

IN TUBERCULOSIS IMMUNE VIOLATIONS LABORATORY OF DIAGNOSTICS MODERN OPPORTUNITIES

Tosheva Dilnoza Rakhmatovna

In the name of Abu Ali ibn Sina Bukhara state medicine institute, microbiology department assistant

Abstract: Last in years delete against of institutions immunological in laboratories modern diagnosis equipment more and more more appear be started it while before used outdated , ineffective and of the inferior ones according to of the body to infection against defense status about significant level reliable information get enable gives - informative techniques . An example of this phagocytes functional and metabolic activity determination for flow cytometry current reach can Current at the time only some in laboratories applied flow cytofluorimeter using of cells absorption ability evaluation short time inside one how many a thousand phagocytes determination enable gives , standard from technique use - microscope through phagocytosis assessment - attention to summarize possibility will give . From 100-200 cells a lot was not Evaluation same about to say can.

Key words: sil disease, microbiological studies quality, to medicine endurance, a lot to medicines resistant tube, wide spread out to medicines resistant tuberculosis.

of phagocytes bactericide ability - of the cell oxidation potential analysis to do Before this on purpose wide used NBT test (nitroblue tetrazolium reduction test) is subjective determination and one more than 100-200 in the sample cells in the calculation difficulty with is described . From this in addition to this ineffective from technique used without , only of neutrophils functional activity determination possible was because in the blood this cells population all 65% of leukocytes organize does ; monocytes , their small the number due to , has not been studied .

Materials and methods: Oxidation return of phagocytes absorption ability and their inflammation to the place transition ability foreign in hand flow cytometry using learning more and more wide spread out clinical application is finding because it is above telling passed to shortcomings have it's not . Last in years this studies some local laboratories of practice one to the part circle started Delete diagnose the most modern methods one done increase for quantiferon test , now mainly used TB GOLD , but this the test flow to cytofluorimetry to adapt attempts already there is . Other cytokines also flow cytometry with more and more more analysis is being done.

close until time peripheral blood of lymphocytes subpopulation content evaluation monoclonal antibodies (mAbs) and fluorescent microscope using done increased Last in years laboratory in practice labeled cells the number measure tool as flow cytofluorimetry more and more wide spread of lymphocytes subpopulation content determination for work producers very a lot in quantity mAbs and their different different fluorochromes with compounds work they issue . Flow cytometry using immunity situation evaluation already many in laboratories regularly of tests one to the part became This of the method to the microscope relatively no doubt the advantage is that sample analysis to do speed and operator microscope using count possible has been number with in comparison device by be counted possible has been cells number

significant level abundance The result of this cell subpopulations better determination and analysis of doing right is the result. From this besides , the microscope using cells one of time in itself one how many fluorescent paints with to paint possible not , flow cytometry using immunocompetent cells analysis in doing each one cell for nine , ten two or even ten six fluorochrome with to paint permission is given

In phthisiatrics wide applied another immunological method – Diaskin test. It's a lot from since known , but only last in years of immunity humorous factors study , in particular , cytokines degree determination for wide apply started Short time inside dozen test past test systems work released

In general when , delete in illness immunity violations of learning diagnostic opportunities present at the beginning of the 20th century and in the middle of immunologists from potential significant level increases , this Undoubtedly this science in the field of scientists last in achievements important role played

Tuberculosis immune system answer : Last in years take went studies us tuberculosis the disease development risk determiner main factors between hereditary important role plays said to the conclusion take will come . These are the features with depends

of phagocytes cell inside cleaning system performance , mucus of floors humorous resistance factors in the system defects with , in pathogens carbohydrates recognize get for in charge has been lectin in the structure changes with . Apparently as sexual hormones level such as hereditary infection is also a factor known role plays because tuberculosis disease to women than in men more occurs . Infection source with contact to do term important important have

Pathogen with in touch has been everyone is sick lack of development defined , new infected in man tuberculosis the disease development danger from infection next the first 2 years inside about 2 % organize does Man of the body many in the tissues tuberculosis to infection natural resistance there is . Micro - and of macroorganisms mutually of influence result a lot in terms of the patient of the body immunological tolerance assumed to be related will be done , therefore for tuberculosis to infection resistance formation processes to understand for the following account get need immunocompetent of cells quantitative and functional situation account get

Pathogen and immune system mutually effect cell and humorous in immunity to the reaction reason will be and of cells phagocyte activity also changes . Past from the 60s-70s of the century to the present until the day *Caused by M. tuberculosis emits infection in development phagocyte cells , lymphocytes and humorous of immunity to the role dedicated many studies take went* Interleukins (IL, interleukin) , that's it including tumor necrosis factor (TNF, tumor necrosis factor) , neutrophil elastase , cationic proteins , myeloperoxidase , nitrogen oxide (NO), reactive oxygen types and leukocytes level apoptosis is being studied .

