Clinico-demographic Predictors of Limb and Bulbar Onset Symptoms in Clinically Definite Amyotrophic Lateral Sclerosis (Modified El Escorial)

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Introduction: Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disorder characterized by progressive loss of upper and lower motor neurons (UMNs/LMNs).

Objective: To systematically analyze the clinico-demographic predictors of the site of symptom onset in ALS.

Methods: Clinico-demographic data of all clinically-definite adult sporadic ALS patients were collected from Carolina Medical Center ALS-MDA clinic between July-2009 and December-2010. Chi-square analysis was performed to identify statistically significant association between the categorical variables and site of onset; T-test or analysis of variance was used for continuous variables.

Results: Clinically-definite, clinically-probable, and probable laboratory supported ALS patients were included (n=69; mean age=57.4 years; 75% Caucasian; 78% right handed; 52% male). Onset site was distributed: 32% bulbar, 33% lower-limb(LL), 35% upper-limb(UL). Average time from symptom onset to diagnosis was longer in LL-onset patients, p=0.04 (16months bulbar, 18months UL, and 24months LL) and 56% had UMN symptoms at onset. LL-onset presented at 51.3 years (47-55.5) as compared to bulbar-onset at 61.4 years (54.5-68, p<0.05). During the course of illness, pseudobulbar-affect (PBA) developed in 30% and fronto-temporal lobe dementia in 6%. Tobacco use occurred in 35% and alcohol in 26%, 49% had an outdoor-profession. Tobacco use, after controlling for gender and alcohol use, is a significant risk factor for PBA [HR=4.19 (1.20-14.7), p=0.025].

Conclusion: Age is a significant predictor of the site of ALS onset with LL onset presenting earlier as compared to bulbar and UL onset. Tobacco use is a significant risk factor for developing PBA among ALS patients, which will need confirmation in additional cohorts.
Bariatric Surgery for Obesity; Now a Leading Cause of Neurological Illness Related to Thiamine Deficiency in Published Case Reports

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**Background/Purpose:** Thiamine deficiency may cause devastating and irreversible nervous system damage if it is not recognized and treated in a timely fashion. The primary aim of this study was to determine if bariatric surgery (BS) has become a leading cause of reported symptomatic thiamine deficiency in medical literature over the past decade.

**Methods:** A PubMed literature review was conducted incorporating the search terms “thiamine deficiency.” Inclusion criteria were: cases published between 2003 - 2013 in English language, availability of full text article, patient age > 18, and presence of neurological symptoms and/or findings. We excluded cases in which the description of the clinical presentation was incomplete, diagnosis of thiamine deficiency was doubtful, or there was another potential explanation of patient’s presentation. Presumptive etiology of thiamine deficiency of each case was noted.

**Literature Review:** We reviewed 229 cases and 160 of those satisfied our inclusion/exclusion criteria. Thiamine deficiency was attributed to BS in 36 (23%). Other common causes of thiamine deficiency were strict dietary restrictions (n=54; 34%), chronic alcohol abuse (n= 26; 16%). The presenting syndrome in those cases associated with BS presented was isolated WE (n=21 ; 58%), isolated PN (n= 4; 11%), or WE+PN (n= 11; 31% ). With respect to timing of patient presentation to medical specialists, 5 patients (14%) presented < 1 month following surgery, 20 (55%) presented 1- 5 months following surgery, 6 patients (17%) presented 6 months- 2 years following surgery, and 5 patients (14%) presented >2 years. Intractable vomiting was present in a 32 (89%).

