IMPORTANCE OF LIPOARABINOMANNAN (LAM) IN THE DIAGNOSTICS AND TREATMENT OF PATIENTS WITH TB / HIV

Gamaniuc M., Vinevschi A.
State University of Medicine and Pharmacy “Nicolae Testemițanu”, Republic of Moldova

Tuberculosis remains a global health problem. In this regard, further search of the effective approaches to diagnostics and treatment have high importance. WHO reports that each new case of untreated tuberculosis with active form of tuberculosis (TB) between 10 and 15 people annually. The most frequently used diagnostic test in low income countries is direct sputum microscopy, a technique that was developed in the 1880s and has remained unchanged. One of the main surface antigens of M. tuberculosis is lipoarabinomannan (LAM), which is a surface glycolipid of M. tuberculosis cell wall and can represent up to 15% of the total bacterial weight [2-4]. Detection of mycobacterial lipoarabinomannan antigen (LAM) in urine is considered to have potential prognostic importance in tuberculosis (TB). Urine testing would have advantages over basic sputum testing because urine is easily collected and stored and lacking risks associated with infection control during sputum collection. Compared to the Xpert MTB / RIF test, the LAM has a lower sensitivity [4]. The main advantages of the TB-LAM test are: it is easy to use and low cost, and results are available in 25 minutes. LAM-ELISA test of a previous generation with high sensitivity of the LAM-HIV co-infection in urine with low CD4 cells, has been studied. This establishment is different from traditional methods of tuberculosis diagnosis for HIV-infected patients [1]. WHO recommends LAM urine test for severe HIV-infected adults' diagnosis. The sensitivity of the test is high in people with decreased number of CD4 cells [5]. The purpose of this study was to analyse importance of lipoarabinomannan in diagnosis and treatment of TB / HIV infected patients with the further evaluation of LAM as a prognostic marker in medical practice.

Materials and Methods. MEDLINE database, Pub Med and HINARY were used as sources of information with reference to the lipoarabinomannan as a prognostic marker in TB / HIV diagnosis and treatment.

Results. Studies of LAM in urine during treatment of pulmonary tuberculosis have been included into analyzes. These studies demonstrated potential factors that affect the sensitivity of LAM in urine for the TB diagnosis and clinical symptoms of active TB form. Qualitative and quantitative estimates of LAM in urine may be useful in the future as biomarkers that reflect response to treatment of active TB. The LAM test is a promising diagnosis for HIV-infected persons who have a low number of CD4 cells and allows a quick diagnosis of active TB. Detection of mycobacterium antigens in the urine as a diagnostic test has several potential advantages compared to all currently used diagnostics, especially for people who have difficulties in sputum production as a biological material in detection of M. tuberculosis. Upon detection of pulmonary tuberculosis, LAM in a soluble form is metabolically active released from the bacterial and degraded cells [6]. LAM inhibits production of TNF-a and IL-12 by human macrophages and dendritic cells. Theoretically, the amount of LAM in the urine should reflect the bacterial load, metabolic activity and / or rate of bacteria degradation in this way allowing a semi-quantitative assessment of the infectious status and response to antibacterial treatment. The diagnosis of latent TB forms is based on the tuberculin skin test (TST) or on the interferon-γ release (IGRA); however, neither TST nor IGRA can help to identify patients with latent TB forms in active progression. TB-LAM testing sensitivity is 56% and specificity is 90% in identifying patients with profound immunosuppression with CD4 + T cells below or equal to 100 cells and detecting latent forms of pulmonary tuberculosis. In conclusion, we can mention the importance of
testing lipoarabinomannan in the urine as a prognostic marker in screening TB / HIV-infected individuals, persons with latent form of TB and progressing in the active form of TB with initiation of therapy as soon as possible with medicine against TB including cost effectiveness for patients’ clinical LAM investigation.

References.

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CLINICAL CASE OF COMBINED GENETIC PATHOLOGY IN A PATIENT

I.M. Sechenov First Moscow State Medical University, Russia

Familial hypercholesterolemia (FH) is a form of genetically determined increase of lipid levels in blood associated with a high risk of cardiovascular disease in young age. Disease is usually diagnosed only when the first symptoms of atherosclerosis appear, including acute cardiovascular disorders; that particularly complicates diagnosis and prevention in patients without clinical signs [1, 2]. Gilbert’s syndrome is a hereditary benign hyperbilirubinemia associated with a mutation in the UGT1A1 gene and a decrease in the functional activity of the enzyme uridine diphosphate glucuronidase (UGT) 1A1 in hepatocytes. The UGT gene is located in the long arm of chromosome 2 (q 37.2). More often there is an increase in the number of TA-repeats, which reduces the activity of the enzyme. The incidence of this syndrome in different regions of the world ranges from 3 to 10 % [3, 4]. Genetic testing of patients with familial hypercholesterolemia is not included in the routine examination, but it can be recommended to first-line relatives for timely diagnosis and assessment of the risk of cardiovascular diseases [5]. Demonstration of clinical observation of genetic combination of familial hypercholesterolemia and Gilbert’s syndrome in a young patient and analysis of possible protective role of increased bilirubin level in atherosclerosis progression in Gilbert’s syndrome.

Materials and Methods. This paper describes a clinical case of a combination of two genetic diseases in a 24-year-old male patient: familial hypercholesterolemia and Gilbert’s syndrome using analytical, logical, monographic methods, the results of anamnesis and objective examination of the patient, methods of laboratory, functional, ultrasound diagnosis and genetic testing.

Results. During genetic testing, a mutation R3500Q of apoB 100 gene was detected, which leads to a violation of low-density lipoprotein (LDL) binding, their accumulation