thickness of the lower torso prevailed over the upper torso. The anatomical parts were made on cadaveric material belonging to the same person, in one case we noticed the obvious difference in how right branching nerve is located on the left side of the face, and in the second case differences were not very pronounced. The results show that the distance between the primary trunks between 10 and 70°, the extent of the upper torso (temporofacial) is between 30 and 130°, the extent of the lower torso is between 25 and 70°, the angles of the branches of the upper torso are between 10° and 70°, lower torso angles between branches vary between 20 and 40°.

Conclusions: (1) The variability of the extracranial portion of the facial nerve branches falls into a wide range of options. (2) Predominant after branching options -fork in primary trunks and cervicofacial temporofacial after long-stem of medium length, as thick - temporofacial trunk are after Ferry - upward. (3) According to surgical anatomical maps, distances between primary trunks between 10 and 70°, the extent of the upper torso (temporofacial) is between 30 and 130°, the extent of the lower torso is between 25 and 70°, the angles of the branches of the upper torso are between 10° and 70°, the angles of the lower trunk branches vary between 20 and 40°.

43. ANGIOGENESIS OF ATHEROSCLEROTIC PLAQUES IN PATIENTS WITH METABOLIC SYNDROME

Munteanu Andrei, Cazacu Eugen

Scientific advisor: **Zota Ieremia** - M.D., Ph.D, Professor, Academician, Department of Pathological Anatomy, State University of Medicine and Pharmacy „N. Testemitanu", Chisinau, Republic of Moldova; **Raica M.–** M.D., Ph.D. Professor, The Victor Babes University of Medicine and Pharmacy, Director of the Angiogenesis Research Center, Timisoara, Romania

Introduction: Numerous studies have demonstrated that endothelial damage is a precursory symptom of atherosclerosis, which leads to an increase of vascular permeability, activation of mast cells and migration of leukocytes, lymphocytes, macrophages, adhesion of platelets, proliferation of vascular smooth muscle cells and eventual vasospasm and pro-inflammatory condition. All of the above listed components can be rightf-ully considered active pathogenetic participants in atherosclerosis and a result of aggregation of all risk factors that accompany a wide variety of cardiovascular diseases, such as coronary heart disease, hypertension, diabetes, dyslipidemia, etc. The influx of monocytes and mast cells during the early stages of atherosclerosis leads to the most pronounced manifestations of vascular inflammation, especially in patients with metabolic disorders. Angiogenesis is a very important pathogenetic element of atherosclerosis in stages of complicated plaques, along with mast cells and macrophages.

CD-105 is a sensitive marker of newly formed endothelial cells, an effective index of activation and proliferation of microvessels, not only in aggressive forms of cancer, but also in atherosclerotic plaques of the affected vessels. The plaque neovascularization process often begins in intima, progresses and leads to further destabilization of atherosclerotic plaques (intramural hemorrhage, ruptures etc.). Also, anti-MCT (mast cell tryptase) and CD-68 demonstrate clearly the important pathogenetic stages and patterns of atherosclerosis development and its complications in patients with metabolic disorders.

Purpose and Objectives: In our study, we analyzed the histotopographic distribution of newly formed blood vessels as a feature of angiogenesis, the extent of mast cell degranulation, the expression of macrophages in different types of plaques, as well as various arterial vessels in patients with atherosclerosis and metabolic syndrome, complicated by atherosclerosis. We have tried to analyze the importance of mast cells and macrophages, the patterns of development of atherosclerosis stages, along with diagnostic and prognostic features.

Materials and Methods: The study included 34 patients, who died of atherosclerosis (no. =17) and atherosclerotic complications of metabolic syndrome (no. =17). Fragments of their cerebral (middle cerebral arteries), carotid, coronary arteries, aorta (thoracic and abdominal segments), renal, iliac and vertebral arteries were collected for research at autopsy. The fragments

were processed using standard techniques. The type definition of plaques was based on morphological classification, as well as on macroscopic and histological images of hematoxylin-eosin stained sections and on histochemical methods – silver and orcein impregnation. To determine the expression of mast cells in the affected vessels, we have used anti-MCT immunohistochemical stain. Macrophages were identified using the CD-68 specific marker and the newly formed vessels – respectively, by using CD-105 (Endoglin), which is specific.

Results and conclusion: The evaluation of the results was based on determining the density and intensity of the final reaction, reflected in the quantitative ratio of different zones of atheromatous plaques. Positively stained mast cells, macrophages and newly formed vessels were found in many types of atherosclerotic plaques, especially in adventitia and in the immediate vicinity of plaques and in subendothelial layers.

We found a statistical correlation between the plaque type and clinical data.

The immunohistochemical method is effective for determining mast cells, macrophages, and newly formed vessels of atherosclerotic plaques, directly reflecting many important pathogenetic elements of atherogenesis in patients with metabolic syndrome.

CD-105 is a valuable marker of angiogenesis of atherosclerotic plaques, intimal arteries and adventitial vessels, an indicator of the degree of variation in the pathological development of atherosclerosis - the factors that may be important in introducing modern methods of research, diagnosis, treatment and prognosis of these diseases.

Keywords: Atherosclerosis, metabolic syndrome, angiogenesis, mast cell, macrophage, stability of atherosclerotic plaque, acute cardiovascular syndromes

44. CLINICAL AND GENETIC STUDY OF NEURODEGENERATIVE DISEASES

Robu Iurii

Scientific adviser: **Capcelea Svetlana**, Associate Professor, State University of Medicine and Pharmacy "Nicolae Testemiţanu", Republic of Moldova

Introduction: Huntington's disease (HD) is a neurodegenerative genetic disorder that affects muscle coordination and leads to cognitive decline and psychiatric problems. It typically becomes noticeable in mid-adult life. HD is the most common genetic cause of abnormal involuntary writhing movements called chorea, which is why the disease used to be called Huntington's chorea.

The purpose: The study of clinical, molecular and genetic aspects of Huntington's disease.

The objectives: (1) Evaluation of the molecular mechanisms involved in the pathogenesis of Huntington's disease. (2) Studying the phenomenon of penetrance and anticipation in Huntington's disease. (3) Determining the clinical and laboratory features of Huntington's chorea and differential diagnosis with other diseases neurogenerative. (4) Evaluation of the possibilities of genetic testing and genetic counseling in families with Huntington's disease.

Materials and methods: There were analyzed clinical data and genetic aspects of 10 patients (5 men, 5 women) diagnosed with chorea Huntington, hospitalized in IMSP Institutul de Neurologie şi Neurochirurgie in 2006 – 2012 period. The patients that were diagnosed with other neurodegenerative diseases were excluded from the study. Used methods: anamnesis; genealogical tree; neurological examination; laboratory tests (CT, MRI, Ecoencefalografy).

Results: Genetic study was partially achieved. Can be confirmed autosomal dominant inheritance in three families; noncomplete penetrance and anticipation in 2 families.

Conclusion: Trinucleotide expansion causes: onset of disease, evolution of the disease, severity of symptoms. Huntington disease is transmitted autosomal dominant: each affected person has a carrier of mutation that is symptomatic or asymptomatic, penetration of gene is dependent on the number of trinucleotide repeats, gene instability causes anticipation phenomenon. Molecular diagnosis can be useful for confirming a diagnosis, assessing prognosis and for presymptomatic diagnosis.

Keywords: chorea, anticipation, penetrance, genetic counseling