**Conclusions:** Over the past decade, bariatric surgery has been the a leading cause of reported symptomatic nervous system dysfunction associated with thiamine deficiency. This procedure has been associated with nearly one -quarter of case reports published in this time frame. Patients most often presented with a WE syndrome <6 months following surgery, however delayed presentation beyond this time point in 1/3rd of patients. As bariatric surgery becomes common place as an intervention for obesity in the future, the number of patients presenting with WE and/or PN related to thiamine deficiency is likely to grow.
Biomarkers of CNS Injury and Degeneration

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A panel of highly specific and sensitive ELISA and chemiluminescent assays for proteins released from the nervous system as a result of damage and disease states is being developed. Such biomarker assays have the potential to diagnose onset of neurodegenerative diseases, develop prognostic information, subclassify disease types and monitor the responses to therapy. The first of these is the phosphorylated axonal form of the major neurofilament subunit NF-H (pNF-H). It was shown that this can be detected in informative amounts in the CSF and blood of animal models and patients suffering from aneurysmal subarachnoid hemorrhage and other states associated with axonal loss, while control individuals express little or no pNF-H in blood or CSF. It was recently shown that blood levels of pNF-H are elevated in patients with ALS and that the blood level of this protein provides a unique predictive marker of the rate of disease progression. Other studies further underline the utility of blood pNF-H measurement as a convenient surrogate measure of axonal loss in mouse models of Multiple Sclerosis and in patients suffering from Leber’s hereditary optic neuritis. It was discovered ubiquitin C-terminal hydrolase 1 (UCHL1) as a biomarker primarily of perikaryal loss which is abundant and resistant to brain proteases and which can be detected both in CSF and blood. UCHL1 can be detected in elevated amounts in the blood of patients recovering from traumatic brain injury, and the levels detected, combined with the levels of S100β, provide a novel means of measuring TBI related blood brain barrier compromise. Novel assays for GFAP, alpha-synuclein and several other potential biomarker proteins have been developed.
Epilepsia Partialis Continua Versus Myoclonus: 
A Diagnostic Dilemma

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Epilepsia partialis continua is a condition uncommonly seen in the adult population. It can present as continuous rhythmic focal motor signs without a march emanating from the motor cortex. Only small series of cases have been published. The most common presentation is Rasmussen encephalitis in children, but other causes such as tumors, vascular etiologies, cortical dysplasia and trauma have been found. Palatal tremor is a rhythmic movement disorder of the soft palate, associated with lesions of the inferior olivary nucleus that persists during sleep.

We present a case of a 53 year old man with intractable epilepsy status post traumatic brain injury. Admission was prompted by continuous rhythmic twitching of palate, uvula and left index finger, which persisted during sleep. Work up included MRI Brain with and without contrast showed focal T2-hyperintense signal in the anterior inferior aspect of the right medulla in the region of the inferior olivary nucleus, most compatible with olivary degeneration secondary to ipsilateral midbrain insult. Continuous video-monitor electroencephalography showed rare right anterior temporal spikes during sleep and left frontal slowing with sharp activity indicating an area of cortical hyperexcitability.

Initial aggressive treatment of the focal seizures led to sedation, worsening gait and failure to self-feed. His regimen was subsequently changed and currently he continues to display rhythmic twitching, at times sparing the pharynx, but remains fully alert. This case engenders a diagnostic dilemma as to whether the twitching movements constituted epilepsia partialis continua versus palatal tremor or a coexisting dual diagnosis.
Cerebral Vasospasm Following Transsphenoidal Pituitary Macroadenoma Resection

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Cerebral vasospasm after aneurysmal subarchnoid hemorrhage is a well-known entity responsible for significant neurological deterioration and morbidity. But Cerebral vasospasm following transcranial pituitary adenoma resection surgery has been extremely infrequent with only few cases described. Cerebral vasospasm and its management has remained a great challenge ever since its description for the first time in 1951.

A 19 year-old young lady with known history of pituitary macroadenoma, which was associated with Cushing’s disease, status post transsphenoidal resection at age 18, who presented with aphasia and right upper extremity (RUE) weakness 7 days post left sided fronto-temporal craniotomy with orbital osteotomy for further resection of cavernous sinus adenoma. Patient presented after she woke-up with above symptoms. Examination revealed RUE weakness, expressive aphasia. NIH Stroke Scale Score was 11.

