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10. Sailersymposium, 18.–19. Juni 2015, Graz - Abstracts

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„Gerinnungssymposium für Innere Medizin und Laboratoriumsmedizin“

mit Workshops der Vereinigung der Primärärzte und ärztlichen Direktoren des Landes Steiermark

in Zusammenarbeit mit:

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Abstracts in alphabetischer Reihenfolge (nach Erstautoren)

Survival in Giant Cell Arteritis – A Cohort Based Analysis

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Introduction Giant cell arteritis (GCA) is a chronic autoimmune disease of the elderly affecting large and medium sized arteries. GCA is characterized by local inflammation of the arteries and systemic inflammatory response. Current research is not in agreement on whether GCA affects the mortality outcomes for patients, with some studies finding a higher mortality rate and some no change. There is some indication that GCA does increase aortic aneurysm and dissection related mortality.

The current study is a retrospective analysis of a cohort of CGA patients, aiming to assess the mortality outcomes for this group and any connection between aortic aneurysms and mortality.

Materials and Methods Records for 177 patients diagnosed with GCA at the division of Angiology at the Medical University of Graz between 1995 and 2012 were collected. The final patient group was 158 after exclusions. Data was collected for diagnostic factors, risk factors, adverse events, adjuvant therapies and survival.

Results The study population consisted of 112 (70.9%) females and 46 (29.1%) males. Average age of diagnosis was 71.4 ± 9.5 years. 62.7% patients were diagnosed with GCA alone and 37.3% had GCA + PMR. Of the 158 patients, 38 patients died (24.1%). The crude mortality rate in the patient group is 1.41% per year. The mean age at death was 80.53 (SD ± 6.7). There was a higher occurrence of death in the patients with cranial manifestation of GCA versus those with PMR ($p = 0.046$). Patients with an older age at diagnosis appeared to have poorer mortality outcomes ($p = 0.023$). Furthermore, a history of stroke, ischaemic opticus neuropathy and other ocular manifestations of GCA were associated with a higher mortality ($p = 0.001$, $p < 0.001$, $p < 0.001$). Patients taking statins had better survival than those who were not ($p = 0.017$). A positive biopsy was associated with poorer outcomes than a negative one ($p = 0.001$). There were no reported incidences of aortic aneurysm as cause of death.

Conclusion This study revealed several factors to be associated with mortality in GCA: older age at diagnosis, cranial manifestation of GCA, presence of ocular manifestations, history of stroke, lack of use of statins and a positive biopsy result. The results are comparable to the literature analysed and represent useful information to inform the clinician on particular patient types that need closer medical monitoring and are at higher risk for complications.

The AST/ALT (De-Ritis) Ratio: A novel marker for critical limb ischemia in peripheral arterial occlusive disease Patients

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Background The AST/ALT (De-Ritis) Ratio (AAR) is an easily applicable blood test. An elevated AAR on the one hand has been associated with an increase in non-alcoholic fatty liver disease (NAFLD). NAFLD on the other hand is associated with an increase in cardiovascular disease, all cause mortality and diabetes. As the AAR is also elevated in case of muscular damage, we investigated AAR and its association with critical limb ischemia in peripheral arterial occlusive disease (PAOD) patients.

Methods and Findings We evaluated 1782 PAOD patients treated at our institution from 2005 to 2010. Patients with chronic alcohol consumption (> 20 grams/day) were excluded. AAR was calculated and the cohort was categorized into tertiles according to the AAR. An optimal cut-off value for the continuous AAR was calculated by applying a receiver operating curve analysis to discriminate between CLI and non-CLI.

In our cohort occurrence of CLI significantly increased with an elevation in AAR. As an optimal cut-off value, an AAR of 1.67 was iden-

tified. Two groups were categorized, one containing 1385 patients (AAR < 1.67) and a second group with 397 patients (AAR > 1.67). CLI was more frequent in AAR > 1.67 patients (166 [41.9%]) compared to AAR < 1.67 patients (329 [23.8%]) ($p < 0.001$), as was prior myocardial infarction (28 [7.1%] vs 54 [3.9%], $p = 0.01$).

