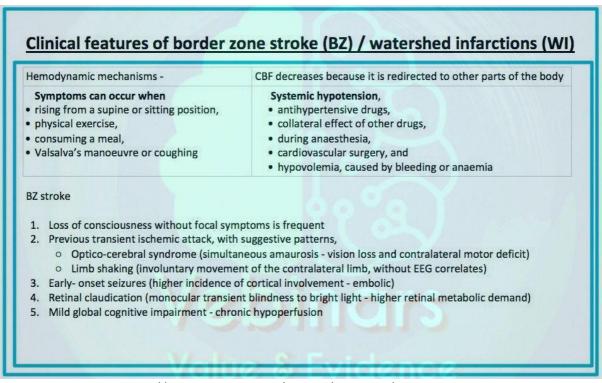


Examples of DWI-MRI associated with the 3D TOF (right).

First patient: internal watershed infarct

- (A) associated with a reduced intensity signal of the internal carotid artery (ICA) and middle cerebral artery (MCA)
- (B). Second patient: posterior cortical watershed infarct
- (C) with diminished ICA signal intensity but a normal MCA intensity
- (D). Third patient: posterior cortical watershed infarct
- (E) with a normal 3D TOF MRA (F).

WATERSHED INFARCTS- CLINICAL FEATURES



Clinical features - https://www.karger.com/Article/Abstract/333638

Karger (https://www.karger.com/Article/Abstract/333638)
Border-Zone and Watershed Infarctions

WATERSHED INFARCTS- CLINICAL FEATURES

Clinical features of border zone stroke (BZ) / watershed infarctions (WI)

Anterior (ACA-MCA) BZ infarcts

Contralateral motor impairment, sparing facial muscles (Position of arm / leg fibres along cortical spinal tract)

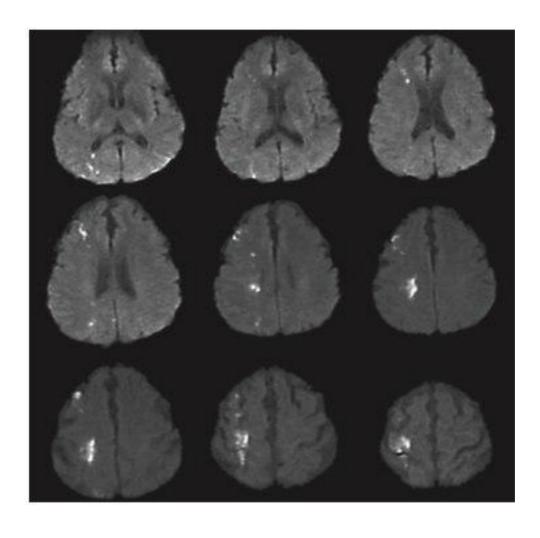
- · Cortical upper limb more involved
 - . Distal (hand / wrist DD radial nerve palsy)
 - · Proximal (Shoulder involved)
 - Bilateral (Man in barrel syndrome can walk but unable to move arms)
- . Subcortical lower limb more involved
 - Bilateral lower limb (DD acute spinal syndrome)
- Left sided Disconnection between Brocas and supplementary motor area)
 - Transcortical motor aphasia (non fluent but comprehension and repetition preserved)
- · Right sided loss of frontal executive functions
 - · apathy or euphoria,
 - If severe akinetic mutism (lack of spontaneous thought, speech and motor activity, but preserved memory or motor pathways)

Posterior (PCA-MCA) BZ infarcts

Contralateral perimetric deficit

- homonymous lower quadrantopia or hemianopia caused by interruption of optical radiations.
- Bilateral Cortical blindness (patients have no vision) with preserved non-cortical functions (distinguish light/dark and pupillary reflex is normal)
- Balint's syndrome (a triad of visuo-ocular symptoms and visual hallucinations)
 - Simultanagnosia inability to perceive simultaneous events in one's visual field
 - optic apraxia inability to fixate and follow an object with eyes
 - optic ataxia impairment of target- pointing under visual guidance
- Cortical involvement on left (Wernicke's area and posterior parietotemporal- associative areas)
 - Wernicke's aphasia (fluent aphasia with neologism, word salad and incorrect language)
 - Transcortical sensory aphasia (fluent aphasia, with poor comprehension, but good repetition)

Diffusion- weighted brain MRI showing anterior and posterior CBZ and IBZ infarcts.