Brain MRI showed acute stroke in the distribution of the left middle cerebral (MCA) and left anterior cerebral (ACA) arteries. Head MRA showed narrowing of proximal left M1 MCA and A1 ACA concerning of vasospasm, patient was immediately started on triple HHH therapy. At the same time, patient was taken to the angio-lab for intra-arterial (IA) Verapamil and Nicardipine treatment with angiographic improvement. This procedure had to be repeated on 9 different occasions within few days. Patient improved with each IA treatment, but symptoms recur shortly after. The patient’s clinical condition slowly improved, her weakness and aphasia markedly improved by the time of discharge.

The pathogenesis of vasospasm after tumor surgery is not very well understood. Various possible causes for vasospasm after tumor surgery include direct mechanical damage to arterial walls, deposition of blood in the basal cisterns, aseptic meningitis, or release of some chemical substances during tumor removal (Craniopharyngioma fluid cause vasospasm of rat’s femoral vessels). The management can be very challenging and the key to success is a high index of suspicion and early proactive management.
Lateral Medullary Syndrome Mimicking Pure Sensory Stroke

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Introduction: Lateral medullary infarction (LMI) is classically thought to induce crossed facial and body spinothalamic sensory deficits. We present a patient with LMI in whom body and facial sensory loss were not crossed. The primary aim of this study was to determine how frequently LMI presents with non-crossed sensory deficits.

Methods: A PubMed literature search was conducted for the following terms: “lateral medullary syndrome” “lateral medullary stroke” or “Wallenberg syndrome”. Inclusion criteria were consecutive case series of LMI, MRI confirmed infarct, and documentation of location of sensory deficits. Single case reports and patients with acute LMI + acute infarction outside the LM were excluded. These results were pooled and analyzed.

Results: Three articles encompassing 91 cases of LMI met study inclusion criteria. Spinothalamic sensory deficits were ipsilateral facial + contralateral body in 33 (36%), a combination of isolated face or body or bilateral facial symptoms in 36 (40%) and 22 (24%) were contralateral face and body. No cases had ipsilateral facial + body sensory deficits.

Discussion: Nearly one quarter of patients with LMI have uncrossed sensory loss contralateral to their infarct. Anatomically this finding is likely explained by selective lesioning of the ascending trigeminal tract and spinothalamic tract in the ventral/medial portion of the lateral medulla. Clinicians should be aware of this pattern of presentation in order to not overlook a small medullary infarct if neuroimaging does not reveal a lesion in an area typically associated with pure sensory stroke (e.g. thalamus, internal capsule, pons).
Morphometric Brain Volume Measurements in Medical Cadaveric Scans with Dementia and Metabolic Syndrome

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**Background:** Several research groups have previously reported that subregional volumetric brain measurements outperform available measures for tracking severity of Alzheimer’s disease. Recent study revealed significantly more brain atrophy in people with diabetes than in a non-diabetic control group with a normal aging brain at age 65 shrinking about one percent a year while in a diabetic that number increases to an astounding 15 percent. Utilizing the USF Center for Human Morpho-Informatics Brain CT and MRI images we developed method for quantification of total and subregional brain volume loss in patients who died with dementia and one or more components of metabolic syndrome. We performed measures of total brain volume, cerebellum, medulla, midbrain and pons and regions such as hippocampus, thalamus and amygdala. We hypothesized that patients who had diabetes and hypertension also suffered more prominent brain atrophy then patients with dementia alone.

**Methods:** Anterio-posterior measurements were made from the most posterior aspect of the tentorium to the most anterior frontal lobe, in a plane just under the genu of the corpus callosum. A perpendicular line was drawn from the basion of the skull base to the superior frontoparietal lobe to obtain the crandiocaudal (CC) dimension. Finally, the transverse dimension was measured from the most lateral aspect of one temporal lobe to the other. 162 cadaveric medical scans of random ages and sexes were used as the sample dataset.

**Results:** Preliminary data show strong correlation between total brain atrophy and increasing number of metabolic syndrome components in Alzheimer’s dementia. Cerebellar atrophy is evident in Alzheimer’s dementia with atherosclerosis, hypertension and diabetes as comorbidities. Moderate atrophy of pons and medulla in AD patients with comorbidities was evident.

