

02: Airhead: A rare case of spontaneous non-traumatic pneumocephalus

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Pneumocephalus, also referred to as intracerebral aerocele or pneumatocele, is defined as a pathologic collection of air in the cranial vault. The primary cause of pneumocephalus is head injury with trauma which counts for approximately 74% of cases with other causes including intracranial neoplasms (13%), infections (9%), neurosurgical interventions, paranasal sinus surgery, or lumbar puncture. Nontraumatic spontaneous pneumocephalus not preceded by neuro intervention or malignancy is uncommon and sparsely represented in the literature.

This case is of a 67-year-old right-handed African-American female who initially presented to our hospital as a code stroke. While the patient was at work, she reported she had used the toilet, stood up and leaned forward after which she began having a severe 8/10 headache bilaterally. This headache persisted for approximately 2 hours when she began to notice numbness of the left side of her body as well as weakness of her left arm. Coworkers called EMS due to concern for stroke and she was subsequently taken to our hospital.

Emergent evaluation with noncontrast CT head demonstrated a large volume of intracranial air. After total of approximately 4 hours, patient's symptoms resolved with her only remaining deficit being some slight tingling in her left fifth finger. CT brain demonstrated what appeared to be air tracking from the right sphenoid sinus, and ENT performed an endoscopy with demonstrated a sinus defect with dural tear.

The defect was repaired, and patient recovered without incident.

03: Imaging of Pediatric Neurovascular Lesions

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Background/Objective:

Neurovascular lesions in children may manifest with a variety of clinical presentations, from completely asymptomatic and incidentally found to life-threatening hemorrhage. To optimally characterize them often requires a multimodality imaging approach. Efficient and cost-effective imaging aids in management decisions, evaluation of potential complications and optimal patient outcomes.

Methods:

Multiple cases of pediatric neurovascular abnormalities of varying pathologies are presented to compare and contrast the imaging appearance using various modalities to help highlight key findings.

Results:

Underlying pathologic differences can have distinctive combination of imaging appearances, which can be used to accurately diagnose & guide further management to minimize risk of potential morbidity and mortality.

Conclusions:

Accurate diagnosis and characterization of pediatric neurovascular abnormalities often requires, in addition to thorough clinical history and physical exam, multimodality imaging approach to fully assess extent, eloquent vs non-eloquent involvement, flow dynamics, and presence of associated abnormalities. Some lesions pose higher risk in the pediatric population given their cumulative nature of risk for hemorrhage & long potential for future growth. Others are benign & require only conservative management. Surgical and endovascular approaches to therapy are often limited to those lesions that are symptomatic or pose significant risk of future complications. Balancing the risks of observation with the potential complications of treatment is also contemplated.

05: Pediatric Abusive Head Trauma

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Introduction:

The evaluation of potential victims of pediatric nonaccidental trauma (NAT) and specifically abusive head trauma (AHT) requires a multidisciplinary team effort. Responsibilities include recognizing new cases of NAT as well as safely excluding cases that mimic AHT. We sought to determine the incidence and type of head injuries encountered in children being evaluated for AHT as well as frequently encountered mimics that subsequently create so much controversy in the courtroom.

Methods:

This study is an IRB approved, 5-year retrospective review of children evaluated for AHT. Inclusion criteria were: children with head trauma formally evaluated by the Mayo Clinic Family Advocacy Program (MCFAP; a multidisciplinary team including pediatricians, neurosurgeons, trauma surgeons, neuroradiologists, social workers and law enforcement.) between 2011 and 2015, age younger than 12 months (as the incidence of NAT/AHT is known to be significantly greater in children less than one year of age), availability of brain/head imaging (CT, MRI and/or US). In the 60 months between January 2012 and December 2016, 275 children were referred to MCFAP for evaluation for potential NAT (both AHT and other). Of the 275, 138 (50%) were age less than 12 months. Of the 138, 87 had brain imaging (63%).

Results:

The study included 87 children with an age range of 11 days to 11 months. Of the 87, 65 (75%) had a positive imaging study of the brain. Of the 65, 15 (23%) were subsequently determined to be the result of AHT, with the remaining 50 categorized as accidental head trauma or other etiologies. As expected, the most frequently noted findings in the cases deemed positive for AHT were the classic imaging triad of abusive head trauma: subdural hematoma, retinal hemorrhage, and encephalopathy.

Conclusion:

The classic imaging triad of subdural hematoma, retinal hemorrhage, and encephalopathy is well known to those who care for victims of AHT. In many of cases that reach the courtroom, contentious exchanges arise between experts representing the prosecutors and those on the defense team. Controversies in abusive head trauma include: dating of subdural hematomas, alternative causes of “the triad,” acute on chronic subdurals vs re-bleed of pre-existing hemorrhage, overlapping findings between accidental and nonaccidental head trauma, the possibility of a “lucid interval” with subdural hematomas, the absence of neck injuries, the possibility of similar injuries from short falls and, whether shaking can actually result in such extensive injuries. Examples of positive cases as well as mimics will illustrate these controversies.

06: CT-Guided Thrombolytic Treatment of Patients with Wake-up Strokes

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Background:

Observational studies of thrombolysis outcomes in wake-up acute ischemic stroke (AIS) patients selected based on non-contrast brain CT criteria suggested that treated patients did as well as or better than those not treated, after adjustment for baseline characteristics. We began offering thrombolytic treatment (IVTPA) to patients presenting with wake-up strokes and normal non-contrast brain CTs, who could be treated within 4.5 hours of being found.

Design/Methods:

A retrospective chart review was performed in patients presenting with AIS between November 2014 and December 2017 who received IVTPA. A planned subgroup analysis compared patients with wake-up strokes and normal non-contrast brain CTs to patients with witnessed stroke treated within 4.5 hours of being found, or of witnessed onset, respectively.

Results:

Three hundred and six patients were treated, 279 with witnessed and 27 with wake-up strokes. Efficacy and safety were similar in both groups. Discharges home, respectively, were 143(53%) and 13(48%); facility discharges were 112(40.1%) and 11(40.7%) and in-hospital mortality was 19 (6.8%) and 3 (11%). Treatment-related symptomatic bleeds were: 5(1.8%) and 1 (3.7%), respectively.

Conclusions:

The findings show that it is safe to treat patients with wake-up strokes and a normal brain CT scan with IV TPA. Demonstrating safe utilization of absence of early ischemic tissue changes on CT to expand thrombolytic treatment eligibility in patients with wake-up stroke suggests that it may be safe to extend beyond 4.5 hours the treatment eligibility window in patients with witnessed stroke onset, when the non-contrast brain CT scan is normal.

07: Transthoracic Echocardiographic Changes in Cryptogenic Stroke Patients with Patent Foramen Ovale

***Resident Travel Award Recipient**

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Background and Purpose:

We sought to evaluate the association between transthoracic echocardiographic changes and patent foramen ovale (PFO) in patients with cryptogenic stroke.

Methods:

Two hundred and eighteen consecutive patients with a recent cryptogenic stroke from Jan 2015 to Jun 2018 underwent transthoracic echocardiography (TTE) and transcranial Doppler (TCD). PFO was diagnosed by the bubble test of TCD. Differences of patient characteristics and TTE findings between PFO group and non-PFO group were compared.

Results:

PFO was detected in 35.8% (78/218) of the patients. Compared with non-PFO patients, significant differences of TTE findings were seen in the diameter of aortic root (34cm vs. 32cm, p=0.005), the early diastolic peak velocity of the mitral annulus (Em) (61.5cm/s vs. 68cm/s, p=0.005) and percentage of mitral regurgitation (33.8% vs. 50.7%, p=0.0016). Threshold of diameter of aortic root and Em in predicting PFO was 33mm and 72cm/s (analyzed with ROC curve and Youden index). A three-point score of TTE criteria (AEM, including diameter of aortic root >33mm, Em<72cm/s and without mitral regurgitation) was independently associated with PFO (odd risk 1.95, 95% CI 1.37~2.78) after the adjustment of sex and history of diabetes. AEM score ≥ 2 predicted PFO with a sensitivity of 0.46 (95% CI 0.37~0.55) and a specificity of 0.77 (95% CI 0.67~0.85).

Conclusions:

Increased diameter of aortic root, lower Em and without mitral regurgitation were significantly associated with PFO. AEM score could be a useful utility to screen out the PFO in cryptogenic stroke patients.

09: Radiologic Errors in Detection of Cerebral Venous Thrombosis

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Introduction:

Thrombosis of the cerebral veins and sinuses is an emergency condition associated with intracranial venous congestion and consequent regional ischemia and cortical infarcts, most frequently hemorrhagic. Clinical presentation of venous infarct is markedly variable and different from ischemic arterial stroke. Approximately 47% of patients present with seizures (focal, focal with secondary generalization, and generalized tonic-clonic with or without further development of status epilepticus). Radiological error may delay prompt treatment with potentially lethal outcome or severe permanent brain damage. The aim of the presentation is to evaluate the presence of radiologic error in reporting cerebral venous thrombosis (CVT).

Methods:

Computerized tomography (CT) and/or magnetic resonance imaging (MRI) of the brain were reevaluated in 10 patients with CVT, affecting cerebral sinuses, deep intracranial veins or cortical veins. Known predisposing conditions in these patients were coagulopathy, malignancy and petrous apicitis.

Results:

Correct diagnosis was established immediately in only 40% of patients with CVT, while the mistake in the final report was evident in 30% of patients. In 10% of reports inconclusive finding was noted, while in 20% of patients with CVT initial report was wrong, associated with the correction in the final report. The disease was associated with two lethal outcomes while permanent neurological deficit was noted in one patient. Subtle curvilinear hyperdensities were detected within the left parietal cortico-subcortical border zone on reevaluation of the initial brain CT in one patient with lethal outcome in whom initial CT study was interpreted as normal. No improvement was noted after administration of anticoagulant treatment and the patient died 11 days after the initial CT scan. Application of MRI with susceptibility-weighted imaging or T2 gradient-echo sequence was the most important in detection of the cortical veins thrombosis.

Conclusion:

Education of both radiology and neurology residents in detecting both direct and indirect signs of cerebral venous thrombosis is extremely needed in order to decrease the rate of fatal outcome. Inclusion of susceptibility-weighted imaging or T2 gradient-echo sequence as obligate part of protocol, especially in patients with acute or subacute headaches may significantly improve the detection of thrombus within the cortical veins.

10: White Matter Hyperintensities in Patients with Sporadic Hemiplegic Migraine

*Oldendorf Award Recipient

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Background:

Migraine headache is known to be associated with white matter hyperintensities (WMHs) but the prevalence in patients with a sporadic hemiplegic migraine (SHM) is not well defined.