Current at the time *M. tuberculosis* lower breath to the roads reached from going then , to infection against the first and main protection line alveolar macrophages the fact that common acceptance will be done . These cells bacteria phagocytosis through their increase straight away to suppress able , also cellular delete against of immunity wide scope in reactions participation - the antigen present swelling , inflammation T-lymphocytes in place accumulation encourage through and another reactions . Macrophages and mycobacteria between mutually effect two in the form to be can If the macrophage own duties do it contact if possible digestion to do and the pathogen no to do with ends However , this each always happens it won't be . Otherwise , the pathogen and host in the middle balance harvest has been hidden chopsticks transport happen will be C3, C5 dysfunction result to be can , as a result macrophage of granules genetic in terms of determined or acquired anomaly (phagocytes in granules peroxidase , acid phosphatase and another of enzymes absence) and of granules own secretion to phagosomes take out can't get Some of the authors to his opinion according to Mycobacterium tuberculosis cell of the wall high to acid endurance because of complete phagocytosis also occurs it won't be . In this case, mycobacteria to macrophages harm deliver and of phagocytosis to completion hindrance doer toxic glycolipids work release ability is also known role plays

for *M. tuberculosis* comfortable in the circumstances they are macrophage of lysosomes activities break it is possible while lysosomal of enzymes too much except activity take will come . Some of the authors to his opinion according to such in order not placed proteolysis alveolar of tissues no to be done take will come . Tuberculosis of the process to go macroorganism for comfortable when , alveolar macrophages by endocytosed of the material accumulation stops because cathepsin D itself lithic effect cause releases

Inflammatory T cells with contact to do through activated in macrophages and INF- γ secretion as a result this to the cells strong antibacterial features give one series biochemical processes starts . Macrophages and inflammatory T cells between mutually effect under conditions of , cell inside pathogens no who does proteolytic of enzymes guards has been lysosomes with bacteria held of phagosomes more efficient merger observed . Phagocytosis process oxygen explosion with passes : bactericidal to activity have has been oxygen radicals and NO 's harvest to be with TNF- α and INF- γ together stimulation conditions this process much more active . From this except , activated macrophages MHC class II molecules (main histocomposition complex) and TNF- α receptors expression increases this while to the process addition naïve T cells attraction to do take will come .

Tuberculosis triggers for macrophages " live place " . Phagocytosis as a result cell into entering , pathogens from both antibodies and cytotoxic T- lymphocytes is protected . From *M. tuberculosis* phagocytosis after macrophage endosomes inside preserved remains . Phagocytosis and of phagolysosomes biogeocenosis the pathogen take throw , re work and *obtained from M. tuberculosis antigens adaptive immune to the cells* present reach for necessary has been is a process . Phagosome harvest to be programmed phagolysosome maturity causes Ca²⁺ and organelles movement regulators small binder proteins around GTP family Rabs (guanosine triphosphates), activated of the cascade effector molecules , for example , lipid kinases , membrane synthesis apparatus and another molecules around manages

by *M. tuberculosis* Rab controlled membrane movement breaks and pathogen lysosomal to enzymes access opportunity non , but nutritious factors get ability have has been stage phagosome maturity prevention takes " The phagosome maturity so-called " stop " . this process their safe stay for very important Lysosomal of enzymes activity suppression through *M. tuberculosis* cell inside active increases and that's it with sharp contagious of the process for the reason becomes *M. tuberculosis to kill ability* genetic to resistance dependence and bcg gene with dependence determined . This locus by coded the main protein product macrophage protein Nramp 1 (natural resistance with depends macrophage protein of macrophages cell inside to pathogens natural resistance with depend In people Nramp 1 defect lungs tuberculosis with dependent. Of the trigger macrophages by phagocytosis SRA class receptors (stop receptors class , " collector class " receptors) with participation happen will be Killing later , only from 20-24 hours then , phagocytosis 6-12 hours after completion after happen will be