**Conclusions:** CT and MR imaging of the brain can be used as a noninvasive method to obtain accurate and reproducible quantitative measures of alterations in brain structure which can be predictive of dementia severity.
Neuro Spectrum – Color in Line Bisection and Quadrisection

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Objective: Does color, gender, and age effect bisection and quadrisection? Might bisecting and quadrisecting on the same line affect bisection or quadrisection?

Methods: We printed 240 mm lines in middle of blue, red, yellow, orange, green, purple, white, gray papers (8.5x11 inches). Participants had normal color vision and counterbalanced dividing lines on each color for each condition; bisect then quadrisect, bisect, quadrisect left, quadrisect right. Lines were measured in millimeters. Averages and standard deviations were found for condition, color, age, sex.

Results: Thirty-one 7th grade students and seven 7th grade teachers participated. Some of the copies were slightly off of 240 mm, corrected by calculating percent of line, (left 0%, right 100%). Color and gender had no effect. There was no difference in line bisection between students and teachers, each showed normal pseudoneglect. Left and right quadrisection were farther from the middle for the combo condition, and for students.

Conclusions: After 1300 measures, we discovered color doesn’t affect horizontal bisection or quadrisection. Different color paper can safely be used for marking papers in horizontal line dividing projects. It may affect vertical line dividing but that wasn’t tested. We concluded that students and teachers bisect similarly, but quadrisect differently with students going farther from middle than adults; we think because the lines seem bigger to children. Both groups divided combos different than single dividing, maybe because in the combo they move away from center when doing on the same line.
Frontal Lobe Agraphia, A Stroke in the Area of Exner—A Case Report

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**Background:** Frontal lobe agraphia is rare as it is more commonly described in parietal lobe lesions. Further rare is a frontal lobe infarct which results in agraphia without causing apraxia, alexia or aphasia. We describe a case of a selective infarct involving the left frontal lobe in an elderly man.

**Design/Methods:** A literature search was performed for comparable case reports.

**Case description:** A 75 year old, right-handed man, presented to the hospital with an inability to write his name properly. He noticed when attempting to buy his wife a dress he was unable to sign the credit card slip. He described the experience as knowing exactly what he wanted to write but that his signature was not his own. He denied any weakness in hands, difficulty with speech, reading or comprehension. His risk for stroke included hyperlipidemia, hypertension, diabetes, alcohol and tobacco use and history of TIA. On physical exam the only new deficit noted was poor hand writing, but grammar and spelling were intact. No apraxia, anomia, simultagnosia, acalculia, finger agnosia, left-right disorientation, or aphasia was found on exam. No deficits were found in mental status, motor function, reflexes, cerebellar testing or gait and station. Decreased light touch, pinprick and proprioception up to wrist and ankle bilaterally were also noted. On CT of head without contrast areas of encephalomalacia were found in the left frontal and possibly right parieto-occipital lobe. MRI of brain showed a small area of remote ischemic change involving the left superior frontal gyrus with cortical encephalomalacia and subcortical white matter gliosis as well as multiple sites of acute ischemic change within the left cerebral hemisphere, which involved the left frontal, parietal, occipital, and posterior temporal lobes. MRA of head and neck showed a high-grade short segment stenotic lesion of the proximal left internal carotid artery and asymmetric appearance of the distal left MCA segmental branches. Carotid US showed mild stenosis of the right ICA and severe stenosis of the left ICA. Left carotid stenosis was repaired with left carotid endarterectomy. Agraphia as an isolated symptom has at least four localizations and in this patient proved complex due to his prior stroke adjacent to Broca’s area and his new stroke involving the superior and inferior parietal lobes. Due to the description of his symptoms the most likely localization is in the frontal lobe.
CT Perfusion (CTP) has fast become the imaging modality of choice in suspected acute stroke. It can also shed light on stroke mimics (seizures, PRES, hypoglycemia, tumors). Many peculiar CTP changes in relation to seizures have been reported in the literature. Our study sought to elucidate specific patterns in CTP that can delineate seizure from stroke. Methods: We retrospectively identified patients who presented to our stroke center as stroke alerts but received a discharge diagnosis of seizure between 2008 and 2011. We excluded patients who did not get a CTP or EEG as part of their work up. We compared the patient’s CTP with their EEG and MRI. Results: 91% of patients who presented with seizure, had an abnormal CTP. Of these 66% had typical CTP changes suggestive of seizure i.e. ipsilateral increased perfusion, or contralateral increased Time To Peak (TTP), or unilateral increased Cerebral Blood Flow (CBF) and decreased TTP. A minority showed globally increased CBF with EEG showing status epilepticus. The Pattern of perfusion abnormality commonly encountered in seizures was cortical ribboning, sparing the basal ganglia, and not following a particular vascular territory. Similarly large vessel CTA changes were absent. Conclusions: Ictal CTP abnormalities are common in patients presenting to the ED with seizure. The most common finding in seizure was focal hyperperfusion (often misread as opposite hemisphere hypoperfusion). CTP is thus a sensitive tool to help differentiate strokes and seizures.
Preventable Errors in Hospitalized Parkinson's Disease Patients