Aim:

To study the prevalence of WMHs in patients with SHM and compare with migraine headache.

Method:

We included patients who met diagnostic criteria proposed by third International Classification of Headache Disorders (ICHD3) for SHM and Migraine headache. WMHs are identified using T2 FLAIR axial sequence and classified based upon location, burden was assessed by Scheltens visual rating scale. Patients with migraine headache were used as a control for this study.

Results:

A total of eighty patients were recruited for our study period of which fifty patients met our inclusion criteria. We also included hundred patients who met the diagnostic criteria for migraine headaches as controls for this study. There was no significant difference in the age and sex between the two groups. Patients in the study group were similar to the control group (47.7 ± 12.2 years vs 48.17 ± 9.7 years; $p=814$) and the gender of patients in the study group (M: F; 14:36) and control (M: F 25; 75) were matched ($p=0.693$). The WMHs were found in 28 (56%) patients with SHM and 44 (44%) in migraine headache patients. However, there was no statistically significant ($p=0.166$) association between the two groups. The most common locations for WMHs in SHM patients are in the frontal lobe 24 (48%) followed by parietal lobe 18 (36%) patients. Similarly, in the migraine headache patients, the frontal lobe lesions were noted in 43 (40%) patients followed by parietal lobe 12 (12%). The distribution of lesions in different locations for both the study and control groups were summarized along with their p-value in table 1. On univariate analysis, WMHs are considerably seen in the parietal lobe, occipital lobe and infratentorial region in patients with SHM when compared to migraine headaches.

38 (90%) patients with migraine and 17 (60%) patients with SHM had lesions that are <5mm in diameter. 11 (40%) patients with SHM and 6 (10%) patients with migraine headache had lesions >5 mm in diameter. The diameter of the lesions is substantial in patients with SHM ($p=0.007$). According to the Scheltens visual rating scale, the mean score of WMHs in SHM was (2.9 ± 4.25) and (1.45 ± 2.6) in migraine patients. The severity of WMHs is intense ($P=0.001$) in patients with SHM. On multivariate logistic regression analysis, the WMH lesion in the parietal lobe ($p=0.043$) and visual analog score ($p=0.097$) were found to be significant and summarized in table 2

Conclusion:

The high prevalence of WMHs were noted in parietal lobe and severe white matter burden in patients with SHM when compared to those with migraine headache.

Table 1

Location of WMHs	SHM n(%)	Migraine headache n (%)	P value
Frontal	24(48%)	43(43%)	.561
Parietal	18(36%)	12(12%)	<.001
Temporal	4(8%)	5(5%)	.347
Occipital	11(22%)	5(5%)	.001
Infratentorial	10(20%)	6(6%)	.009
Basal Ganglia	8(16%)	9(9%)	.350

Table 2

Variables	Odds Ratio	95% CI	P value
Parietal	3.972	1.044-15.118	0.043
Occipital	3.173	0.677-14.865	0.143
Infratentorial	3.013	0.600-15.123	0.180
>5mm WMHs	0.182	.0.17-2.002	0.164
Visual analog rating scale	0.750	0.534-1.054	0.097

11: Rapidly Progressive Global Cerebral Atrophy in The Setting of Anti LGI1 Encephalitis

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Background:

Autoimmune encephalitis is a rare and potentially treatable disease with early intervention. Roughly, around 70% of the patients with autoimmune encephalitis have normal imaging findings, and the diagnosis is based on the presence of antibodies in the appropriate clinical setting. Hippocampal atrophy and hyperintensity of the limbic structures on T2 weighted MRI sequence have been reported in the past in patients with anti-LGI1 encephalitis. In this case report, we present a case of anti-LGI1 encephalitis that was associated with a rapid progression of global cerebral atrophy.

Case:

A 47-year-old male presented with complaints of breakthrough seizures along with psychiatric, behavioral changes, inability to speak, and catatonic features. Magnetic resonance imaging (MRI) of his brain showed mild cerebral and right hippocampal atrophy while the electroencephalogram (EEG) showed intermittent right temporal slowing. Serological and Cerebrospinal Fluid (CSF) testing was negative for infection. With a working diagnosis of autoimmune encephalitis, he was treated with a combination of immunoglobulin (IVIG) and methylprednisolone which significantly improved his cognition with complete resolution of his seizures. Paraneoplastic antibody testing in the serum later came positive for anti-leucine-rich glioma inactivated 1 (LGI1) voltage-gated potassium channel (VGKC) antibody. Two months after initial presentation, he had a relapse of his symptoms without any further episodes of seizures. Repeat MRI of the brain showed a significant rapidly progressive diffuse cortical atrophy and hippocampal atrophy on the right more than the left along with hydrocephalus ex-vacuo when compared with the previous MRI. He was treated again with immunomodulation therapy, which improved his symptoms at the time of discharge.

12: Central Nervous System Prostate Metastatic Disease: A case series

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Objective:

Report three cases of patients with prostate cancer and central nervous system involvement, and to highlight that although rare, metastatic disease must be included in the differential diagnosis to avoid delay in treatment.

Background:

Dural metastatic disease due to prostate cancer is a rare manifestation that occurs in approximately one to six percent of cases. Most patients with prostate cancer brain metastases present with increased intracranial pressure, headaches, seizures, cranial nerve palsies, and deteriorating level of consciousness. Median survival, from the time of diagnosis of CNS involvement, has been estimated at 3-4 months in large group studies.

Methods:

Case 1: 56-year-old male patient with metastatic prostate cancer with myelophthisis status post chemotherapy, radiotherapy, bilateral orchietomy, who presented with a right-sided trigeminal neuralgia, right-sided facial palsy, and right-sided sensorineural hearing loss. Brain MRI revealed multifocal metastatic disease to the dural surfaces, including the left parasellar, right cerebellopontine angle, and right Meckel's cave regions with possible compromise of the right fifth cranial nerve, right seventh cranial nerve, and left superior orbital fissure.

Case 2: 61-year-old male patient with metastatic prostate cancer who presented with speech and swallowing difficulties, and left sided facial paralysis. Brain MRI revealed diffuse supratentorial and infratentorial pachymeningeal thickening and enhancement with superimposed mass-like nodularities, asymmetric dural thickening and enhancement at the left porus acusticus extending into the left internal auditory canal, with continuous asymmetric thickening and enhancement seen at the left facial nerve; nodular extra-axial enhancing lesions were also seen along the bilateral trigeminal nerves.

Case 3: 67-year-old male patient with metastatic prostate-cancer s/p radical prostatectomy who presented initially with headaches and Brain MRI remarkable for left parietal metastasis treated with cyber knife. Over the next 10mo, headaches worsened and follow-up Brain MRI showed progression of disease with two new lesions in right frontal and parietal lobe, and worsening of edema of left parietal metastasis. Patient started on dexamethasone treatment followed by cyber knife. Follow-up evaluation in 4mo remarkable for development of dysarthria and right-side tongue paresis. Brain MRI showed stable right frontal lesion, decrease in size and edema of right parietal and left posterior parietal vertex lesion. New right skull base extra-axial lesion adjacent to cerebellum and medulla compressing lateral aspect of the medulla and insinuating through the foramen magnum and new right parietal high convexity lesion. Radiation oncologist evaluation was recommended for treatment with additional cyber knife and patient continued on dexamethasone.

Conclusions:

Central nervous system involvement from metastatic prostate cancer is rare. With these cases we emphasize the importance of including this entity in the differential diagnosis of patients with advanced prostate cancer and neurological symptoms to avoid delay in treatment, prevent further neurological decline, and maintain patients quality of life.

13: Comparison of Patients with Right to Left Shunts: TCD bubble test positive at rest versus test positive only with Valsalva

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Objective:

To evaluate if the proportion of patients with right to left shunt present only with Valsalva is significantly different between CVA and migraine. Objective was also to evaluate if activity at stroke symptom onset correlates with shunt absence at rest.

Methods:

We retrospectively evaluated TCD bubble studies in consecutive patients with cerebrovascular accidents and Migraine treated at the Mayo hospital in Phoenix, Arizona from August 2017 through August 2018. We performed a simple chi square test to examine difference of proportions between CVA and migraine with regard to shunt absence at rest (only seen with Valsalva). We evaluated if activity at symptom onset correlated with shunt presence or absence at rest.

Results:

A total of 47 patients underwent microbubble TCD studies, of which 26 were positive for right-to-left shunt. The indication for bubble study was stroke or TIA in 15 patients. The indication among the remainder was migraine or spell. Shunt was absent at rest and present only with Valsalva in 7 patients with stroke or TIA. Among those with migraine and positive shunt study, 7 had a shunt that was only seen with Valsalva. There was no difference between stroke/TIA and migraine with regard to shunt presence or absence at rest ($p=0.39$). Activity at symptom onset did not correlate with shunt presence/absence at rest.

Conclusion:

Presence or absence of shunt at rest was not significantly different between stroke/TIA and migraine. Activity at stroke onset had no correlation with absence of shunt at rest. Most patients developed stroke while awake.

14: Cerebrovascular Reactivity Transcranial Doppler as a Tool to Individualize Treatment of Focal Intracranial Stenosis

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Background:

The optimal treatment of patients with symptomatic intracranial stenosis who fail medical management is unclear. Per current AHA/ASA guidelines stenting with the Wingspan system for patients with stroke/TIA attributable to intracranial artery stenosis is not recommended.

Method:

We describe the management of a patient with TIAs due to focal intracranial stenosis treated with stenting after impaired cerebrovascular reactivity studies.

Results:

A 34-year-old man with hypertension and asthma developed episodes of sudden right hemiparesis and aphasia. Most spells occurred with a change of position or activity. MRI brain, echocardiogram and carotid ultrasound were normal. Aspirin and lisinopril led to longer attacks. Anticoagulation was started for elevated lupus anticoagulant without improvement. Keppra for possible seizures was ineffective. After one year of symptoms, evaluation at our institution revealed MRA, TCD and catheter angiography consistent with severe stenosis of the left MCA. Cerebrovascular reactivity TCD with breath-holding changed right MCA velocity (cm/sec) from 81 to 116 and left MCA from 78 to 82, consistent with impaired left MCA vasoreactivity (breath holding index <0.69). Angioplasty and stenting of the left M1-M2 MCA with the Wingspan system resulted in resolution of TIAs.

Conclusion:

Cerebrovascular reactivity studies with TCD are a potential tool to individualize risk-benefit analysis for intracranial stenting in patients with recurrent stroke/TIA due to focal intracranial stenosis refractory to medical therapy. Further research of the role of vasoreactivity studies and endovascular therapies for medically refractory intracranial stenosis is needed.

