EP2201
Profile of triggers and cerebrovascular risk factors in 321 patients with transient global amnesia (TGA)
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Introduction: The pathogenetic mechanisms causing transient global amnesia (TGA) remain elusive. Various aetiologies have been proposed, amongst are vascular causes, a migrainous phenomenon, cortical spreading depression, metabolic disturbance and epilepsy. Remarkably, cerebrovascular risk factors (CVRF) are not believed to be associated with TGA. Thus, we wanted to characterize the profile of CVRF in a large cohort of patients with TGA.

Methods: We performed a retrospective study of patients with TGA (from 1/2003 until 1/2009) according to the criteria by Hodges and Warlow (1990). The analysis included potential triggers and CVRF including arterial hypertension, obesity, hyperlipidaemia, diabetes, smoking and carotid stenosis over 50%.

Results: A total of 321 patients (female 65%) were studied, mean age was 64 y (range: 18-86).

Triggers were present in 31%. Most frequent were physical (20%) and psychological (8%) triggers. The most common CVRF was arterial hypertension (58%), followed by hyperlipidaemia (42%), diabetes (8%), smoking (3%), carotid stenosis over 50% (3%) and obesity (2%). One CVRF was present in 41%, 2 in 31%, 3 in 3%, and 4 in 1%.

Conclusions: Our data indicate the presence of CVRF in a subgroup of patients with TGA. While TGA is considered a benign, self-limited syndrome, this cohort may be prone to future cerebrovascular events. Further studies are required to evaluate long-term prognosis based on the profile of atherosclerotic risk factors.

Disclosure: Nothing to disclose

EP2202
Clinical, cognitive and neuroimaging features differentiating vascular from degenerative mild cognitive impairment. Results from a nested case-control study
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Introduction: Mild cognitive impairment (MCI) can be subsided by vascular or atrophic brain lesions. Clinical and neuroimaging markers selective of the two conditions are still incompletely determined, mostly because of the definitions and potential confounders. We investigated factors differentiating degenerative (deg-MCI) from vascular MCI (vas-MCI).

Methods: Using a nested case-control design we identified from two larger prospective registries two groups of 30 patients with vas-MCI or deg-MCI. Both groups had been assessed by a clinical, neuropsychological, laboratory and neuroimaging protocol.

Results: Familiar dementia was prevalent in deg-MCI group (67% vs 33%, p=0.019), while hypercholesterolemia (13% vs 67%, p<0.001), stroke (3% vs 37%, p=0.002), migraine (23% vs 53%, p=0.033), psychiatric disorders (40% vs 73%, p=0.018), gait disorders (10% vs 70%, p<0.001), and urinary disturbances (20% vs 50%, p=0.029) were more common in the vas-MCI group. Logistic regression showed an independent association between story recall test and deg-MCI (p=0.003), and depression severity with vas-MCI (p=0.045). On MRI lacunar infarcts (p=0.001) are associated with vas-MCI, while global atrophy (p=0.001) and enlarged perivascular spaces (EPSV) in hippocampus and centrum semiovale with deg-MCI (p=0.001 respectively). We found no difference in distribution of Apolipoprotein E gene polymorphisms analysis (27% vas-MCI vs 33% deg-MCI).

Conclusions: Few selective markers may discriminate vas-MCI and deg-MCI, as impaired episodic memory and depressive disturbances on clinical grounds, lacunar infarcts, global atrophy and EPSV on brain imaging grounds. If confirmed from larger series, this information may be useful to the setting of clinic-functional screening of patients with different MCI types.

Disclosure: Nothing to disclose
**EP2203**

**LDL subclasses in ischemic stroke: a risk factor?**

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**Introduction:** Elevated low density lipoprotein (LDL) plasma concentration is a primary risk factor in the development of atherosclerosis. By polyacrylamide gel electrophoresis methods, seven LDL subclasses were identified. Small dense LDL (sdLDL) is the main subclass responsible for development of the atherosclerotic process. In this study, the relationship between ischemic stroke and LDL subclasses was investigated.