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Objective: Parkinson’s disease (PD) patients are more likely to be hospitalized and we aimed to investigate preventable management errors during hospitalization.

Methods: An IRB approved protocol used a retrospective, consecutively admitted PD patient’s chart review for the study. Patients were admitted to the UF Health Hospital from November 2012 to March 2013. SPSS statistics was used to analyze data.

Results: Of 176 cases, 85 had a PD diagnosis. There were 51 (60%) males, age was 74.1 (±9.0), and 95% were on levodopa (LD). LD dosages were written with specified times in only 15%. These were administered late in most patients: 1 hour (67%), 2 hours (49%), 3 hours (39%) or dosages were missed in 49%. Length of stay for those who received LD on time was 3±3 days vs. those with delayed and missed dosages (7±6 days). Swallowing and physical therapy consultations were performed in 27% and 57% of cases. Fall risk evaluation was performed in 80%. Falls occurred in 2.4% during the hospital stay. There were no falls in the group that received counseling. Dopamine blockers were administered in 23% of cases with quetiapine prescribed most frequently (53%). Contraindicated dopamine blockers included haloperidol (21%), olanzapine (5%), risperidone (5%), and promethazine (5%). Length of stay for those receiving quetiapine was 5±5 days vs. 10±9 for those receiving contraindicated DA blockers. The death rate was 6%.

Conclusion: Hospitalized PD patients are victims of frequent medication errors. Potentially modifiable factors could be contributing to prolonged hospitalizations, and to morbidity and mortality.
Neuroinflammation in Stroke- Review

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Cerebral ischemia results in a complex cascade of reaction involving energy failure, excitotoxicity, ionic imbalance, channel dysfunctions, neuroinflammation and oxidative free radicals in the central nervous system. If the blood flow is not restored within a reasonable time frame the outcome becomes devastating. Many systemic inflammatory conditions, exogenous toxins, environmental factors, infections involve central nervous system. The damage to the neuroendovascular unit comprising of the blood vessel, astrocytes, neurons and surrounding microglia trigger the whole spectrum of reaction which is diverse in nature. The cerebral ischemia results in activation of deadly latent proteases and immediate early genes destroying the neuronal microenvironment and blood brain barrier. The cascade of cerebral injury also involves activation of microglia and astrocytes leading to release of chemical mediators like cytokines, oxygen free radicals, neurotoxic and neurotropic factors further contributing to the damage. Neutrophil activation and binding to endothelial surface using adhesion molecules and their subsequent transmigration to the ischemic core will enhance the injury. Monocyte and macrophage will also play a role in brain injury by its release of cytokine and transformation into phagocytes. Strategy to target various players of neuroinflammation to halt or minimize the cerebral damage concentrate on inhibiting intracellular adhesion molecules (ICAMs), vascular cell adhesion molecule (VCAMs), neutrophils, microglia, major histocompatibility complex (MHC), cytokine, chemokine and free radical scavenger system.