15: Transcranial Doppler Simulator for Hands-On Training and Assessment

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Introduction:

Transcranial Doppler (TCD) is underutilized despite having many clinical indications. The problem is that TCD is the most difficult diagnostic ultrasound procedure to learn.

Methods:

We developed a TCD simulator with a mannequin that the user can scan using a mock transducer. Each simulated case is built from a CT angiogram acquired from a patient. The intracerebral arteries are reconstructed in 3 dimensions. Computational flow modeling (CFM) is used to calculate time-varying blood flow velocity for all points within the lumen. The simulator displays power M-mode and spectral waveforms in real-time corresponding to transducer position on the mannequin and user-defined settings (sample volume depth and size, PRF or scale). To validate the accuracy of the simulation itself we measured peak systolic blood flow velocity from the spectral waveform rather than mean velocity. The measurements made on the simulator were compared with the true velocity computed by the CFM at the sample volume location. We defined the threshold for accuracy of the simulator as mean absolute error in velocity measurement less than 10%, the upper limit of acceptable clinical error. We also compared the appearance of the simulated waveforms at each segment with the patient's clinical TCD exam.

Results:

Measurements were made at 14 arterial segments in a case with pathology. The measured velocity underestimated true velocity with a mean absolute error of $11 \pm 10\%$ of the true value over all segments. Error for the middle cerebral, anterior cerebral, terminal internal carotid, ophthalmic and basilar arteries was significantly lower than for vertebral arteries ($8 \pm 9\%$, N=12, vs. $20 \pm 9\%$, N=4, p

Conclusions:

This is the first TCD simulator with hands-on scanning. These preliminary results validate the accuracy of the simulation for intracerebral arteries other than the vertebral arteries. With calculation of mean velocity and collection of cases representing a range of pathology, this TCD simulator will be a useful tool for training and for assessment of competence in an objective and quantitative manner.

16: RAPID™ Based Treatment Algorithms Lead to Faster Activation of Neurointervention Team and Reduce Recanalization Times

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Background:

Delay in recognition and treatment of acute ischemic stroke patients has been shown to worsen outcome. The recent use of an automated CT perfusion analysis tool (RAPID™) has been demonstrated to provide rapid and accurate prediction of core stroke volumes and therefore early recognition of large vessel acute ischemic stroke (AIS) patient's that can potentially benefit from endovascular treatment. We aimed to compare the activation time of our neurointervention team before and after implementing using the RAPID software at our institution

Methods:

We reviewed the IRB approved stroke registry of patients who presented to our community based, university affiliated comprehensive stroke center with ischemic strokes during January 2014-July 2018. Patients who underwent neuroendovascular interventions were included. Patients were divided into three groups based on which imaging modality was used by our stroke team in deciding to activate the neurointervention team. (Group A-CT ASPECT, Group B-CT Perfusion, Group C-RAPID™). Stroke team activation (Code Stroke) to neurointervention team activation (Code Neurointervention) mean times were analyzed and compared.

Results:

There were total of 2423 patients with ischemic strokes. Of those, 262 patients underwent neuroendovascular interventions. The three groups of patients were: Group A (n=232); Group B (n=22); and Group C (n=8). Mean times from Code Stroke to Code Neurointervention were Group A=77 minutes (SD=55, 95% CI= 69-84), Group B=78 minutes (SD=53, 95% CI=53-100), Group C=53 minutes (SD=40, 95% CI=25-80). These groups were statistically significant and there was a definitive suggestion of improved response times from assessment based on RAPID™ vs the other two modalities (CT ASPECT and CT Perfusion).

Conclusion:

Implementation of RAPID™ software has significantly reduced the time to activate the neurointervention team. Multifactorial time delays may have been reduced with ease of algorithmic decision node. Larger 'real-life' practical studies are required to corroborate our findings.

17: The impact of Chronic Kidney Disease Stage on Intracranial Vascular Resistance on Transcranial Doppler

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Introduction:

Individuals with chronic kidney disease (CKD) have an increased risk of stroke and manifest impaired cerebral autoregulation. CKD is associated with systemic inflammation and accelerated arteriosclerosis. Although there is a graded, independent relationship between estimated glomerular filtration rate (eGFR) and risk of stroke, little is known regarding the effects of CKD stage on cerebrovascular hemodynamics. Despite awareness of increased stroke risk in CKD, screening and stroke prevention in patients with CKD continues is generally similar to that in the non-CKD population. Transcranial Doppler (TCD) is a helpful tool to non-invasively measure indices of cerebral blood flow and intracranial vascular resistance by mean flow velocity (MFV) and pulsatility index (PI). This retrospective study assessed the effect of CKD stage on anterior and posterior circulation in acute post-stroke patients with CKD stages 1 through 5.

Methods:

After IRB approval was obtained, the electronic medical records of all acute stroke patients admitted to a single-center tertiary care facility in 2017 were reviewed. All patients with CKD who received TCD ultrasound to assess intracranial circulation within one month following stroke were identified. TCD data included mean flow velocities and PIs for the bilateral middle cerebral arteries (MCA), anterior cerebral arteries (ACA), posterior cerebral arteries (PCA) and vertebral arteries (VA). TCD MFVs and PIs were reported as the average of the bilateral values. Patients with significant unilateral intracranial stenosis were excluded. CKD stages were determined by stable serum creatinine and CKD-Epi eGFR equation over three months. Those with unstable creatinine or <3 months of lab data were excluded. Patient demographics, cerebrovascular disease risk factors, and stroke etiologies were collected.

Results:

The analysis included 28 patients, mean age 62.5 years, 57% male. 43% of strokes were large vessel etiology by TOAST criteria, and 46% of patients had multifocal intracranial atherosclerosis diagnosed by head CT-angiogram; CKD classes did not differ significantly in these metrics. Among patients with CKD stage 1 (CKD1; n=12), MCA MFV was 66.2 ± 24.5 with PI 0.81 ± 0.09 , ACA MFV 54.3 ± 17.6 with PI 0.82 ± 0.07 , PCA MFV 41.5 ± 17.3 with PI 0.83 ± 0.07 , and VA MFV 32.8 ± 11.1 with PI 0.96 ± 0.08 . Patients with CKD2 showed significantly decreased MFV in both anterior and posterior circulations in comparison to those with CKD1, as well as higher PIs. Among CKD2 patients (n=8), MCA MFV was 46.7 ± 12.7 with PI 1.15 ± 0.13 , ACA MFV 37.8 ± 11.8 with PI 1.21 ± 0.15 , PCA MFV 29.9 ± 9.1 with PI 1.21 ± 0.22 , and VA MFV 26.6 ± 7.4 with PI VA 1.17 ± 0.15 . These significant differences in MFV and PI (all p<0.05) persisted in CKD stages 3, 4 and 5, when compared to CKD1. CKD2 patients did not differ significantly from patients with CKD3-5 based on these TCD metrics.

Conclusion:

These data suggest mean flow velocities decrease and distal resistance indices are increased at early stages of CKD and these changes persist in patients with CKD3-5. Cerebral hemodynamics and their effect on stroke and white matter disease burden may be valuable identify CKD patients who are at high risk for stroke, as well as allow for modification of risk factors. Further research regarding TCD cerebral hemodynamics in CKD patients is needed.

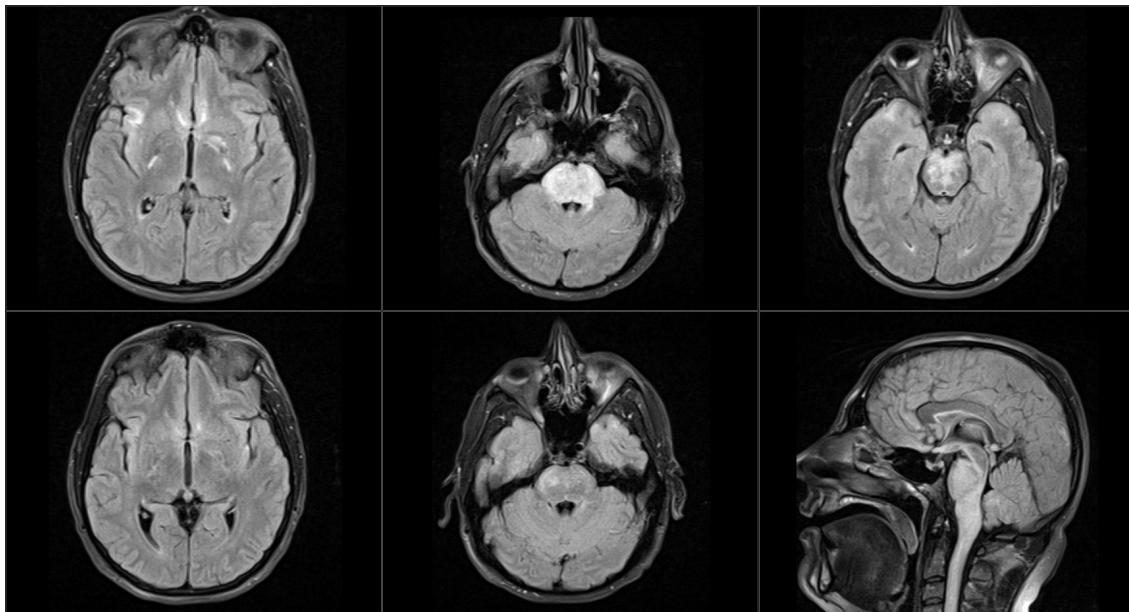
18: Posterior Reversible Encephalopathy Syndrome Affecting Entire Neuraxis

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Posterior reversible encephalopathy syndrome (PRES) is a clinical radiographic syndrome of heterogenous etiologies, including but not limited to hypertensive encephalopathies, autoimmune disorders and use of immunosuppressant and cytotoxic medications. PRES is usually associated with characteristic neuroimaging findings of cerebral white matter edema. PRES affecting the spinal cord has also been described in literature, especially pediatric population.^{[1][2][3]} However here we describe an unusual case of a young 28-year old male who suffered from PRES affecting his entire neuraxis.

