**Medical virology**

**Viral carditis**

In this lecture the doctor didn’t say many things out of the slides so this sheet just includes the comments that were added to each slides:

If a slide is not mentioned it means that the doctor read it all as it is without adding any comment about it.

**Slide 3:** in active myocarditis: Microscopic morphology of infarction due to ischemic myocardial disease (e.g. Occlusion of a coronary artery) is different from the active myocarditis.

Borderline myocarditis: less sever because necrosis isn’t involved.

**Slide 4:** Diphtheria cases usually presents in out breaks (when you diagnose a case expect to see another), in case of diphtheria we are mostly concerned of the toxin produced by it (not the local invasion of it) which will cause myocarditis.

 Diphtheria was known a long time ago and there are 3 biotypes (subspecies) that are recognized: *C. d. mitis*, *C. d. intermedius*, *C. d. gravis*,

Diphtheria can be isolated using Loeffler serium (for primary isolation ) then we use potassium tellurite agar which differentiate these 3 biotypes.

In 1873, doctors noticed a degenerative heart in some cadavers and observed indications of an inflammatory process. (Thus the term myocarditits was first introduced)

Biopsy through fine needle aspiration is an example of biopsy techniques that were not known before which allowed the sampling of human myocardial tissue.

**Slide 5:**  **This slide is not for memorization it is just to demonstrate that there has been a shift in the causative agent of viral myocarditis with time.**

In 1948 the most common cause of myocarditis was CVB and CVA especially **CVB**

In 1950 enterovirus e.g. poliovirus (entero: means gut)

**(**Extra information irrelevant to the subject: 1.enterotoxin is a toxin that affect our gut e.g. the one produced from staphylococcus aureus (exotoxin) 2.there is a difference between enterotoxins (exotoxins) and the endotoxin of gram – bacteria.**)**

In the nineties adenovirus

In 2000 HHV Type 6 and PVB type19

After that is HCV and EBV

EBV is part of the latent virus family which includes cytomegalovirus and herpes

Notice the shift from enteroviruses to non-enteroviruses as a causal agent of viral endocarditis.

Vaccination was one of the causes that lead to the shift e.g. the vaccine against poliovirus

Also cases of meningitis due to streptococcus pneumoniae and haemophilus influenza decreased due to vaccination (irrelevant but was mentioned)

**Slide7**: Doctor said that pathogenesis will be left to the pathology class (I doubt) and he only mentioned the following about this slide: Il-1, IL-6 and TNF cause fever.

**Slide8**: doctor focused on the fact that viral carditis occurs in 3 phases and phase 1 is the infectious phase, which lasts 7-10 days, and is characterized by active viral replication. The rest of the slide he said that it is not important to know

**Slide 12** : was skipped

**Slide13**: the Dr focused on the following viruses:

1. **Coxsackievirus** types A and B, **especially type B**, are the most common viral causes of myocarditis.

2. **Adenovirus** (types 2 and 5 most common) 3.**Cytomegalovirus**

4. **Echovirus** 5.Epstein**-Barr virus** 6. Poliomyelitis virus (enterovirus)

7. Rubella virus 8.Varicella -Zoster virus

Varicella-zoster virus is a latent virus, it, stays dormant in dorsal root ganglia, and when person is under stress (e.g. viral infection, common cold, long exposure to sunlight) virus will be reactivated and it will clinically manifest as shingles.

Also EBV is a latent virus with a dormant phase that can be reactivated

**Slide 15**: The early symptoms of the coxsackie -induced cardiac myopathy include some generalized viral symptoms-fever, fatigue, malaise-with the addition of chest pain**. Those are General constituent symptoms.**

As the virus enters the heart cells, the immune system attacks and damages both infected and normal heart cells, and that’s because the antibody raised here are against antigens in infected cell however the antibody doesn’t recognize infected cell from normal cells so it will damage both!

Some of the antibodies will be against normal cells antigen and will manifest unfortunately like an auto-immune disease!

There will be antibodies against coxaki viruse antigen and normal cardial cell antigen so infected and not infected cells will be affected!

Like in rheumatic fever caused by streptococcus pyogenes group A, where antigens of streptococcus pyogens are similar to some antigens expressed on myocardial cells and other cells leading to the production of antibodies that can react with both antigens of the pathogen and other normal cells.

I think that the dr. was talking about cross reactivity!

**Slide17:** CBC includes WBC count, RBC count and platelets with the differential count for each WBC (eosinophils basophils etc…)

In bacterial infection we have neutrophilia and in viral infection lymphocytosis

Blood culture are usually only done to detect bacterial and fungal infection!

To detect increased titer we must take 2 samples at different time interval, if we detected at least a 4 fold increase it means acute infections.

**Slide18:** Creatine kinase is usually produced by all muscle cells eg. skeletal muscles but we have other isoenzymes like CKMB (primary source of **CKMB** is myocardium, although it is also found in skeletal muscle.)

**Total** -all isoenzymes- Creatinin concentration is not a good biomarker because if patient have issues in other places of body (skeletal muscles) it could be elevated (not specific )

Troponin T and Troponin I are more specific

ESR is Erythrocyte sedimentation rate; usually high during infection but sometimes as in this case it is not significant

**Slide 20** irrelevant note: Granulomatous diseases could be caused by non-functional WBC

Nitro blue tinitroblue tetrazolium test we use this test to test the function ,after isolating the buffy coat (WBC) we activate the cells and add this dye ,if colour changed it means that WBC are function well (extra info from doctor not important )

**Slide 21:** was skipped (doctor just read the headline epidemiology)

**Slide 22:** doctor focused on the fact that mortality rate are higher in new born

**Slide 24:** Psittacosis transmitted by birds.

Chagas’ disease cause achalasia in oesophagus (also known as American trypanosomiasis) which is caused by (trypanosoma cruzi )

Tryponosoma brucei cause sleeping sickness transmitted by tsetse flies (irrevelant to myocarditis)

 trichinosis (trichinella spiralis found in pork meat(most common) and affect muscle tissue)

Done by loai azar