Different strategies tried to suppress inflammation secondary to ischemia have shown promise in experimental environment but have failed to demonstrate successful clinical translation and further studies are underway.
Spontaneous Spinal Epidural Hematoma is Most Frequently Idiopathic in Cause

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**Background:** Spontaneous spinal epidural hematoma (SSEH) often presents with acute onset of neck and/or back pain with sensory and/or motor deficits. These symptoms can mimic an acute ischemic event of the brain or spinal cord. The primary purpose of this study was to review the medical literature to discern how frequently no cause of spontaneous spinal epidural hematoma is identified.

**Methods:** Literature search was conducted of case reports and review articles in PubMed spanning 1959 to 2013. Search terms were “spontaneous”, “spinal”, “epidural hematoma”. Abstracted information included patient age and gender, use of imaging modality to confirm hematoma, presenting symptoms and etiological factor(s) if known. Patients with non-spontaneous epidural hematomas were excluded from this analysis.

**Results:** Mean patient age was 65 years old and males represented 63% of cases. Eight-five percent of cases presented with sudden onset of pain at the site of epidural hemorrhage. No identifiable etiology was found in 39% of cases. The second most common etiological factor was use of antiplatelet therapy (17%). Vascular malformations comprised 9% of cases, followed by hemorrhage of tumor (6%). Prior trauma was associated with 5% of cases.

**Conclusion:** No defined etiology is uncovered in approximately 1/3rd of cases. Clinicians should have spontaneous spinal epidural hematoma on their differential when evaluating a patient with neck and/or back pain who also experienced sudden onset of sensory and/or motor deficits.
Interleaving: Might be a Better Programming Technique for PD and Dystonia Patients

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Objective: To determine if patients with Parkinson Disease and Generalized Torsional Dystonia (GTD) would benefit from the use of Interleaving Programming.

Background: Currently Deep Brain Stimulation (DBS) is the surgical procedure of choice for patients with Parkinson’s disease (PD), Essential Tremor and GTD. Recently, a novel programming technique called Interleaving has emerged, enabling the delivery of different combinations of stimulation in the same lead, resulting in alternating stimulation of two different anatomical areas. There is only one recent case report comparing DBS interleaving with single DBS programming in Parkinson’s patients.

Methods/Design: We prospectively conducted four patients using interleaving and compared ON Single Setting versus ON Interleaving Mode. All patients were from the Movement Disorders Center at Cleveland Clinic Florida. The Unified Parkinson Disease Rating Scale (UPDRS) and/or Dystonia Rating Scale (DRS) were used. The scoring was done by a blinded physician trained in the use of UPDRS and DRS rating scales.

Results: Three patients had Parkinson’s disease and one patient with Generalized Torsional Dystonia. Each patient was examined and assessed ON Single stimulation, Interleaving and OFF Settings and they were evaluated on each setting with the use of Unified Parkinson Disease Rating Scale (UPDRS) and the Dystonia Rating Scale (DRS). PD patient on Interleaving stimulation demonstrated a 51% improvement on the UPDRS when compared to the 37% using single settings. Similarly, the patient with GTD showed a 54% reduction in the Dystonia Rating Scale on Interleaving when compared to single setting mode.

Conclusions: The use of interleaving programming may give patients a better clinical outcome by allowing us to use the submaximal amplitude. In addition, a more localized stimulation and a broader captured region with no side effects while managing the cardinal parkinsonian/dystonia symptoms. Therefore, DBS interleaving programming may be useful in patients with PD and/or dystonia.
Antiplatelet Therapy Benefit in Secondary Stroke Prevention in Patients with Suboptimally Controlled Treatable Cerebrovascular Risk Factors

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Objective: To assess the impact of antiplatelet therapy for secondary stroke prevention in patients with suboptimally controlled treatable cerebrovascular risk factors per guidelines.