28-year old male with past medical history of uncontrolled hypertension, CKD stage 4 secondary to autoimmune glomerulonephritis on mycophenolate admitted with new onset headaches, confusion and first time single generalized tonic clonic seizure in setting of hypertensive crisis with SBPs in 200s. Patient's headache and confusion had resolved shortly after admission with intensive BP control and neurological exam was only concerning for retinal edema on slit lamp and otherwise unremarkable exam. He underwent extensive testing upon admission, including neuroimaging. MRI brain without contrast revealed T2 prolongation with mass effect involving pons with extension into cerebellar peduncles, cerebellar nodulus, and white matter of the brain. There was also symmetric T2 prolongation involving bilateral gyrus rectus, anterior subinsular white matter and bilateral subcortical frontal lobe white matter. Also seen were bilateral retinal fluid collections with cupping of optic discs with fluid around nerve sheaths. MRI cervical, thoracic and lumbar spine without contrast revealed diffuse T2 hyperintensity and swelling of medulla, entire cervical spinal cord, multifocal T2 hyperintense signal consistent with edema throughout thoracic and lumbar spinal cord also affecting conus medullaris along with T6, T7, T11 and T12 compression fractures which the patient suffered presumably secondary to fall during seizure. Despite mismatch between clinical symptoms and radiological findings, patient underwent extensive testing for other conditions including ADEM, NMOSD and neurosarcoidosis. His CSF analysis was grossly normal except mild protein elevation and few RBCs due to traumatic tap. CSF infectious panel was also negative. His serum ACE, NMO/AQP4 and MOG antibodies were also negative with absent oligoclonal bands. There was no suspicion of transverse myelitis. Patient did not receive any immunomodulatory treatment including steroids given relatively asymptomatic clinical picture compared to the imaging except intensive blood pressure control. Patient underwent follow up neuroimaging one week later which revealed marked improvement of intracranial and spinal lesions and MRI obtained 2-weeks later revealed near-total resolution of spinal cord lesions. This experience expands the spectrum of neuroimaging findings of PRES and demonstrates lack of correlation between imaging findings and clinical involvement.



MRI Brain FLAIR (Presentation; top left and middle. 1-week later; bottom left and middle. Right top; retinal fluid collection. Right bottom; Sagittal FLAIR)



MRI spine (Top; cervical, thoracic and lumbar STIR on presentation. Bottom left; cervical T2. Bottom middle and right; thoracic and lumbar STIR 2-weeks later)

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19: Chronic Sinusitis Leading to Massive Spontaneous Pneumocephalus from an Ethmoid Sinus Encephalocele and Fistula

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Background & Purpose:

Spontaneous pneumocephalus is a rare finding in the absence of trauma or instrumentation. Spontaneous tension pneumocephalus is an even rarer and refers to pneumocephalus with associated brain compression. Here we describe a case of chronic sinusitis leading to massive spontaneous tension pneumocephalus.

Methods:

An 84-year-old with a past medical history significant for schizophrenia, hypothyroidism, degenerative joint disease, hypokalemia, DM-Type II, and chronic sinusitis presented on multiple occasions to the ED between Winter 2017 and Spring 2018 for various medical issues including hallucinations, hyperglycemia, and encephalopathy. CT head obtained in the ED on March 2018 did not reveal any acute cerebral process but did demonstrate sinusitis involving the right ethmoid, right sphenoid, and left maxillary sinuses as well as a retention cyst. In April 2018, the patient presented to the ED for persistent dizziness and CT head revealed persistent sinusitis. Due to her risk factors, the patient was admitted to the hospital for stroke work up for persistent dizziness. MRI brain revealed no acute intracranial process but persistent sinusitis. Ultimately she was discharged home. In May 2018, the patient returned to the ED complaining of right ear pain, nasal congestion and left sided headache. CT head demonstrated massive tension pneumocephalus attributed to a defect in the right sphenoid wing. Pt was admitted to the hospital and underwent MRI brain which also showed massive tension pneumocephalus with posterior displacement of the frontal lobes. The patient was treated for potential meningitis due to communicating fistula and her encephalopathy improved. ENT and Neurosurgery were consulted to repair the defect but were hesitant due to the patient's advanced age, non-focal neurological exam, and multiple medical comorbidities. Given the clinical improvement with antibiotics, decision was made for ENT to re-evaluate the patient as an outpatient. During the outpatient visit the patient revealed that she has had watery discharge out of the right nare for years. In retrospect, the patient likely had had CSF rhinorrhea which had gone unreported. Thereafter, she underwent a follow up CT of the sinuses and facial bones which again demonstrated the fistula but increasing hydrocephalus. The patient was sent for urgent endoscopic repair of the fistula with AlloDerm grafting. Post-op the patient's encephalopathy improved with time and she was discharged home. In July 2018, on follow up CT of the sinuses and facial bones, the patient's pneumocephalus had completely resolved.

Result:

Spontaneous tension pneumocephalus from chronic sinusitis is a very rare finding with only a handful of case reports in the literature. We believe that our patient's chronic untreated sinusitis led to a sphenoid sinus fistula allowing air to enter the skull causing massive tension pneumocephalus, and meningitis. With surgical repair of the fistula the pneumocephalus resolved with time.

Conclusions:

Communication between the atmosphere and the skull through a sinus fistula can cause massive pneumocephalus and place a patient at risk for meningitis. Ultimately, surgical intervention is required for closure of the fistula.

20: Cerebral Blood Flow Velocity Monitoring after Endovascular Treatment Tells the Story

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Background:

Endovascular treatment (EVT) is an effective therapy for acute ischemic stroke due to anterior circulation large artery occlusion (LAO). Yet, some patients do not improve in spite of successful recanalization, while others recover even if partially recanalized. Post-EVT cerebral hemodynamic parameters were recorded by transcranial ultrasound (TCCS) to explain this clinical variability.

Materials and Methods:

Serial TCCS examinations assessed the vessel status and cerebral hemodynamics of 185 [109 (58.9%) men, mean age 69.5±12.3 years] consecutive patients with acute anterior circulation LAO soon after, at 48 hours and one week after EVT.

Results:

Successful recanalization [OR 0.25 (0.11-0.61)], normal PSV Ratio (PSV of recanalized MCA/PSV of contralateral MCA) at 48 hours [OR 0.22 (0.15-0.64)] and after one week [OR 0.11 (0.07-0.31)] from EVT were independent predictors of good outcome at 3 months. Thrombectomy failure [OR 10.22 (1.47-45.53)] and pathological PSV Ratio at one week from EVT [OR 15.23 (4.54-46.72)] were associated with a worse 90-day outcome. Patients with intracranial hemorrhage (ICH) had higher mean PSV Ratio (3.5±0.2 vs 2.4±0.1, P<0.0001) soon after successful recanalization. In multivariate analysis, early PSV Ratio was independently associated with post-interventional ICH [OR 8.474 (95% CI 3.066-45,122), P<0.01]. At one week from EVT, 15/21 (71.4%) ICH patients who resumed normal PSV values had a better 90-day outcome (mRS 0-2 40% vs 0%).

Conclusions:

Post-EVT ultrasound monitoring of stroke patients might be an effective bedside method for assessing treatment efficacy, shedding light on outcome variability and identifying patients at increased risk of ICH.

22: Cerebral Angiolipoma of the Parietal Region Presenting as Olfactory Hallucinations

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Introduction:

Angiolipomas are benign tumors of mesenchymal origin typically found in the subcutaneous tissue of the extremities. CNS involvement is rare, with most cases found in the spinal extradural space¹⁻¹⁸. Intracranial angiolipomas are exceedingly rare and typically located in the sellar-parasellar region. We present a rare and unusual case of a cerebral angiolipoma located in the right parietal region.

Methods:

The patient is a 46-year-old female, BRCA+, with a 2 year history of olfactory hallucinations occurring for 1-2 minutes, with spells lasting in clusters for several hours. She also reported headaches related to her menstrual period as well as recent visual disturbances, which were difficult for her to describe. On examination, multiple raised areas were noted involving the skull bilaterally, the most prominent over the right parieto-occipital region. Her neurological exam was normal. Contrast enhanced brain MRI revealed a subtly enhancing extra-axial mass adjacent to the right parietotemporal calvarium, hyperintense in all sequences with punctate areas of hypointensity intermingled. The mass attenuated on fat saturation images with serpiginous intermingled signal suggesting vascular channels. A pituitary mass was also noted. EEG revealed slowing and epileptiform discharges from the right temporal region. She was placed on levetiracetam with resolution of her olfactory hallucinations. The patient underwent surgical excision of the parietal mass, and a year later, of the pituitary mass.

Results:

The histopathology revealed the lesion to be composed of mature lobulated adipose tissue with proliferations of connective tissue fibers and numerous thin-walled vessels. She remains seizure free and is off levetiracetam. Her BRCA+ status may be unrelated to the tumors noted here, but this status has been shown to have an association with gliomas¹⁹.

Conclusions:

Angiolipomas are rare in the brain parenchyma. We present a rare case of intracranial angiolipoma involving in the parietal lobe. Angiolipomas have a distinctive appearance on MRI, with fat suppression sequences playing an important role in the differential diagnosis.

23: An Interesting Case of Moya Moya in a 30-year-old with Review of Imaging and Recommendations for

Management

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Introduction:

Moya Moya Disease (MMD) is an uncommon cerebrovascular disease that is characterized by progressive stenosis of the terminal portion of the internal carotid artery and its main branches. This is condition that is primarily seen in Asian populations and African American populations - associated with (Sickle Cell Disease). This can lead to development of dilated and fragile collateral vessels at the base of the brain. Diagnosis is based on different neuroimaging modalities. We present a case of a right hemispheric stroke caused by Moyamoya identified on CT angiography and confirmed by cerebral angiography with review of imaging findings and treatment options.

Patients (or Materials) and Methods:

A 30-year-old Caucasian female presented as a medical transfer for intractable headaches and abnormal imaging. Her headaches had been ongoing despite conservative management for presumed diagnosis of migraine with multiple Emergency Department visits over the prior three weeks. As symptoms progressed, she developed associated dizziness, weakness and visual field loss which resulted in completion of a head CT. Imaging revealed extensive abnormalities in the right temporal, parietal and occipital lobes concerning for vascular insult. Her presentation was further complicated as she carried a known diagnosis of hypercoagulability (not on anti-coagulation) with prior history of multiple prior miscarriages. She underwent multiple imaging evaluations to further clarify diagnosis including CT Angiography, CT Venography, MRI Brain and eventual Conventional angiography which resulted in a diagnosis of Moyamoya disease. She did have clinical improvement when treated with hydration, Aspirin and statin therapy and has been scheduled for definitive management (intracranial-extracranial bypass surgery).