**Material and methods:** In one year period, consecutive 110 ischemic stroke patients who were classified according to TOAST classification as cardioembolism (n=40), large-artery atherosclerosis (n=40) and small-vessel occlusion (n=30) and 60 healthy controls were included to this study. LDL subclasses were established by Lipoprint system polyacrylamide disc gel electrophoresis.

**Results:** The ischemic stroke group consisted of 61.8% (n=68) males and 38.2% (n=42) females and mean age was 66.7±12.0 years. The mean age of the control group was 61.2±4.2 years and 50% (n=30) were male, 50% (n=30) female. The LDL-2, LDL-3 and LDL-4 subclasses were significantly higher in the ischemic stroke group compared to the control group (p<0.05). There were no statistically significant differences between the LDL-1 levels of ischemic stroke and the control groups (p>0.05). Also, there were no significant differences between levels of LDL subclasses of cardioembolism, large-artery atherosclerosis and small-vessel occlusion subgroups (p>0.05).

**Conclusion:** sdLDL is an established risk factor for ischemic heart diseases, similarly LDL subclasses are higher in ischemic stroke patients. The examination of LDL subclasses may influence the treatment strategy and prognosis in ischemic stroke.

**Disclosure:** Nothing to disclose

**EP2204**

**Evaluated serum levels of brain-derived neurotrophic factor (BDNF) in acute ischemic stroke**

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**Introduction:** Neurotrophic factors are important molecules in recovery process of stroke patients. Brain-derived neurotrophic factor (BDNF) replies for proliferation, differentiation, structural and functional stability, viability of neurons. The aim of the study was to find relationships between serum levels of BDNF and clinical characteristics of patients with ischemic stroke.

**Methods:** We measured serum levels of BDNF in patients with acute ischemic stroke (n=39) within 24 hours from symptom onset, in patients with clinically stable cerebrovascular disease (n=28) and in healthy controls (n=26). Intensity of neurological impairment was estimated by the NIH Stroke Scale, kinesthetic praxis - by the Denckla tests, cognitive impairment - by the Mini-Mental State Examination and the Frontal Assessment Battery, depression level - by the Beck Depression Inventory.

**Results:** The serum levels of BDNF were significantly lower in acute stroke patients as compared to patients with clinically stable cerebrovascular disease and healthy controls. The serum levels of BDNF were also lower in patients with clinically stable cerebrovascular disease in comparison to healthy controls. The serum levels of BDNF in acute stroke patients were strongly correlated with the NIH Stroke Scale (p<0.01) on admission and kinesthetic praxis disorders (p<0.05), degree of cognitive impairment (p<0.01), depression level (p<0.01) at days 30 and 60 after stroke.

**Conclusions:** The received data may be important for individualization of diagnostics and therapeutic interventions in patients with acute ischemic stroke.

**Disclosure:** Nothing to disclose
EP2205

Cerebral venous sinus thrombosis: an analysis of 46 patients

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Introduction: Cerebral venous sinus thrombosis (CVT) is seen infrequently than arterial stroke and effects every age group. Onset and process is variable and prognosis is usually good, but early development of coma and intracranial hemorrhage leads to bad prognosis.

Methods: In our retrospective study, it’s aimed to search neurological deficits, etiological factors, localization and prognosis of the 46 patients who are hospitalized with the diagnosis of cerebral venous thrombosis in Neurology Clinics of Bezmi Alem Valide Sultan Vakıf Gureba and Okmeydani Education and Research Hospitals.

Results: Of 46 patients (30 women, 16 men) mean age was 39±12.29. 22 patients in subacute and 20 patients in acute stage admitted to our clinics. The most frequent symptom was headache (69.6%) and the most seen neurological sign was papillaedema (56.5%). 13 patients had hemorrhagic infarct, 6 had ischemic infarct, 24 had no parenchymal lesion, 1 had epidural abscess and 2 subarachnoid hemorrhage. 16 had isolated sinus thrombosis, the most commonly affected sinus in isolated or combined sinus thrombosis is transverse sinus (n=37). No etiological reasons were found in 11 patients, 17 had one, 18 had more than one reason. The predisposing factors were infections (n=8), puerperium (n=5), oral contraceptive pills (n=7), hereditary thrombophilia (n=21), Behçet’s Disease (n=2), ulcerative colitis (n=1) and head injury (n=1).

