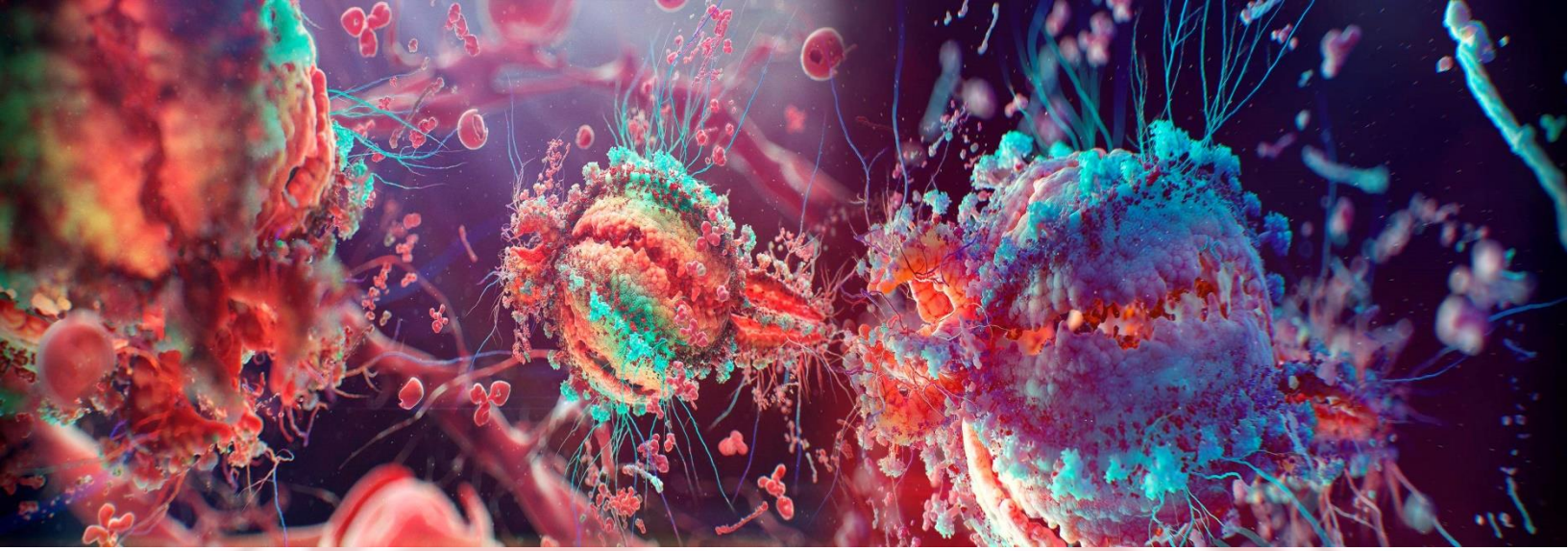

Immunology

Lecture (7)

Done by:
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Lecture 7

In this not I'm not gonna add the slides and I'm only gonna write whet the dr said about each slide

Inflammation and leukocyte migration

Leukocyte extravasation :

Neutrophils are found in circulation
While lymphocytes are found in lymph nodes
And could also be found in circulation but mostly in lymph nodes

First cells to reach the site of inflammation
1-neutrophils 2-macrophages 3-lymphocytes B then T

Endothelium is closed to prevent cells from leaving the circulation
So neutrophils have a special way to leave circulation

So neutrophils will leave the circulation by 4 steps (stages)

1- Rolling
2- Activation by chemoattractants
3- Arrest and adhesion
4- Migration

Normally , neutrophils are moving with the shearing force and they are not close to the endothelium but if inflammation happened they will get closer to the endothelium

And they will try to hold the selectin receptors but because of the shearing force the neutrophils won't stay bounded to the selectin receptors
So they are not tightly bound (rolling)

Next step we have chemoattractants that will activate the neutrophils
The activated neutrophils will express another type of receptors called integrins
So integrins will bind tightly to there Ligands on the endothelium

Finally PECAM-1 on neutrophils will bind with PRCAM-1 on the endothelium so the leukocyte will squeeze it self between two neighboring cells and go to the site of infection

The dr asked a question , ما هي شروط الي لازم تكون موجوده عشان تطلع ال نيوترفيل ؟
1-selectins
2-chemokines
3-integrins
4-PECAM-1

Then someone asked about the receptors and the dr said that we have two types of receptors

1- Receptors found on the surface of the cell
2- Receptors that are inside the cells (which means that thy are not expressed)

Selectins : some of them are on the surface and some are not expressed
Integrins: mostly are not expressed

What helps the receptors to be expressed is the chemokines

Lymphatics in inflammation:

What is the difference btw transudate , exudate and pus

Transudate : fluid with little proteins
Plasma

Exudate : fluid with proteins
Plasma with cells

Pus: fluid with proteins and cells and debris

The dr said that pus is the worst and transudate is better (easiest to deal with)

Function of inflammatory exudate

The dr read the slide and he talked about the last point
Fibrinogen is soluble and fibrin is insoluble
The main function of it is to stop the bleeding
And here it's important to prevent the microbe spreading