Results:

An MRI of the brain revealed a large area of restricted diffusion involving the right occipital, temporal and parietal lobe consistent with a large acute infarct with mild mass effect including slight uncal herniation and right-to-left shift. As findings cross multiple vascular territories; CT Venogram / CT Angiogram were completed. Venogram did not demonstrate any venous occlusion or vascular malformation. CT Angiogram demonstrated slow vascular flow throughout the right hemisphere and to a lesser extent the left with prominent proximal arterial structures bilaterally. No aneurysm or hemorrhagic findings were noted. Findings were felt to be consistent with Moyamoya. Conventional angiography was completed and demonstrated bilateral occlusion of the supraclinoid ICA with Moyamoya vessels seen bilaterally. Bilateral superficial temporal arteries were visualized with robust flow compatible with candidacy for intracranial-extracranial bypass surgery.

Conclusion:

Moya Moya Disease is a rare condition. This case provides an excellent example of classical imaging findings in the setting of a typical clinical history in a patient whom does not have a genetic predisposition for this disorder. By understanding the associated imaging findings this patient was diagnosed in a short period of time, is doing well and is scheduled for definitive therapy with surgical bypass.

24: Feasibility and Utility of Early Continuous Transcranial Doppler in Traumatic Brain Injury admitted to Critical Care Unit

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Introduction:

Cerebral hemodynamic changes after sustaining Traumatic brain injury (TBI) are a major factor responsible for morbidity and mortality in this disease population. A plethora of research is available on continuous monitoring of the brain's hemodynamic response following such injury using invasive modalities like brain tissue oxygenation, regional blood flow, microdialysis and electroencephalography. There is emerging interest in non-invasive monitoring of cerebral hemodynamics in guiding clinical paradigms in TBI. Transcranial Doppler ultrasound (TCD) is a non-invasive, portable modality to study the large intracranial vessels, and measure cerebral blood flow velocities. Numerous hemodynamic indices can be derived from TCD waveforms, which can serve as indicators of cerebral events, including vasospasm, hypoperfusion, hyperperfusion, vasoreactivity, and autoregulation. TCD can also be used to elucidate phenomenon such as the triphasic hemodynamic response with continuous monitoring after TBI. This ongoing study focuses on the feasibility and utility of TCD to evaluate changes in cerebral hemodynamic patterns following TBI.

Methods:

This IRB-approved study is enrolling adult patients \geq 18 years old admitted to the Neuro-Sciences Intensive Care Unit (ICU) or Trauma ICU at a tertiary level academic medical center with a diagnosis of traumatic brain injury. Diagnosis is confirmed with historical and radiographic evidence of head injury including, but not limited to traumatic subarachnoid hemorrhage, intra-parenchymal contusion, intraventricular hemorrhage, diffuse axonal injury, or epidural hematoma. Eligible patients need to have a leveled trauma code (1-3), defined by ED admission and trauma team evaluation. Patients are excluded for pregnancy, incarceration, or penetrating skull trauma. Continuous TCD recordings are obtained from the bilateral MCAs using a monitoring headband from the bilateral MCAs for 15 minutes to 1-hour duration with a max of 4 hours of monitoring. Informed consent is obtained from the patient or surrogates prior to use of any of the collected data for research. Physiological parameters, including blood pressure, intracranial pressure, end-tidal carbon dioxide, and arterial oxygen saturation are recorded during TCD monitoring.

Results:

In this ongoing study, between 06/01/2018 and 09/15/2018, 400 patients were screened for study enrollment. 38 patients screened positive for TBI, with 13 found eligible for inclusion. Of these eligible patients, 9 expired prior to TCD scanning or declined consent. Data was able to be analyzed from 3 of the remaining 4 patients. Descriptive parameters of this sample ($n=3$) included mean age 62 yrs. and mean GCS score 13.6 on admission. All patients had neuroradiographically confirmed SAH with 1 patient with Diffuse Axonal Injury. All met criteria for mild TBI. Mean values for physiological parameters included mean arterial pressure 98 mmHg, systolic BP 148.33 mmHg, diastolic BP 74.67 mmHg, hemoglobin 7.77 g/dl, pulse 90 bpm during TCD. An average of 30 minutes of TCD recording was obtained on each patient. The TCD monitoring was well tolerated with no adverse effects. Mean duration of time between admission and TCD study was 5.33 days. Mean length of ICU stay was 13 days. MCA peak flow was aggregated for all patients with a mean flow of 82.4 and 51.55 for right and left recordings, respectively.

Conclusion:

At this time, no abnormal results have been observed. Our ongoing study is limited by the small sample size reported. While no anomalous flow patterns have been recorded, we have shown that monitoring cerebral hemodynamics with continuous TCD is feasible in ICU patients. This ongoing study provided a practical assessment of the feasibility of continuous TCD and allowed for the creation of a workflow to continue to pursue recording the triphasic response in moderate to severe TBI patients.

25: Case Series: Duplex ultrasonography adds incremental value to the evaluation of acute ischemic stroke despite elucidated pathology on CT angiogram

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Introduction:

Stroke is the fourth leading cause of death in the US. Approximately 1 of 5 ischemic strokes originates from atherosclerotic plaque and stenosis in the carotid arteries; isolated cases may have atheroembolic or thromboembolic phenomena in the absence of stenosis. Current guidelines for evaluation of known or suspected carotid stenosis recommend duplex ultrasonography as an initial test for both symptomatic and asymptomatic disease. With increasing emphasis on identifying large vessel occlusion for revascularization, many patients receive two vascular imaging modalities early in the course of their stroke workup: CT-angiogram (CTA) and digital subtraction angiogram (DSA). The utility of adding duplex ultrasonography as a third imaging modality for stroke workup in these patients is often questioned. The latest AHA/ASA stroke guidelines do not address the indications for duplex ultrasound in the early evaluation of acute ischemic stroke, nor do imaging guidelines. We present four descriptive cases where duplex ultrasonography added value in elucidating the etiology of stroke and led to change in management despite having results of CTA or DSA available.

Case 1. 74-year-old male with multiple comorbidities presented with right hemibody weakness. Initial stroke workup revealed left hemispheric infarcts in MCA/PCA watershed areas. CTA showed severe stenosis of the proximal left ICA resulting in near occlusion (no degree of stenosis specified but appeared to be 99% for that segment). Tissue plasminogen activator (tPA) was not administered due to presentation outside the therapeutic window. Clinical worsening prompted evaluation for acute carotid stenting, but cerebral catheter angiography demonstrated less significant left ICA stenosis with a luminal diameter of 2.4 mm (normal distal vessel diameter 4.00 mm, 40% stenosis) with an intraluminal thrombus just distal to the stenosis measuring 9 mm x 5 mm. Carotid duplex ultrasonography (CDU) done on next day confirmed the degree of stenosis for which criteria did not warrant surgical or endovascular intervention and the presence of large plaque in the left ICA and bifurcation prompting treatment with therapeutic anticoagulation. The patient was discharged to acute rehab. Earlier ultrasound could have avoided an invasive procedure and guided earlier initiation of management.

Case 2. 80-year-old male with hypertension and history of seizures presented with acute aphasia and received IV tPA. His initial stroke workup included CTA that showed calcified and noncalcified atheroma at the left common carotid bifurcation resulting in mild narrowing of the left internal carotid artery origin, with a low-density filling defect and patency of the anterior and middle cerebral arteries. Carotid Ultrasound showed no stenotic flow in either carotid system but revealed a complex plaque with low-level echoes suggesting intraluminal thrombus projecting into the lumen in the left carotid bifurcation/ICA origin. Embolus detection monitoring did not detect high-intensity transient signals (HITS). The patient was started on systemic heparin given concerns for a thrombus. Follow up MRI later that night revealed multiple acute infarcts in the left frontal, parietal, and occipital lobes in a distribution suggestive of watershed ischemia and/or embolic infarcts. Repeat carotid ultrasound showed persistent thrombus with protrusion into the lumen. Given these findings, the patient was continued on systemic heparin, which was later converted to therapeutic enoxaparin before discharging the patient home.

Case 3. 71-year-old male with atrial fibrillation (on therapeutic anticoagulation) and prostate cancer was transferred from an outside hospital with acute left hemibody weakness and sensory abnormalities. CTA showed right ICA occlusion with a distal M2 cutoff. TPA was not administered due to presentation outside the therapeutic window. MRI showed acute versus subacute infarct of the posterior right insula, temporal operculum, and frontoparietal lobe. Given CTA showing right ICA occlusion, the patient was not deemed an endovascular candidate. Follow up carotid ultrasound suggested that the right ICA was open, albeit with absent diastolic velocities suggesting either very severe stenosis with forward flow only during systole, or more distal stenosis or occlusion beyond the field of view. Given these findings, a diagnostic cerebral angiogram was performed which showed 99% right ICA stenosis just distal to the origin with a string sign. The patient reported transient vision loss that resolved within 5 minutes the next day. Given these new symptoms and the possibility of asymptomatic carotid stenosis, an urgent right carotid endarterectomy (CEA) was performed. He was ultimately discharged to a low-intensity rehab facility on warfarin, statin, and antiplatelet therapy.

Case 4. 81-year-old male with gastroesophageal reflux disease and benign prostate hyperplasia presented with acute left hemibody weakness. Initial CTA head/neck showed complete occlusion of the right ICA with possible occlusion of the distal right M2 segment. MRI demonstrated scattered right MCA territory infarcts. Given initial CTA showing complete occlusion, the patient was not deemed an endovascular candidate. Follow up carotid ultrasound showed a string sign in the right proximal ICA, consistent with 70-99% stenosis with a residual lumen of 1.1 mm and spontaneous echo contrast distal to the stenosis with a possible mobile plaque at the distal edge of the string sign on B-mode imaging. Based on these findings, the patient underwent a right CEA which was well-tolerated and without complication. The patient was discharged home on antiplatelet and statin therapy.

Discussion:

We present four descriptive cases where carotid ultrasound added complementary and extra information affecting clinical management despite patient receiving CTA as part of initial acute stroke evaluation. In two cases, ultrasound showed the absence of hemodynamically significant stenosis and presence of an active thromboembolic source amenable to anticoagulation. In two cases, Carotid ultrasound provided reasonable evidence for a lesion amenable to acute surgical intervention. Carotid Ultrasound has been shown to have comparable effectiveness to CTA and DSA in the diagnosis of carotid stenosis, with a sensitivity of 81-98%, specificity of 82-89%, and positive predictive value of 70-90%. However, studies of the overall incremental value of ultrasound in stroke etiologies irrespective of stenosis are limited. Future prospective studies are warranted addressing the cost-effectiveness of carotid ultrasound as well its differential impact on management after patients have undergone vascular imaging like CTA or DSA.