Conclusions: CVT needs highly clinical suspicion. Early treatment reduces the risk of exitus and severe disability. CVT should be remembered in the differential diagnosis of headaches of unknown etiology.

Disclosure: Nothing to disclose

EP2206

Thrombolysis with alteplase for acute ischemic stroke in octogenarians in Croatia

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Introduction: According to the Croatian licence alteplase is not to be used for treatment of acute ischemic stroke in octogenarians. Main concern in this patients group is post-thrombolytic intracranial haemorrhage.

Objective: To assess baseline and clinical outcome details in octogenarians compared to younger stroke patients treated with intravenous alteplase.

Methods: Data on patients treated with alteplase within 4.5 hours from stroke onset were prospectively collected from January 2008 until November 2013. Patients were categorized in two groups, younger and older than 80. Baseline and outcome details between groups were compared.

Results: 260 patients were included in this study, 140 male and 120 female, mean age 69±11.5 (range 28 to 90). There were 19.6% (51) octogenarians; 13% of male patients and 28% of female patients. 23.5% (12) octogenarians and 15.8% (33) younger patients had “time-to-treatment” longer than 3 hours. There was no statistically significant relationship between age and previous history of hypertension; modified Rankin Score at admission or post-thrombolytic intracranial haemorrhage. “Time-to-treatment” >3 hours was not related to post-thrombolytic intracranial haemorrhage in any group. Results indicate that there was statistically significant relationship between age and mortality (Pearson Chi-Square = 21.3, p<0.001), indicating that the mortality rate is higher in octogenarians. Patient age and atrial fibrillation are statistically significant related (Pearson Chi-Square = 17.9, p<0.001), indicating that the FA is more likely in older patients.

Conclusions: Among patient who receive thrombolytic therapy for ischemic stroke the octogenarians have greater risk of death but not of post-thrombolytic intracranial haemorrhage.

Disclosure: Nothing to disclose
EP2207
Cardiac troponin-T changes in acute ischemic stroke
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Introduction: Elevation of cardiac troponin-T (cTnT) in serum reflects myocardial injury, but it was also observed in other conditions with cardiac injury including acute ischemic stroke. The objective was to identify the relationship between elevated cTnT and stroke severity, location and outcome.

Methods: cTnT levels were prospectively performed in 385 patients with different subtypes of acute ischemic stroke admitted in NICU within 72 hours of onset, as TOAST criteria. The patients were divided into two groups: an elevated cTnT (group 1) (n=42) and a normal cTnT (group 2) (n=343). The short-term prognosis was assessed by 30-days modified Rankin Scale responder analysis and the NIHSS. Serum cTnT levels were determined using a high sensitive Troponin-T assay (Roche Elecsys Troponin, Mannheim, Germany), cut-off value at 0.01 ng/mL. Statistical analysis was performed.

Results: Serum cTnT level was elevated in 10.91% (42/385) of patients. cTnT positivity on admission is an independent and powerful prognosis predictor in acute ischemic stroke. More frequently insular lobe involvement was observed in elevated cTnT group (17/42) (31%) than in group 2 (55/343) (16%) (p=0.040). Stroke severity was greater in elevated cTnT group. The outcome was worse in elevated cTnT group as compared to group 2 (13/42) (30.95%) vs. (68/343) (19.82%) (p=0.013).

Conclusions: cTnT in acute ischemic stroke is a marker of stroke severity, of insular lobe lesion and of prognosis prediction. cTnT is a highly specific and sensitive marker of myocardial damage in acute ischemic stroke due to insular lesion that induces disturbances of autonomic balance.