Regarding inflammatory parameters, C-reactive protein (median 8.1 mg/l [2.9–28.23] vs median 4.3 mg/l [2.0–11.5]) and fibrinogen (median 427.5 mg/dl [344.25–530.0] vs 388.0 mg/dl [327.0–493.0]) also significantly differed in the two patient groups (both $p < 0.001$). Finally, an AAR > 1.67 was associated with an OR of 2.0 (95 % CI 1.7–2.3) for CLI even after adjustment for other well-established vascular risk factors.

Conclusions An increased AAR is significantly associated with patients at high risk for CLI and other cardiovascular endpoints. The AAR is a broadly available and cheap marker, which might be useful to highlight patients at high risk for vascular endpoints.

Venous Thromboembolism during Concomitant Presence of acquired Inhibitor to Coagulation Factor V – A case Report

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We report a case of a 55-year-old male who developed a deep vein thrombosis in the presence of an idiopathically acquired, asymptomatic factor V inhibitor (2.65 Bethesda Units; factor V activity < 8%) which was diagnosed 7 months before during a routine preoperative coagulation testing.

The patient was successfully treated with steroids regaining a factor V activity of 42% within 8 weeks. After 7 months of treatment the patient developed a deep vein thrombosis in the right femoral vein as well as a pulmonary embolism. Coagulation testing showed again a reduced FV activity of 21%, a titer of 1.08 Bethesda units and a prothrombin time-international normalized ratio of 1.99.

These findings clearly indicate that even if substantial pro-coagulant inhibition exists, thromboembolic events must be considered in such patients.

Eukaryotic Translation Initiation Factors in Gastroenteropancreatic Neuroendocrine Tumors – A TMA based Study

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Eukaryotic translation initiation factors (eIFs) are mediators of start codon recognition in eukaryotic cells. If mutated, they might alter cell growth and cell proliferation, and thus contribute to carcinogenesis.

In the course of the neuroendocrine tumor- (NET-) study project at the Medical University of Graz, we have analyzed various eIF subunits in gastroenteropancreatic (GEP-) NETs in a tissue microarray- (TMA-) based immunohistochemical analysis. To the best of our knowledge, not much is yet known about eIFs in NETs in literature, and thus we aimed to gain knowledge about their expression patterns.

We have analyzed several eIF subunits in NET- and neuroendocrine carcinoma- (NEC-) samples by assessing the cytoplasmic expression intensity (score 0, 1+, 2+ or 3+), and the density of cells featuring a positive expression (%). As healthy controls, we have analyzed eIFs in the corresponding normal tissues (e.g. normal exocrine or endocrine pancreatic tissue).

Furthermore we correlated the patients‘ prognosis, e.g. time-to-relapse and overall-survival, with eIF expression levels. Thereby we aim to assess whether eIFs may be useful as future disease biomarkers.

Our analysis shows that eIFs are obvilously de-regulated in NETs and NECs, as compared to healthy controls. Interestingly, some eIF subunits seem to be up-regulated in tumor tissue, whilst others are down-regulated in tumors, compared to the normal tissue samples.

As conclusion, eIFs display altered expression patterns in NETs and NECs. Thus most likely differential expression of eIFs in NETs influences NET- and NEC-tumorigenesis.

In future eIFs may be potentially useful as prognostic biomarkers, and they might also serve as therapeutic targets.

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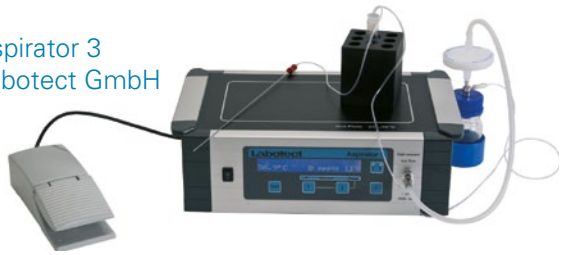

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