Reference

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26: Sonographic Demonstration of a Perfusion Dependent Stroke with MRI Negative Flow Limiting Stenosis

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Introduction:

Perfusion dependence of stroke is a phenomenon often investigated in an acute ischemic stroke caused by large vessel occlusion while the patient is being evaluated for endovascular interventions. We present a case of a patient who had flow limiting lesions causing neurological symptoms with a negative MRI with symptomatic improvement after permissive hypertension. We captured this change in cerebral perfusion with transcranial dopplers when systemic perfusion was augmented.

Methods:

Case report and neuroimaging review

Results:

A 67-year-old male presented with acute stroke-like syndrome with left hemiparesis and NIHSS score of 8 at 2330 hours. Initial BP was 200-220 mmHg systolic. TPA was not given due to presentation outside of clinically indicated time window. CT angiogram/perfusion done at admission revealed complete occlusion of the right internal carotid artery from bifurcation to cavernous portion with increased mean transit time in the right cerebral hemisphere, and slightly increased peripheral blood volume and decreased blood flow. This was attributed to right ICA occlusion and compensatory vasodilation, in addition to multifocal intracranial vessel stenosis including the basilar artery, right V4, left P2, right M1, and left cavernous carotid. Given large vessel occlusion, a cerebral angiogram was done at 0030 hours, showing occlusion of the R ICA distal to the origin, occlusion of the cavernous RICA with reconstitution from the external carotid through the ophthalmic, and non-filling of the ACA. Mechanical thrombectomy was performed, and TICI 3 achieved in the R ICA. Post-procedural NIHSS score was 8 at 0130 hours. Symptoms of right MCA stroke with persistent left hemiparesis right gaze preference and left neglect continued despite complete R ICA recanalization. Nicardipine infusion was initiated given successful recanalization to target SBP <160. MRI done at 0512 hours during the symptomatic phase showed no restricted diffusion despite patient being hemiparetic with gaze preference. The systolic BP remained mostly in the 150s with isolated values in 130s and 160s. During morning rounds, concern was discussed for persisting symptoms despite negative MRI and recanalization. Bedside point of care transcranial Doppler ultrasonography (TCD) showed relative hypoperfusion of right MCA compared to left MCA. This was confirmed by a full diagnostic study done emergently at 1031 am. Right MCA M1 and ACA mean velocities were below normal (35 and 24 cm/s, respectively), and were asymmetrical compared to the left MCA M1 and ACA (81, and 53 cm/s, respectively). There was 50-69% stenosis by velocity criteria in the bilateral CCAs, worse on the right than the left. The BP in the left arm was 142/65 mmHg with a pulse of 77 bpm at the time of TCD. The BP was augmented by stopping the nicardipine infusion and allowing permissive hypertension. Systolic BP (SBP) rose to 160-170 mmHg, without much clinical improvement. When SBP reached the 180s-200s there was a rapid improvement of symptoms with return to a near normal state. Follow up TCD at 1511 hrs at that point showed improvement in R MCA and ACA velocity to 55 and 38 cm/s respectively. Right arm cuff BP was 171/65 mmHg at the time of follow up TCD. Repeat NIHSS at 1900 hours was 2 due to mild right leg weakness and sensory loss, but there were no persisting symptoms the next day. Oral antihypertensive medication was started and titrated to a target BP of 160-180 mmHg over the next few days. Other stroke workup revealed an LDL of 126. TTE was unremarkable with EF of >70%, mild concentric left ventricular hypertrophy, and no evidence of PFO or atrial enlargement. The patient was discharged to home on hospital day 3 with an NIHSS score of 0, with 24-hour supervision and assistance. He was discharged on oral antihypertensive, statin, and antithrombotic medication.

Discussion:

This patient demonstrated sonographic evidence of a perfusion dependent stroke that responded to systemic perfusion augmentation. Perfusion dependent ischemia in the absence of MRI evidence of infarct poses several management challenges, for which ultrasound may provide important data to help identify the problem and inform treatment. This patient had multiple intracranial lesions with an engagement of collaterals to maintain cerebral perfusion and probable loss of autoregulation on the right side of the brain from chronic ICA stenosis that acutely occluded. CTP showed localizing perfusion deficit in the R MCA and ACA territory suggested the R carotid as the culprit lesion. Despite

recanalization, collaterals that depended on systemic hypertension in a non-autoregulated side of the brain were not adequate to maintain cerebral perfusion. Despite recanalization, the patient had persistent symptoms. Systemic augmentation restored cerebral perfusion and helped in the resolution of stroke symptoms and this was captured on the change in hemodynamics of spectral waveforms on distal MCA insonation.

There have been multiple trials with varying results of the effect of BP modulation of acute ischemic stroke but none of these trials correlated markers of cerebral perfusion in assessing immediate effects of systemic BP on cerebral perfusion or identifying subgroups of patients with flow-limiting lesions that may potentially show a difference clinical response to blood pressure augmentation than other stroke etiologies. Future studies assessing target BP in large vessel strokes should employ the use of noninvasive strategies like TCD in an acute evaluation, management and follow up of stroke.

Reference:

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4828558>

	Right Velocities on TCD	Left velocities on TCDs	Posterior circulation
ASYMPTOMATIC	RIGHT BP range MCA: 62/55/1.0. ACA: 68/38/1.0. Best neuro exam Terminal ICA: 64/51/1.0. OA: FORWARD 43/19/1.0. IC Siphon: 58/16/1.2. PCA: 68/17/.96. VA: 58/49/1.3.	LEFT MCA: 64/81/1.2. ACA: 72/53/1.3. Terminal ICA: 64/21/1.5. OA: FORWARD 4/18/1.3. IC Siphon: Attempted. PCA: 62/51/1.0. VA: 62/12/1.2.	BASILAR BA: 95/18/1.5.
SYMPTOMATIC	RIGHT BP range MCA: 62/35/1.2. ACA: 73/24/1.3. Terminal ICA: 66/61/.90. OA: FORWARD 40/25/1.8. IC Siphon: 62/16/1.3. PCA: 66/17/.98. VA: 65/21/1.2.	LEFT MCA: 62/77/1.3. ACA: 70/66/.97. Terminal ICA: 67/19/1.2. OA: FORWARD 34/20/.98. IC Siphon: 61/22/1.3. PCA: 58/24/1.1. VA: 66/12/1.2.	BASILAR BA: 93/25/1.2.
	Best neuro exam		

27: Case Report: B mode ultrasound images of the thoracic spine

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Introduction:

Ultrasound provides the clinician an additional decision-making tool when used at the point-of-care. In the hands of a skilled user, most bodily structures can be visualized distinctly with minimal risk to the patient. Ultrasound is used in the operative room to visualize neuro-anatomy to help in operative-decision making when image guidance is not available or feasible. We present a unique case showing the ultrasound appearance of the spine captured by imaging the spinal cord of a post thoracic laminectomy patient.

Case:

84-year-old male with extensive medical history including previous T10-S1 posterior spinal fusion, coronary artery disease with stents, heart failure with ejection fraction of 40%, chronic kidney disease, hypertension, bipolar disorder, acoustic neuroma status post resection, and hypothyroidism presented with one week of worsening back pain with symptoms suggestive of claudication. CT in ED revealed previously known T9 and T10 compression fractures as well as left lower lobe changes suspicious for a pulmonary lesion. Labs on admission were unremarkable except for leukocytosis to $17 \times 10^3/\mu\text{L}$ and elevated c-reactive protein to 257.3 MG/L. Neurosurgery was consulted with the recommendation of T8 vertebroplasty and T9 kyphoplasty and thoracic and lumbar MRI. Cardiology and rheumatology were consulted for perioperative recommendations regarding management of dual antiplatelet therapy and chronic steroids use, respectively. On hospital day #3, blood cultures returned positive for *Staphylococcus Aureus*. Operative plan was postponed, infectious disease was consulted for bacteremia recommendations, and the patient was started on broad-spectrum antibiotics. On hospital day #3, MRI of thoracic which was concerning for discitis osteomyelitis at T9-T10 with paraspinous and epidural abscesses and pathologic fractures of T9 and T10, with advanced canal stenosis and cord compression resulting in cord edema. Additionally, there was a T2 hyperintense fluid collection along the dorsal margin of the thecal sac at L3-L5 of undeterminable origin. On hospital day #4, patient experienced worsening symptoms of acute bilateral lower extremity weakness. The patient was emergently taken to the OR for posterior spinal fusion from T5 to T12 using bilateral segmental, and complete laminectomy and facetectomy bilaterally and evacuation of epidural abscess at T9-10. His postoperative course was complicated by pericarditis and acute lower extremity DVT. The patient was deemed medically stable and discharged to subacute rehab for recovery on post-operative day #16 on colchicine, anticoagulation, and dual antiplatelet therapy.

Discussion:

During the patient's postoperative course, we were allowed the unique opportunity to perform serial spinal ultrasound. US revealed distinct, unobstructed views of the spinal cord due to the removal of the vertebral lamina. We present these unique images as proof of the further use of point-of-care ultrasound as a tool to examine the epidural space for any residual fluid collections postoperatively and minimize the need for imaging with radiation risks to the patient.

28: Management of Tandem Occlusion: Novel approach

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The patient is a 59-year-old Hispanic right-handed male with PMH of HTN, DM2, who presented with right sided weakness, right facial droop, and slurred speech. The patient was last known well 26 hours prior. His NIHSS was 17. A CTA head and neck demonstrated a left ICA occlusion from the level of the bifurcation through the carotid terminus, involving the proximal MCA. CT Head showed no evidence of acute infarction, but remote left inferior temporal lobe ischemic stroke. The patient was outside of the window for IV tPA and he was taken for an emergent thrombectomy.

Digital subtraction angiography revealed an occluded left ICA after the bulb. An 035 glide wire (Terumo) and Neuron Select catheter (Penumbra) was navigated across the occluded carotid bulb but the Neuron MAX (Penumbra) would not cross into the ICA. The Neuron Select was exchanged for an ACE68 catheter (Penumbra). Then a mechanical thrombectomy was performed of the intracranial ICA and proximal MCA using a Solitaire 6 x 40 mm stent retriever (Medtronic). After 3 passes, we achieved a mTICI 2b reperfusion.

We then turned our attention to the extracranial ICA. The patient was given a loading dose of aspirin PR and an IV bolus of heparin. We attempted a balloon angioplasty of the carotid bulb occlusion. Although this resulted in temporary improved flow, the vessel re-occluded 5 minutes later. We then performed a diagnostic angiogram of the right CCA and left VA and found that the patient had robust collateral supply to the left MCA territory via the Circle of Willis. Therefore, the decision was made to not pursue extracranial left ICA revascularization and terminate the procedure.