CC_01 - Watershed infarcts (Treatment follow up and Prognosis) https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5576255/ BMC Neurol. 2017; 17: 166. Published online 2017 Aug 29. doi: 10.1186/s12883-017-0947-6 Clinical features and the degree of cerebrovascular stenosis in different types and subtypes of cerebral watershed infarction Cortical Watershed infarcts Anterior - more prone to critical ICA stenosis than those with posterior watershed infarction Both Deep / Internal watershed infarcts Partial (P) Critical ICA stenosis was more prevalent in

patients with partial IWI (P-IWI) than in those

IWI patients were more frequently found to have clinical deterioration during the first 7 days of hospitalization and a poor prognosis at the 90th

with confluent IWI

day than in CWI patients

CC 01 - Watershed infarcts (Treatment follow up and Prognosis)

https://www.ncbi.nlm.nih.gov/pubmed/31997490

Both (CWI & IWI)

Eur J Neurol. 2020 Jan 29. doi: 10.1111/ene.14156. [Epub ahead of print]

Confluent

Higher early recurrence risk and potential benefit of dual antiplatelet therapy for minor stroke with watershed infarction: subgroup analysis of CHANCE

Patients with watershed infarction had higher risk of stroke recurrence than those without in first week
of stroke. Dual antiplatelet therapy - <u>Aspirin & Clopidogrel</u> may be safely implemented,
yet watershed infarction mechanisms of hypoperfusion and emboli may not be addressed

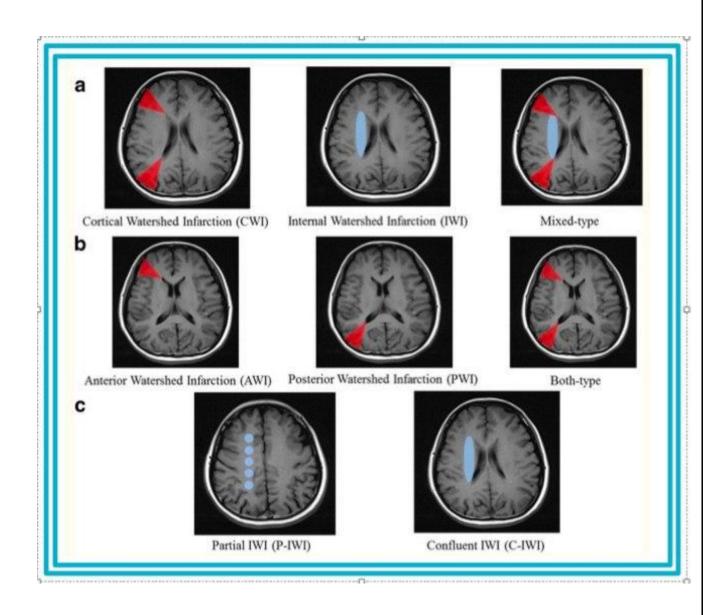
https://www.ncbi.nlm.nih.gov/pubmed/28004005

Biomed Res Int. 2016;2016:6241546. doi: 10.1155/2016/6241546. Epub 2016 Nov 28.

Clinical Comparison of Outcomes of Early versus Delayed Carotid Artery Stenting for Symptomatic Cerebral Watershed Infarction due to Stenosis of the Proximal Internal Carotid Artery

- Rate of second stroke in early CAS group (within 1 week) was lower when compared to that of delayed CAS (after 4 weeks) group. There was no difference in periprocedural complications in both groups.
- · Early CAS group had a significant better good outcome than delayed CAS group.

https://www.ncbi.nlm.nih.gov/pubmed/31997490 www.ncbi.nlm.nih.gov (https://www.ncbi.nlm.nih.gov/pubmed/31997490)



ICA NARROWING WITH CRITICAL STENOSIS AND POST STENTING ANGIOGRAPHY

WATERSHED INFARCTS- SUMMARY

CC 01 - Watershed infarcts in Aortic Dissection(Summary / Learning points)

- ► Types cortical (anterior / posterior) vs deep (partial / confluent)
- ▶ Pathophysiology Hemodynamic (deep more likely and cortical) / embolic (cortical more likely)
- ▶ Red flags for aortic dissection-Loss of consciousness, Pain (Shoulder / neck / chest / Jaw etc.) at presentation of stroke
- ▶ Asymmetrical pulses / BP between limbs might help in narrowing the suspicion
- ▶ Role of imaging Exclude proximal stenosis (most common location carotid bifurcation)
- ▶ Bilateral watershed infarcts aortic dissection needs exclusion
- ▶ Imaging Protocol: CTA should always cover aortic arch in vascular imaging of Stroke patients
- ▶ Treatment Medical- Dual antiplatelets / high dose statins initially 3 weeks followed by single agent
- ► Endovascular management / Surgical endarterectomy (Early within 1 week vs delayed after 4 weeks)

CC_01 - Watershed infarcts in Aortic dissection (Companion case)

65 years old male with history of inferior wall MI and post PCI with sudden onset weakness and unresponsiveness

DWI showing bilateral watershed infarcts (cortical and deep)
Additionally left cerebellar infarcts were seen with indistinct visualization of CCA and left VA

Source neck MRA shows artefacts at level of aortic arch - Always exclude aortic dissection with CTA covering arch. Patient follow up awaited (poor clinical status for aggressive evaluation)