Many the authors of macrophages to *M. tuberculosis harmful effect about speaks that 's* it NO in the process and reactive oxygen types main importance they give to *M. tuberculosis* against efficient control to do NO produce for release it *is* necessary iNOS (inducible NO synthase) deficiency of mice to infection sensitivity increase with is confirmed . G. O. Kaminskaya (2004), OGKomissarova (2011) and others lungs of tuberculosis different in the forms of producing NO release they learned Own studies as a result this of work authors often producing NO in release changes about different different to information have shared , this is probably the results tuberculosis of the disease different forms and pathological of the process different stages received in groups received with depends to be can

With that together , another point of view look appear it has been . In particular , according to S. Nathan (2000). according to pathogens macrophages by of killing main mechanism oxygen and of nitrogen reactive to the forms depends it's not .

Results and discussion Phagocytosis of *M. tuberculosis* basically complement system proteins receptors

through happen will be and NADPH oxidase of phagocytes (nicotinamide adenine dinucleotide phosphate) is significant activation with together does not come ; this fact of researchers reactive of species irrelevant role about his opinion explanation can this to the pathogen against cytotoxic in effect. Monocytes and of macrophages many to research however , in the macroorganism the pathogen no to do mechanisms according to still a lot uncertainties available. To neutrophils As for , they are also bacteria with mutually effect does and them phagocytose . of neutrophils bacteria with mutually effect straight away recognize get (for example , TLR receptors (paid through receptors) , also opsonized bacteria Fcy receptors (FcyRI , FcyRII , FcyRIII) and complement receptors (CR1, CR3. Main receptors, phagocytosis provide FcyRIIA and CR3 receptors . This of receptors connection in the cell cell inside signaling of the ways activation take comes as a result phagosomes appear to be , their neutrophil granules with to merge and oxidizer to explode take will come .

of neutrophils bactericide effect reactive oxygen types and less level nitrogen work release as well neutrophil granules contained bactericide peptides and proteins release with is provided . Reactive oxygen types of neutrophils bactericide of activity main factors is considered They are neutrophils to himself special in granules harvest will be and NADPH oxidase and superoxide dismutase enzymes by harvest has been superoxide anion , hydrogen perox and hydroxyl radical , as well as myeloperoxidase under the influence of harvest has been hypochlorite and chloramines own into takes Nitrogen own into received oxidizer to substances strong have has been peroxy nitrite enters oxidizer properties and relatively long life

Macrophages such as neutrophils cell on the wall galactomannan and arabinomannan recognize pathogens with mutually effect does For them main to kill mechanism oxygen peroxide radicals work release , as well as neutrophils degranulation they did This is the process fast happen will be because 50% of pathogens are cells of incubation the first 2 hours inside dead will be Endosomal proteases the pathogen no to do in the process known role plays Pathogens to the wall harm deliver main molecule phagocytes of granules one part is a serine protease . Neutrophils different different to pathogens against of the body the first protection of the line important is a member . In tuberculosis inflammation in development of neutrophils to the role many the authors attention gave However , this in the field take went many to research despite the authors one to the idea did not come - neutrophils macroorganism tuberculosis to infection against standing up really necessary role does he play or tuberculosis of the disease inflammation during into their body tissues pathological fatal effect superiority will

Neutrophils and macrophages own functions perform requires MHC molecules for HLA molecules (human leukocytes antigen , human leukocyte with antigen). in the complex antigenic peptides recognize get for T-cell presented to the receptors CD4 and CD8 Phagocytosis of the process necessary stage is adhesion . Adhesion molecules of phagocytes inflammation to the place migration control does Foreign researchers CD11c and CD11b adhesion molecules to learn separately attention give local affairs while this cover takes. Last in years immunity according to researchers each different kind of pathogens who knows and of the body the first protection the line which provides TLRs to the group big attention they gave on the surface of *M. tuberculosis* major TLR ligands arabinomannan is recognized by TLR2 . From this In addition , TLR4 is also recognized in getting participation is enough Recognition of Arabinomannan phagocytosis strengthen through to microbes against protection which provides lungs surface active substance SP-A (surface active article protein) and SP- D surface active proteins with mutually effect to do through happen will be of *M. tuberculosis* Recognition by TLR2 and TLR 4 is both macrophage and dendritic cells by done is increased . Mast cells and epithelium cells also antigen in determining participation TLR receptors have also been found in them . This in cells of TLRs composition complete not expressed and their expression macrophages and dendritic to cells than lower . However , their TLR system through activation of neutrophils infection to the place to move help giving one series cytokines and of chemicals to release take will come . Platelets , epithelium cells , basophils and eosinophils such as another cells are also TLR

receptors through binding, neutrophil mediated by the answer strengthening, as well as defensins work release through to pathogens harmful effect show through *M. tuberculosis* from illness protection in doing participation reach can