Postoperatively the patient improved. MRI demonstrated minimal stroke burden in the left hemisphere. The patient was discharge to acute rehab with an NIHSS of 6. He was recently seen in follow-up and had an NIHSS of 2 and mRS of 1.

Acute tandem occlusions are of clinical interest because they are challenging to treat and are generally associated with poor outcomes. Much conversation revolves around whether to treat the intracranial occlusion or extracranial occlusion first. Additionally, there is also debate as to the superiority of angioplasty alone or angioplasty plus stenting. The latest evidence suggests that both approaches are equal. Our case is an excellent example of an additional option, which is leaving the extracranial ICA occluded as long as the patient has a robust Circle of Willis. This approach reduces the complexity of the procedure and may limit complications as patients will not be committed to dual antiplatelet therapy.

29: Ancillary Diagnostic Protocol of Brain Death using Transcranial Color and Power Doppler Duplex Imaging in Infants

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Introduction:

Brain death is accepted as an objective indicator of end of life with irreversible loss of all brain functions. In Its consensus statement, the American Academy of Neurology stated that single-gated transcranial Doppler (TCD) without imaging has limitations which make it unsuitable as an ancillary study. We evaluated the suitability of transcranial color and power Doppler Duplex imaging in Infants.

Methods:

Patients with hypothermia (35°C or less), intoxication, neuromuscular blockade, severe metabolic disturbance and hypotension were excluded. Each patient was imaged through the temporal and anterior fontanelle using power or color Doppler imaging with to purpose of optimization of the acoustic window. Attempt was made to identify all intracranial arteries in anterior and posterior circulation. Once identified arterial spectral pattern was evaluated. Brain circulatory arrest was declared if brief reverberating systolic flow signal lasting for a brief portion of the cardiac cycle was found and/or alternating flow (anterograde in systole and retrograde in diastole) with mean flow velocities below zero was seen. In addition, special attention was paid to venous flow. All studies were performed for a total duration of 30 minutes

Results:

Five infants less than 12 months of age (mean age 110 days) were evaluated over the last 5 years. Acoustic window was adequate in all patients. Venous flow was seen in two infants: both infants had adequate cerebral perfusion and did not fulfill diagnostic criteria of brain death; one of the two infants was found to have a Duret hemorrhage and was made DNR. The other three infants had a brief venous "blip". Color Doppler showed-rapid red blue arterial signal in proximal main arteries with loss of cerebral perfusion. Power Doppler showed a similar brief pulsatile pattern of flow in early systole and loss of tissue perfusion. They fulfilled diagnostic criteria of brain death.

Conclusion:

Power and color Doppler Duplex Imaging appears suitable as an ancillary study in diagnosing brain death. In the presence of flow, a poor outcome can still be predicted in selected cases.

30: Ultrasonographic Features of Closed Lip Schizencephaly in a Term Neonate

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Introduction:

Although intraoperative ultrasound using high resolution probe has gained in popularity in recognizing cortical dysplasia, bedside ultrasonographic diagnosis of focal dysplasia in neonates is rarely reported. We report our experience in a rare case of unilateral closed lip schizencephaly.

Method:

Bedside cranial ultrasound is obtained through the anterior, posterior and temporal windows in a term infant who was found to have a 2 vessels cord on prenatal ultrasound. Brain MRI is performed to confirm ultrasound findings

Results:

In the coronal plane in the frontal region, there is septal dysplasia and a focal squared appearance of the lateral edge of the left lateral ventricle leads us to recognize at the same level an area of hypoechoic gray matter penetrating the white matter laterally. In the sagittal planes, at the same level, hypoechoic gray matter is bulging into the left lateral ventricle with in its center a curvilinear string of variable echogenicity. More laterally the curvilinear string progressively becomes longer disrupting the normal morphology of the insular triangle and surrounding it the hypoechoic gray matter progressively becomes thicker. No vascular anomaly is detected on power Doppler. The brain MRI confirms diagnosis of septal dysplasia and focal cortical dysplasia with heterotopia and closed lip schizencephaly.

Conclusion:

Neurosonographic diagnosis of focal cortical dysplasia is rarely reported. We hope that this paper will increase the awareness of the neurosonographic pathognomonic features of closed lip schizencephaly.

33: Pure Cortical Ischemia-like Posterior Reversible Encephalopathy Induced by Hyperglycemia Hyperosmolar State: A case report

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Posterior reversible encephalopathy syndrome (PRES) is caused by many medical conditions, most commonly hypertensive crisis, renal failure, eclampsia, immunosuppressive and cytotoxic agents. PRES can present with headache, vomiting, altered mental status, cortical blindness, and seizures. Here, we describe a case of hyperglycemic hyperosmolar state (HHS) as the primary culprit for reversible cytotoxic edema. A 49-year-old male was found on the ground in his apartment and brought to the emergency department (ED). Past medical history included hypertension, uncontrolled diabetes mellitus type 2, hyperlipidemia, but also polysubstance abuse and seizures. In the ED, the patient had a very decreased level of consciousness, an extreme serum glucose of 1425 mg/dL, but an anion gap of only 17 mEq/L, and beta-hydroxybutyrate of 1.28 mMol/L. Patient was diagnosed with HHS and treated with insulin drip and isotonic fluids. Initial non-contrast computerized tomodensitometry (NCCT) of the head showed a small area of subcortical edema in the left occipital lobe in absence of any significant elevation of his blood pressure. His mental status rapidly recovered over the course of 6 hours in the ICU. At the next neurological evaluation, the patient had left temporal homonymous hemianopsia in the right eye but no other focal neurological deficit. MRI of the brain, 12 hours after the initial presentation, revealed a pauci-centimetric laminar purely cortical left calcarine lesion accompanied by a smaller right high parietal lesion, both linear and only hyperintense on diffusion-weighted imaging (DWI) with corresponding hypointensities in the apparent diffusion coefficient (ADC) maps. There was no evidence of vasospasm, arterial thrombosis, hemorrhage or necrosis on susceptibility-weighted imaging (SWI), and absolutely no extravascular edema on fluid-attenuation inversion recovery (FLAIR), notably in the adjacent subcortical white matter originally described as edematous on NCCT. EEG ruled out ongoing or recent seizures. Patient glucose levels were controlled and the patient was transferred to the regular floor. Stroke and seizures were ruled out as explanations for the ischemia-like transient lesions in the rostral aspect of the brain. This clinical case demonstrates that PRES can be elicited by diverse causes that have in common a susceptibility to the posterior circulation to dysregulation of the cerebral blood flow. PRES pathophysiology may lead to cerebral edema and increased oxidative stress, with greater vulnerability in the grey matter, as opposed to the classic PRES presentation with edema alone, vasogenic in nature and white matter predilection.

34: Unilateral Isolated Hypoglossal Nerve Palsy: A rare case due to jugular foramen dural arteriovenous fistula

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Introduction:

Isolated hypoglossal nerve (CN12) palsy is uncommon among cranial nerve palsies, usually resulting from postoperative trauma, idiopathy or malignancy, and less commonly due to vascular pathology. Hypoglossal canal-dural arteriovenous fistulas (HC-dAVF) are considered rare, making up 4.2% of all cranial dAVF patients. We present an unusual case of a patient who suffered a jugular foramen dAVF extending to the HC with concurrent transverse sinus thrombosis.

Case Report:

A 69-year-old woman with a past medical history of hypertension, hyperlipidemia, basal cell carcinomas of the skin s/p excision and lumbar disc herniation with vertebral compression fractures of the spine, who was referred to the neurology clinic for progressive worsening of tongue movements and inability to whistle following a dental surgery one year earlier. Her symptoms started after a complex dental procedure performed for a left molar tooth extraction after which in the PACU, she endorsed severe left jaw pain and neck pain. She reported worsening tongue weakness which persisted after the pain subsided a few weeks later and described a high-pitched pulsatile tinnitus in her left ear. She also developed left-sided tongue atrophy in the months following the procedure. Her cranial nerve examination was notable for left tongue scalloping, with occasional fasciculations, prominent deviation to the left upon protrusion, and noticeable weakness on the left side compared to the right. There was also an audible bruit heard more prominent on the left and loudest at the left angle of the mandible. CTA of the head and neck was ordered and revealed arterial flow within the venous structures around the jugular foramen and sigmoid sinus with invasion of the hypoglossal canal, and a chronic thrombosis of the left jugular bulb. MRI confirmed compression of the hypoglossal nerve within the hypoglossal canal from collaterals of a dAVF at the left skull base. Months later, a diagnostic catheter angiogram evaluation demonstrated a Borden Type 1 left sigmoid sinus dAVF, with thrombosis of the sigmoid sinus proximal to the venous pouch. No evidence of cortical venous reflux was appreciated. No surgical treatment was offered; instead, medical management with anticoagulation treatment led to partial recanalization of the dAVF, followed by rethrombosis in the hypoglossal canal and complete occlusion of the anterior condylar vein with obstructed arterial flow within the fistula, shrinking the fistula within the HC and leading to spontaneous resolution of the fistula.

Discussion:

Interestingly, the presentation of this patient's jugular foramen dAVF leading to only isolated CN12 palsy may be due to the lateral presentation of the jugular foramen dAVF, thus only affecting CN12.

Conclusion:

It is important to recognize dAVF as a rare cause of a progressive unilateral cranial mononeuropathy. This case is a unique example of jugular foramen dAVF leading to an isolated CN12 palsy. Prompt recognition of neuroanatomical features may lead to faster and more accurate diagnostic evaluation of dAVF subtypes, which may require individually tailored therapeutic management.

Late Breaking Abstracts

37: Intravenous Alteplase Safety in Stroke Patients with Early Ischemic Changes on CT and Prior Use of Antiplatelet Agents: Getting out of the ENCHANTED Forest

*Resident Travel Award Recipient

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Background and Purpose:

Intravenous alteplase (tPA) has been the standard of care for acute ischemic stroke (AIS) since approval in 1996, but its safety in patients receiving dual antiplatelet therapy (DAPT) has been debated. The ENCHANTED trial showed an increase in any intracranial hemorrhage (ICH) and symptomatic (sICH) complications in patients on self-reported DAPT prior to tPA. We sought to evaluate the rate of ICH and sICH in patients that were on DAPT and received tPA at our center with or without early ischemic changes on pre-tPA CT scan.

