

rather than to specific psychiatric conditions. There were no or only small associations between individuals' general psychopathology and their siblings' cardiometabolic complications, suggesting that the associations were not attributable to genetic or environmental confounding factors shared within families. The same pattern was evident for the specific internalizing and psychotic factors.

Conclusions: Individuals with mental disorders in early life had an increased long term risk of cardiometabolic complications, which appeared attributable to a general liability toward psychopathology. Sibling analyses suggested that the elevated risk could not be attributed to confounds shared within families. This highlights the importance of transdiagnostic and lifestyle based interventions to reduce the risk of cardiometabolic complications, particularly in patients with several mental disorders.

Disclosure of Interest: None Declared

O0023

Course of untreated depression in people with tuberculosis in Ethiopia: a cohort study

F. A. Getahun

School of Public Health, Bahir Dar University, Bahir Dar, Ethiopia
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Introduction: Co-morbid depression is common in people with tuberculosis (TB) but little is known about the course over time.

Objectives: Our objectives were to determine the level of remission and factors associated with failure to remit in depressive symptom scores in people with TB undergoing treatment in primary care facilities in Ethiopia.

Methods: We assessed 648 people with newly diagnosed TB for depressive symptoms using Patient Health Questionnaire (PHQ-9) at the time of starting anti-TB medication, and again at two and six months. Remission was defined as more than 50% reduction in baseline depressive symptom scores. We analyzed factors associated with failure of depressive symptom score to remit at the end of the follow up using multilevel mixed-effects logistic regression by taking individuals as nested within 14 health institutions. Adjustment was made for socio-demographic characteristics, baseline depression score, stigma, type of TB, outcome of TB treatment, perceived severity of TB, substance use, perceived social support, substance use, and HIV status.

Results: Compared to the baseline, the mean PHQ-9 scores declined at two months (Hedge's G: 0.82; 95%CI: 0.71, 0.94) and six months (Hedge's G: 1.20; 95%CI: 1.08, 1.33). However, depressive symptom scores failed to remit in 176 (33.1%) of the 532 people with TB who completed the follow up. Stigma (AOR: 2.23; 95%CI: 1.09, 4.55), older age (AOR: 2.2; 95%CI: 1.13, 4.29), and treatment completion without a bacteriological proof of cure (treatment complete as compared to treatment cure) (AOR: 2.47; 95%CI: 1.37, 4.48) were independent predictors of failure of depressive symptom score to remit. Surprisingly, baseline lower depressive symptom score was more persistent than higher baseline depressive symptom score (AOR: 2.93; 95%CI: 1.56, 5.47).

Conclusions: In one-third of people with newly diagnosed TB, baseline depressive symptom scores did not remit after full course of TB treatment. TB treatment guidelines require in-built mental health component. Studies are required to understand course of depression beyond six months and effective interventions in this population.

Disclosure of Interest: None Declared

O0024

Preliminary study on changes in BDNF in patients with depression after percutaneous coronary intervention

S. Medved^{1*}, S. Sović², L. Ganoci³, N. Božina^{3,4}, M. Šagud^{1,4}, J. Bulum^{4,5} and A. Mihaljević-Peješ¹

¹Department of Psychiatry and Psychological Medicine, University Hospital Centre Zagreb; ²Department of Medical Statistics, Epidemiology and Medical Informatics, "Andrija Štampar" School of Public Health; ³Department of Laboratory Diagnostics, University Hospital Centre Zagreb; ⁴School of Medicine University of Zagreb and ⁵Department of Cardiovascular Diseases, University Hospital Centre Zagreb, Zagreb, Croatia

*Corresponding author.

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Introduction: Depression and coronary artery disease (CAD) are often comorbid conditions. The presence of depression significantly interferes with the recovery after CAD therapy intervention, such as percutaneous coronary intervention (PCI), one of the most common medical procedures in developed countries. Brain derived neurotrophic factor (BDNF) has a major role in angiogenesis and neuromodulation. Its levels were previously shown to be reduced in patients with depression, and latest studies indicate similar in patients with CAD. However, the correlation of BDNF levels and depression after CAD treatment is unknown.

Objectives: The aim of this preliminary study is to assess the changes in BDNF levels in patients with depressive symptoms during a six-month period upon PCI.

Methods: Antidepressant-free participants that underwent PCI with stent placement due to myocardial infarction or angina pectoris were enrolled in the study. Depressive symptoms were evaluated at baseline using the Beck's Depression Inventory II (BDI-II) with a cut-off score ≥ 20 indicating moderate depression. Serum BDNF levels were measured from blood samples drawn a day after (baseline) and six-months upon a successful PCI without complications. The t-test for dependent samples was used with marked significant differences at $p < 0,05$.

Results: Altogether, 76 participants were included in the study, of which 25 finished a six-month follow-up. Participants with $BDI-II \geq 20$ at baseline had higher serum BDNF levels in the second measurement ($M=23,12$, $SD=6,20$; $M=32,02$, $SD=12,26$, respectively). No significant difference was found in serum BDNF levels in measurements between participants with and without depressive symptoms ($t=0,33$, $p=0,74$; $t=-1,40$, $p=0,18$, respectively). Statistically significant difference was found between serum BDNF in the first and second measurement in the overall sample ($t=-2,28$, $p=0,03$) and in participants with baseline moderate depressive symptoms ($t=-2,46$, $p=0,03$), but not in those without ($t=-0,59$, $p=0,57$).

Conclusions: Serum BDNF levels in participants with baseline moderate depressive symptoms increased after a six-month period upon successful PCI treatment, whereas that trend was not observed in participants without depressive symptoms. This highlights the potential synergistic role of BDNF in comorbid depression and CAD.

Disclosure of Interest: None Declared