Sharp contagious process conditions in the body of the pathogen spread hindrance doer forces there is and they are the first primarily CD4 inflammatory T cells with depend T- lymphocytes macrophages with together of the pathogen a person body with mutually under the influence of big role plays This kind of lymphocytes immunity reaction organize in reaching participation of macrophages activation, by "waiting" for the signal done is increased. Of them the first is $\text{INF-}\gamma$, inflammatory CD4 T cells by released is a cytokine. Macrophages activation for the second signal is surface $\text{TNF-}\alpha$ is inflammatory T cells macrophage in the membrane the immunogen from acquaintance after to the expression take will come.

T cells main role to play shown. of CD8 + T cells delete against in defense role is also discussed will be done. To *M. tuberculosis* in humans against immune Th1 cells in response of the pool formation *more note but* the participation of Th2 cells (P-helper-2-cell, T-helper 2) showing studies there is tuberculosis of the disease in pathogenesis.

Macrophages with mutually effect doer inflammatory T cells not only intramacrophage biochemical processes strengthens, perhaps themselves activates and to the antigen a lot edged immune of the answer organizers as works Cell inside bacteria with chronic infected macrophages T cells by activation ability loss can To the process new of macrophages massive the introduction TNF-0 (lymphotoxin) of pathogens and activated CD4 + T - inflammation of $\text{INF-}\gamma$ products of cells infected to the cells synergistic effect effect under happen will be. T-effectors except for macrophages they are also involved will be done. That's two method done is increased: first, induction through

IL-3 and granulocyte-macrophage colony warning factor under the influence of bone in the marrow of macrophages differentiation. Second, new harvest has been macrophages TNF-0 and macrophage chemotactic factor under the influence of blood from the flow infection to the place moved to pass they start and there they settle down and of macrophages migration reducing factor (MIF) effect from the head they forgive their mobility.

of $\text{TNF-}\alpha$ tuberculosis disease in the process role to learn now local and foreign researchers big attention is looking Some the authors even $\text{TNF-}\alpha$ inhibitors the first times from ordering before the patient tuberculosis the disease check need said to the conclusion they came because this cytokine work release decrease sick in people tuberculosis of the disease development probability increases. Antibodies to $\text{TNF-}\alpha$ second of the signal effect void does Cytotoxic T cells the antigen recognize received after immediately activates and molecular of the hardware purposeful cells apoptosis or necrosis through no to do readiness understands. From this different unlike, CD4 + inflammatory T cells macrophages on the surface the antigen recognize received then, macrophages activator mediators synthesis to do for one how many hour time spends In microvesicles collected new synthesis done cytokines T cells with contact to do in place to macrophages enters It is direct path, cytotoxic T- lymphocytes in the example that it was like, eng thrifty and functional in terms of is justified, because it is a neighbor, not infected to cells effect does not

Cytokines this combination is also immunocompetent of cells infection to the place come in to go which provides binder of tissues main structural parts has been of fibroblasts death effective for Apparently apparently immune the answer mobilization to do conditions of effector T- cells pool high level storage need Macrophage by activated inflammatory T cells to the antigen of specific T cells increase and differentiation encourage through IL-2 for addition effectors attraction does The pathogen clearly admitted after by activated CD4 work issued cytokines collection cell immune of the answer multidisciplinary development provides.

Obstacle members and tissues, cells and biological liquids system organize doer man organism nonspecific

resistance factors *Recognizing tuberculosis M to the reaction enters* later on to kill and from the body take to throw with . Efficiency nonspecific protection a lot to factors depend Macrophages , neutrophils granulocytes , monocytes and natural killer cells pathogens straight away to kill or cell inside bactericide substances (lysosomal cations , superoxide radicals , hydrolytic enzymes) active respectively release and / or phagocytosis , later phagocytosis the way with their increase ability suppression can Complement system , lysozyme , O- lysines and another one series components of the pathogen from the cell except digestion in being different level success with service does The above summary if we do , mucus floors on the surface pathogens efficient take to throw ability , as well as phagocytes intraphagosomal infectious to agents endurance tuberculosis of the disease main protection factors is said to be we can conclude .

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