Background: Aspirin (50 mg-325 mg/d), clopidogrel and extended-release dipyridamole are all acceptable options for initial therapy for secondary ischemic stroke prevention in patients with noncardioembolic stroke. Aspirin 325 mg/d is also recommended in selected patients with atrial fibrillation who have contraindications to anticoagulation. There are no data available assessing the benefit of antiplatelet therapy in secondary stroke prevention in the absence of optimal cerebrovascular risk factor control.

Materials and methods: We collected retrospective data from all patients in the Cleveland Clinic Florida EMR with a history of stroke between November 2006 and December 2012. A total of 1755 patients who met this criteria were analyzed. Data includes date of first stroke, treatable risk factor control, type of antiplatelet therapy used, date of second stroke, and stroke classification. Risk factors including hypertension, hyperlipidemia, and diabetes were monitored and analyzed on the basis of follow up clinic visits.

Results: Of the 1755 patients, 219 (12.47%) patients had a second stroke event between November 2006-December 2012. Mean age of this group of patients was 66.4 years old with male predominance (M:F=1.5:1).

192 (88%) patients were on antiplatelet therapy at the time of the second stroke. 27 (12 %) were not on antiplatelet therapy.

Of the total cohort, treatable risk factors were poorly controlled in 130 (59%) patients: 121 (55%) hypertension; 78 (36%) hyperlipidemia, 44 (20%) diabetes.

Antiplatelet agents included aspirin, clopidogrel alone or in combination with aspirin, and extended-release dipyridamole. The data showed no significant difference among antiplatelet agents for secondary stroke prevention (p<0.05).

Stroke classification was as follows: 162 (74%) ischemic and 57 (26%) hemorrhagic. Categories of ischemic stroke were as follows: large-artery atherosclerosis 40 (18%), small vessel disease 45 (21%), cardioembolic 42 (19%), 35 (16%) cryptogenic / stroke of undetermined etiology. Of the 192 patients on antiplatelet therapy with stroke, 116 (60%) had poorly controlled risk factors.
Conclusions: This study suggests that the use of antiplatelet therapy, regardless of which agent, for secondary stroke prevention may not be important or beneficial in the absence of optimal treatable cerebrovascular risk factor control. Further studies are needed to support this finding including further analysis of our data to assess risk factor control in those patients on antiplatelet therapy who did not suffer a second stroke during the period of surveillance.
Brain Atrophy Associated with Susac Syndrome

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Introduction: Susac syndrome (SS) is a microvasculopathy that produces a clinical triad of encephalopathy, branch retinal artery occlusion, and hearing loss. Brain MRI imaging classically shows multiple, punctuate, T2/FLAIR hyperintensities involving cerebral hemispheric white matter and the corpus callosum. We describe a patient with SS who exhibited progressive cortical, subcortical, and callosal brain atrophy over a decade. We report this case in the context of a literature review evaluating the prevalence of this radiographic finding in SS.

Methods: Literature search utilizing PubMed was conducted with the following terms: “Susac syndrome,” “RED-M,” and “SICRET.” Cases were included for review if they were published in English, met both clinical and radiographic diagnostic criteria for SS, and MRI report and/or image was available for review. Cases were considered to have atrophy if “atrophy” was mentioned in the description of the MRI findings.

Results: 200 cases met inclusion criteria. In 13 (6.5%) of these cases brain atrophy was described in the MRI findings. The MRI in which atrophy was described was conducted as early as symptom onset to 19 years after presentation. Age range of these patients was 21-45. In 10 reports, detailed regional description of atrophy was included. In 6 of these cases, the corpus callosum was prominently involved.

Conclusion: SS is a multi-organ disease in which brain MRI findings contribute to its diagnosis. Brain atrophy has been documented to occur in a minority of SS patients. The presence of whole brain atrophy or atrophy of the corpus callosum, in conjunction with typical T2/FLAIR sequence findings should not dissuade neurologists from making a diagnosis of SS. Precisely why atrophy occurs in some patients with SS is unknown.