Methods:

A retrospective analysis of 1288 stroke patients receiving IV tPA from 2015 – 2018 was conducted. Patients were blindly evaluated using the ASPECT score and were assigned to one of two groups: ASPECT score 10 or ASPECT score of 9 or less. Patients within each group were assessed for use of DAPT, single anti-platelet therapy (SAPT), or no antiplatelet therapy (NAPT). Primary endpoint was any ICH (HT1-2, PH 1-2 or rPH) and sICH (PH2 plus neurological worsening by 4 or more NIHSS points) and 30-day modified Rankin Scale (mRS). The mRS was dichotomized as favorable (score 0-1) or unfavorable (score 2-5) at 30 days for each group.

Results:

Among tPA patients and ASPECTS of 10 (n=1020), there was no increase ICH/sICH with DAPT or NAPT (OR = 0.935, CI 95% = 0.569 - 1.535, p=0.789, OR = 0.746, CI 95% 0.515 - 1.082, p=0.112). In tPA patients with ASPECTS 9 or less (n=268), there was also no ICH/sICH difference between the DAPT or NAPT groups (OR = 0.645, CI 95% 0.369- 1.160, p =0.123, OR = 0.896, CI 95% = 0.434-1.850, p =0.765,). In tPA patients on SAPT vs. NAPT risk of ICH/sICH remained statistically not significant, irrespective of ASPECT score. After adjustment for absence or presence of early ischemic changes on pre-treatment CT, mRS scores were better among tPA patients on DAPT vs. aspirin alone (p= 0.012), similar for Clopidogrel alone (p=0.116) and better overall for DAPT vs. NAPT (P = 0.003).

Conclusion:

IV alteplase treatment of stroke patients on DAPT did not lead to an increased risk of ICH/sICH at our center. Pre-tPA use of DAPT may lead to better functional recovery after stroke, and therefore prior DAPT use should not be used as a reason to withhold or reduce IV alteplase dose irrespective of presence of early ischemic changes on CT.

38: Optic Nerve Sheath Diameter as a Non-Invasive Intracranial Pressure Measurement

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Introduction:

The use of bedside ultrasound measurement of a patient's optic nerve sheath diameter (ONSD) has been validated as an indirect screening measure for elevated ICP. A measurement of >0.5cm has been shown to correlate with an ICP >20cm H₂O ^[1,2,3]. These measurements have been published and used in the field of Emergency Medicine, however there are no described cases of using this measurement for decision making in the management of Neurosurgical patients.

Case Presentation:

We present a case of a 69-year old male, with a past medical history of colon cancer and diabetes mellitus, who was admitted with multi-compartment intracranial hemorrhage after a syncopal fall from standing. Computed tomography (CT) scan findings were significant for bilateral anterior acute subdural hematomas, diffuse subarachnoid hemorrhage, bilateral cerebral contusions, and skull fractures. Repeat imaging eventually showed stability of his hemorrhages with no development of significant mass effect, and thus he was initially managed non-operatively in the Neurosciences Intensive Care Unit.

The patient was transferred on the ventilator, however, he was eventually extubated. He had a non-focal neurologic examination but was unable to follow simple commands. Multiple electroencephalograms (EEGs) were performed, that did not demonstrate any epileptiform activity. The patient then had a significant decline in his neurologic examination and required re-intubation and vasopressor support. Repeat CT scans demonstrated interval development of bilateral low-density subdural fluid collections, however there was no midline shift or basal cistern effacement appreciated. Without clear explanation for his persistent encephalopathy, the family contemplated withdrawal of care.

Using a bedside ultrasound, the patient's ONSD measurements averaged to 0.6cm in the right eye, and 0.66cm in the left eye. With this information, a low-volume lumbar puncture was performed that confirmed intracranial hypertension, with an opening pressure of 52cm H₂O. The patient was taken to the operating room for bilateral subdural hematoma evacuation with the use of burrhole craniotomies. He tolerated the procedure well, had a tracheostomy placed post-operatively, and returned to following commands prior to discharge.

Discussion:

This case illustrates the role that ONSD measurements can have in critical decision making for Neurosurgical patients. ONSD is a safe and valid alternative to invasive ICP measurements.

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39: Acute Multiple Intraparenchymal Hemorrhages as a Rare Complication in a Case of Severe Diabetic Ketoacidosis

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Neurological deterioration in children with diabetic ketoacidosis (DKA) is well known and is commonly caused by cerebral edema (1). However, stroke (thrombotic or hemorrhagic) should also be suspected when there are focal neurological deficits or if there happens to be deterioration or no improvement in mentation during or after metabolic correction of the DKA (1). The prevalence of stroke as a risk factor for DKA ranges from 0% (3) in some studies to as much as 7% (4). Children with hyperglycemia and DKA are prone to thrombosis. Case reports of thrombotic stroke and DKA, though limited, have been reported before (2), but multiple intraparenchymal hemorrhages (IPH) as a complication of severe DKA have been rarely reported.

We would like to report a case of a 13-year-old girl with a known history of type 1 diabetes mellitus who presented to the emergency department with acute altered mental status (Glasgow Coma Scale, GCS, of 8) and was found to be in severe DKA. Some of her initial laboratory findings included a blood pH of 6.77, serum osmolality of 364mmol/kg, serum betahydroxybutyrate of 117mg/dl and a serum lactate level of 3.3mmol/L, indicating severe keto acidosis. Standard hospital protocol for DKA management with insulin and intravenous fluids was being followed and the patient was admitted and monitored in the critical care unit. It was noted that despite the correction and improvement in the metabolic parameters of the patient, her mental status showed no improvement, and she continued to be obtunded with a GCS of 8. An emergent computerized tomography (CT) of the head was obtained and the patient was noted to have an acute intra parenchymal hemorrhage in the frontal area of the left cerebral hemisphere. No acute neurosurgical intervention was done at that time. Three days later, a magnetic resonance imaging (MRI) of the brain and brainstem including susceptibility weighted imaging (SWI) sequences was obtained which showed multifocal subacute intra parenchymal hemorrhages in the left parietal and bilateral temporal lobe without any evidence of mass effect (Figure 1)

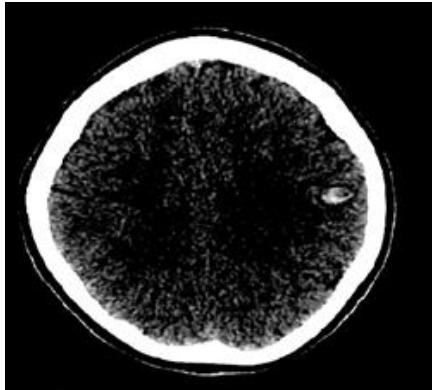


Figure 1-A

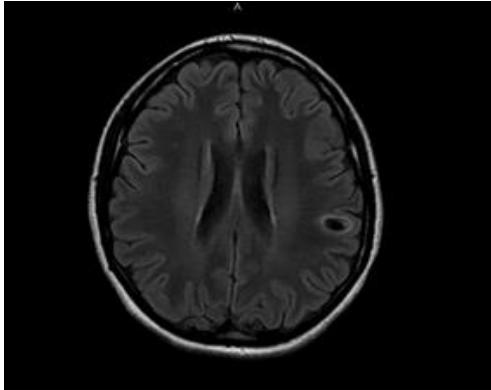


Figure 1- B

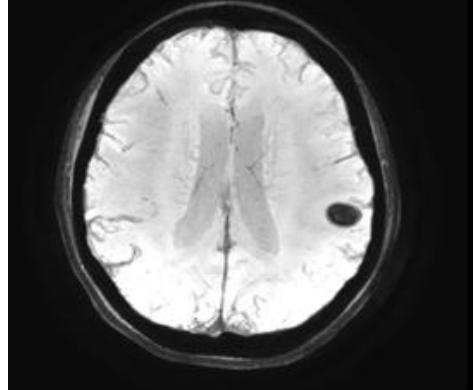


Figure 1- C

Figure 1: CT head (Fig 1-A), Axial T2 FLAIR sequence (Fig 1-B) and Axial SWI sequence (Fig 1-C) showing acute intraparenchymal hemorrhage in the frontal region of the left cerebellar hemisphere.

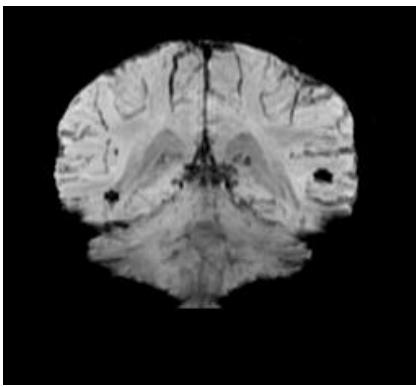


Figure 2-A

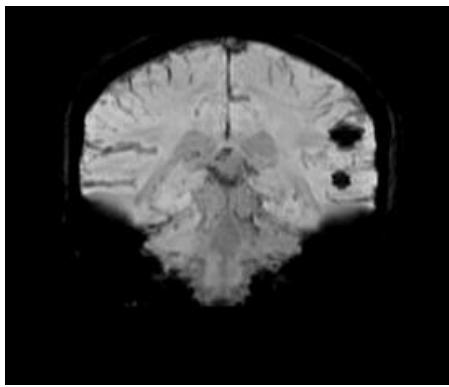


Figure 2-B

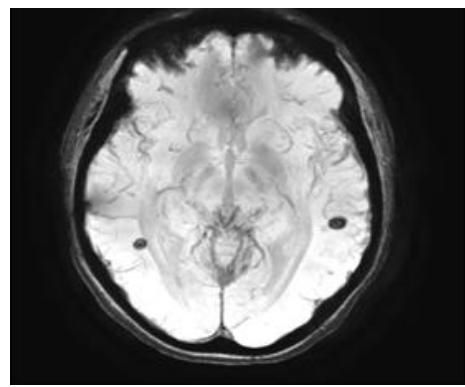


Figure 2-C

Figure 2: Coronal (Fig 2-A, 2-B) and Axial (Fig 2-C) SWI sequences showing multifocal and bilateral distribution of the acute intraparenchymal hemorrhages.

So, we would like to emphasize on the CNS complications of DKA and describe the various relevant pathophysiologic mechanisms involved dehydration, hyper osmolarity, tissue hypoxia, pH reduction, disseminated intravascular coagulation, lactic acidosis, increased platelet aggregation, and hyperlipidemia, resulting in hyper viscosity (5-10). Although cerebral edema is a well-known complication of severe DKA or rapid correction of underlying metabolic derangements, the possibility of acute thrombotic strokes and intra cerebral hemorrhages cannot be excluded without appropriate imaging, especially in the setting of worsening mental status or with the development of new focal neurological deficits. Furthermore, the lack of clinical data on the management of DKA associated with thrombotic strokes and intra cerebral hemorrhages increases the complexity of its management. Hence, we will make few recommendations based on the available data and review the literature.

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