Disclosure: Nothing to disclose

EP2208
Imaginary and clinical correlations in progressive ischemic stroke
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Introduction: Progressive ischemic stroke (PIS) represents a particular form of stroke with a worsening clinical course, more specific an alteration of the neurostatus with consecutive examinations.

Methods: The studied group consisted in 51 patients. Based on the clinical and imaginary criteria were diagnosed with PIS. Considering the time elapsed from the debut to the aggravation of the neurological symptoms, patients were divided in early worsening of the neurological status (24 to 36 hours since the debut of the stroke) and late worsening of the neurological status (36 hours up to 1 week). The methods used to assess the patient’s neurostatus were the NIH stroke scale and notable changes in the neurological deficits. Complementary lab tests and neuro-imaging -computed tomography (CT) and magnetic resonance were performed also. Risk factors, affected arterial territory, associated neurological deficits, extracerebral complications and the aspect of the cerebral lesions in the CT performed at hospital admittance, were analyzed as well.

Results: Out of all ischemic strokes in our clinic, 4.70% have been PIS. Motor deficits were the most frequent neurological signs (88.23%). Hypertension was the first incriminated risk factor (94.1%), followed by diabetes mellitus (29.41%), hypercholesterolemia (28.29%) and atrial fibrillation (23.52%). In 17.64% it has been noticed early hypodense brain lesions. Early worsening of the neurological status was present at 47.05% and the rest of 52.94% had a late worsening.

Conclusions: The progression of the ischemic strokes is a multifactorial, dynamic process, probably triggered by intracerebral events, followed by systemic events.

Disclosure: Nothing to disclose
EP2209

Lipoprotein-associated phospholipase A2 as a vascular-specific inflammatory enzyme related to plaque vulnerability is an independent predictive marker of ischaemic stroke and coronary heart disease

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Introduction: Inflammation plays an important role in plaque vulnerability. Lipoprotein-associated phospholipase A2 (LP-PLA2) hydrolyses oxidized phospholipids (generated in atherosclerotic plaques by oxidative stress) and produces lyso-phosphatidylcholine and oxidized fatty acids which are strong attractants for monocytes and macrophages. The inflammatory progression reduces fibrotic cap of plaque and increases a rupture-proneness of plaque. The increased serum level of LP-PLA2 is a perspective predictive marker of ischaemic stroke and myocardial infarction.

Methods: Material consists of 436 subjects divided in four subgroups
1. Ischaemic stroke (IS) (n=171), mean age 69±11 years, men 52%.
2. Coronary artery disease (CAD) (n=87), mean age 70±9 years, men 32%.
3. Arterial hypertension (AH) (n= 124), mean age 60±10 years, men 46%, healthy controls (C) (n=56), mean age 47±13 years, men 55%.

In all subjects: Neurological and cardiological examinations, IS confirmed by CT/MRI, SPECT, battery of biochemical/haematological investigations, LP-PLA2 using ELISA, intima-media thickness (IMT) using ultrasonography by radio-frequency data analysis, augmentation index (Alx) and pulse wave velocity (PWV) using aplanation tonometry, Alx and PWV are indices of arterial stiffness. Statistical software STATISTICA Base Cz Version10, Kruskal-Wallis test, linear regression and Pearson correlation coefficient.

Results: The study showed statistically higher values all followed parameters in IS, CAD, AH comparing to C (p<0.01-0.0001). Close correlation between LP-PLA2 and IMT, arterial stiffness was documented in all followed groups.

Conclusions: Our results documented significant changes in LP-PLA2,IMT and arterial stiffness in all followed groups (IS, CAD, AH) comparing to C. The measurement of LP-PLA2, IMT and arterial stiffness are very useful parameters for assessing cerebrovascular and cardiovascular risk. They represent significant prognostic power to ascertain subjects with increased risk for onset of cerebrovascular and cardiovascular events.