Inflammatory mediators

Cytokines are a lot some of them work in innate immunity and some in adaptive immunity
Usually the innate cytokines are called inflammatory cytokines

The adverse effect of cytokines is important

We can give anti -IL1 , anti-IL6 , anti-TNF to stop the adverse effects but we have to think twice cause inflammation is important to kill the microbe

TNF – apoptosis of the Tumor cells

Cytokines and inflammation the drug read the slide

Negative regulation of inflammation which means how to shut down inflammation

When the microbe is killed no more cytokines will be released

بالوضع الطبيعي لما ترتفع نسبه السيتوكينز بصير في عنا أفراز لل glucocorticoid
وهاي تعتبر anti inflammatory

Inflammation outcomes

The dr said that the best outcome in number 3 cause we have complete healing so the inflammatory response is successful

Then the next best outcome is repair and scarring

The dr gave an example
Someone got injured by msmarr 7aded
First day : First hour bleeding /vasoconstriction/platelets activation to stop bleeding
Neutrophils and cytokines
2^{nd} 3^{rd} hour : vascular permeability /transudate
Next day : swelling /redness / pain / thick fluid coming out (exudate)
1 week : puss and almost abscess formation/ fever local and systemic

12^{th} day : unconsciousness/fever /low pulse (systemic shock)
What to do?
IV antibiotic but it takes 24 hrs to work
Body fluids to raise his pressure
Anti inflammatory as anti histamine
If his heart stopped CPR

If the patient came at the first day : antibiotics and maybe pain killer

7^{th} day : surgery to remove the puss
Open and drainage every day morning(why do we have to remove the puss ? Cause it will become chronic and it's responsible for all symptoms , and the antibiotics can't make the puss lose we have to remove it)
And give antibiotics

Ttt for abscess :

What organisms Do we see in puss ? Staph / strep.pyogens /

Fistula formation: can only be removed by a surgery and it's a like a channel for the puss to get out

Last slide : NSAIDS AS IBUPROFEN AND VOLTAREN

GLUCOCORTICOIDS IN SPECIAL CASES ONLY

HUMORAL IMMUNITY

Slide 1
Adaptive immunity is 2 parts humoral (b cell) and cellular (T cell)

B lymphocytes could be found in circulation but most of its time in the lymph nodes

Major function not only secretion of antibodies but also APC (antigen presenting cell)

Next slide : t helper cell can't kill but it activates B cells

ال B cell هي احد أذرع خلايا تي ا، اتفقنا انه خلايا تي المساعدة ما بتقدر تقتل بس يتفعل البي ف لو البي مش موجوده كيف حتشتغل خلايا تي المساعدة

Why B cell is special for extracellular microbes and toxins ?

1- Cause it secrets Antibodies. And one of the antibodies is IgA which is capable of neutralisation
2- Cause it has MHC11 on its surface
3- Found free in circulation

If it's a carbohydrates the chance of binding B cell is higher
If protein it's hard for the B cell to see it

If endogenous microbes can't be seen by B cell unless it's presented by APC or its activated by t helper cells

So B cell deals with endogenous microbes

3rd point in the slide
Why microbes with capsule ? Cause the capsule is a polysaccharide

Next slide : B cell maturation
The dr read the slide and explained the picture in the next slide

Lymphoid stem cell : just came out of bone marrow and has no receptors
First Ig that's gonna be added to the lymphoid stem cell is Ig alpha and Ig beta so it's called pro B cell
Next step : genes rearrangement , we start to put heavy , light chain and joining chain so now it's called pre B cell
Then addition of VlJl to the light chain , now it's called immature B cell
Then Naïve B cell and when we add igD it's completely mature
If the naïve cells saw the ag activation
If it didn't see the ag it will die

Next slide
In the babies a group of B cells will bing to any ag so these are called innate like cells

So the fort version of the B cells when they secrets antibodies
These antibodies are weak

When baby gets older IgD increases and CD5 decreases

Next slide the dr read the schedule

Next slide : B cells clonal selection

Cross linking on igM May lead to cell death or allergy
In the case of allergy cells will not be killed but will be put aside

What is the importance of this process ?
If we have hyperactive BM as in cancer
Or in infection
BM will produce lots of lymphocytic fastly which are hyperactive so the selection process is important

Next slide : we have a B cell in circulation which is mature and still didn't react with an Ag yet , what happens when it sees an ag ? Recognition and it will bind to it

B cell may not see the ag
So it could be activated by t helper cell (here no direct binding btw B cell And the ag)

Look at the picture up
IgM
Ig alpha and beta for IgM signaling

Any receptor has 3 parts 1- external
2- part anchoring the membrane
4- Inner part signaling

As we see in the picture IgM has no inner part so it will use Ig alpha and beta for signaling

Two ways for B cell to work

A) T dependent : t helper cell saw an APC and took the ag from it then it called the B cells and gives it the ag by TCR and at the same time it will bring a receptor called CD4/CD40L
Both of them have to work at the same time and they will activate B cells

B) T independent : ag binds to receptor directly then produce a signal

And the signal will say to B cell that it has to produce antibodies