Disclosure: Nothing to disclose

EP2210

Tumour mimic due to an anatomical variant - bilateral anterior cerebral artery and Heubner’s artery territory infarction

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Introduction: Bihemispherical lesions that infiltrate the corpus callosum and enhance with Gadolinium generally have a poor prognosis. Butterfly glioma and central nervous system lymphoma are the two most common causes that can present with this type of lesions on MR-imaging. However, more benign options should not be ignored.

Case report: A 56-year-old woman presented at the Emergency Room with an acute abulia and aimless, involuntary movements of the right arm. After this abated somewhat, she displayed a persisting change of personality; she remained indifferent and detached. MR FLAIR T2 sequences of the brain showed bilateral hyperintensity in the genu of the corpus callosum, caudate nuclei, anterior limb of the internal capsule, lentiform nuclei; with strong enhancement on T1 sequences after intravenous injection of Gadolinium.

Methods: Additional diffusion-weighted images were suggestive of recent ischemia. CTA of the circle of Willis demonstrated an anatomical variant - the right A1 segment was absent. Follow up MRI after one month showed lessening of Gadolinium enhancement.

Conclusions: Bilateral ACA and Heubner’s artery infarction can appear as a tumour on MR imaging. In patients who present with an acute onset of symptoms, a vascular etiology should be considered. In these cases CTA or MRA can demonstrate the absence or hypoplasia of one A1 segment as an anatomical variant.

Disclosure: Nothing to disclose
EP2211
Abstract withdrawn

EP2212
The evaluation of aggregation activity of platelets in patients with ischemic stroke after i.v. thrombolysis and its influence on reocclusion after successful recanalization therapy

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Introduction: The i.v. thrombolysis is effective in selected patients with acute ischemic stroke (IS). However there is a high percent of reocclusion after successful recanalization therapy. The mechanisms of reocclusion and ways for its prevention are unclear.

Methods: 60 patients (42 males; age 61±11 years, mean NIHSS 14±4) with IS were treated with i.v. rt-PA thrombolysis according to ESO recommendations. The recanalization and early reocclusion rates documented by MR angiography were 51.7% and 22.6%. 60 patients with IS (39 males; age 64±12 years; mean NIHSS 12±4) were included in the control group. We evaluated the ADP-induced (ADP-A) and adrenalin-induced (Adr-A) platelets’ aggregation 24 hours after stroke onset.

Results: The ADP-A and Adr-A in patients after i.v. thrombolysis were 39.6±8.8% and 42.6±11.5%, greater than in patients of control group where ADP-A and Adr-A were 29.1±9.7% and 28.5±12.3% (p<0.05). The ADP-A and Adr-A in patients after i.v. thrombolysis with reocclusion were 45.9±10.5% and 47.1±14.2%, greater than in patients without reocclusion where ADP-A and Adr-A were 36.2±9.3% and 39.5±10.2% (p<0.05).

Conclusions: We demonstrated the increase of platelets’ aggregation activity in patients after i.v. thrombolysis compared to control as well as in patients with reocclusion compared to those without reocclusion after i.v. thrombolysis. This may reflect the platelets’ activation after thrombolysis. Additional data is needed to prove the necessity for antiplatelet therapy earlier than 24 hours after i.v. thrombolysis.

Disclosure: Nothing to disclose
EP2213
Endovascular treatment of acute basilar artery occlusion
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Introduction: The most effective therapeutic approach in management of acute basilar artery occlusion (BAO) has not been established yet. The aim was to evaluate safety and efficacy of multimodal endovascular treatment (EVT) of acute BAO, including bridging therapy (intravenous thrombolysis [IVT] with subsequent EVT).

Methods: In the retrospective study, the set consisted of 62 BAO patients (46 males; mean age 58.7±12.5 years) with radiologically confirmed BAO. Following data was collected: baseline characteristics, risk factors, pre-event antithrombotic treatment, neurological deficit at time of treatment, time to therapy, recanalization rate (with successful recanalization defined as Thrombolysis in Cerebral Infarction score 2-3), post-treatment imaging findings. 30-day and 90-day outcome was assessed using modified Rankin scale (mRS) with good clinical outcome defined as 0-3 points.

Results: Successful recanalization was achieved in 91.9% patients. Stepwise binary logistic regression analysis identified presence of arterial hypertension (OR=0.121, 95% CI: 0.028-0.531; p=0.005) and treatment type - the use of bridging therapy (OR=6.64, 95% CI: 1.56-28.1; p=0.01) as significant independent predictors of good 30-day outcome and, time from symptoms onset to treatment (OR=0.714, 95% CI: 0.543-0.939; p=0.016) as significant independent predictor of good 90-day outcome.

Conclusions: Data in this series showed that multimodal EVT was an effective recanalization method of acute BAO. Bridging therapy was associated with better 30-day clinical outcome. EVT should be started as soon as possible after IVT and not considered only as a rescue strategy. Supported by IGA MH CR grants NT/11046-6/2010, NT/11386-5/2010, NT/13498-4/2012, and by the grant project CZ.1.05/2.1.00/01.0030.

Disclosure: Nothing to disclose

EP2214
Symptomatic cerebral fat embolism - long-term follow-up
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Introduction: Symptomatic cerebral fat embolism (CFE) is a rare complication after traumatic injury or orthopaedic surgery. It may present with an altered state of consciousness, various neurological signs and is diagnostically challenging. Data about long-term follow-up are missing so far.

Methods: After identifying nine patients with CFE in the medical records and revising the clinical signs and the diagnostic process we performed a telephone interview targeting clinical course after discharge, neurological impairment and quality of life at present, using the Barthel Index.

Results: All nine patients showed severe neurological deficits in the beginning, including disturbance of consciousness from somnolence to coma. On follow-up three to 58 month after the insult two patients had died. The other patients had either recovered completely or showed only minor neurological deficits after rehabilitation even in cases with initial coma. They were nearly independent in daily life and needed only minimal assistance.

Conclusions: The prognosis of CFE can vary. Most patients had a good outcome after long-term follow-up. We conclude that patients with an unexplained coma after traumatic injury or orthopaedic surgery a diffusion-weighted MRI needs to be performed to find the pattern of disseminated hyperintense lesions in the white matter that are associated with CFE.

Disclosure: Nothing to disclose
EP2215

Intracranial varicella zoster virus vasculopathy manifesting as transient ischaemic attacks

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Introduction: Intracranial vasculopathy associated with varicella zoster virus (VZV) infection is a rare cause of transient ischaemic attacks (TIA's) after Ramsay-Hunt syndrome, with ipsilateral cerebral hemisphere most often affected.

Case report: A 62-year-old male without vascular risk factors had Waldenstrom macroglobulinemia since 2010. In February 2013, while being on chemotherapy, he developed a left Ramsay-Hunt, treated with acyclovir. In August 2013, he presented repeated transient episodes of right motor deficits over 2 weeks, suggestive of TIA’s. Brain DWI MRI revealed several acute ischemic lesions in the left middle cerebral artery (MCA) territory (small cortical frontal and parietal lesions, lenticular and caudate nucleus); angio-MRI showed a severe proximal left MCA stenosis; blood analysis, cervical vessels ultrasonography, 24h Holter, transthoracic echocardiography, HIV, hepatitis and syphilis serologies were normal; CSF revealed 6 cells/mm³ and PCR for VZV was positive. The patient was treated with acyclovir (10mg/Kg/day) for 21 days and prednisolone (1mg/Kg/day) for 5 days. He had no new focal signs during admission. He was discharged on valaciclovir, and had no further symptoms during the follow-up. Serial assessments with transcranial Doppler and repeated angio-MRI showed maintenance of MCA stenosis. Repeated CSF analysis showed 2 cells/mm³ and PCR for VZV was negative.

Conclusions: VZV vasculopathy is rare but must be considered in the differential diagnosis of patients with cerebral vascular events, particularly if immuno-compromised. This case of VZV vasculopathy has also the particularity of involvement of a large intracranial artery, while involvement of both large and small arteries is the most common presentation.

Disclosure: Nothing to disclose