Pathophysiology

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**Scenario # 1**

**Tonsillitis**

Jennifer has been diagnosed with tonsillitis with all the symptoms she possesses. The pathogenesis of inflammatory / infectious disease of tonsils and adenoids probably involves its anatomical location and material processing function infection (through which paradoxically become foci of infection). It is not sure about what decides the onset of endless disease. Viral disease connected with bacterial contamination may be one of the activating components constant diseases, however the impacts of environment, individual's own variables, and eating regimen, between others might likewise be included. Late studies demonstrate that aggravation and loss of grave epithelial uprightness brings about unending cryptitis and block sepulchers, prompting stasis and industriousness of buildups antigens. From that point, even microorganisms occasional in the tombs could duplicate and reason endless contamination (Aydın, et al., 2011).

The tonsils (palatine) and adenoids (pharyngeal tonsils) are structures that have a protective function to tissues, as well as lymph nodes. Strategically placed at the entrances of the digestive and respiratory tube, “fight” simple bacteria and viruses that enter through the nose or mouth (Aydın, et al., 2011). Result in an initial inflammation of the tonsils for the immune system to produce antibodies (create antibodies and markers) against future infections.

The best way to prevent strep throat is to wash your hands frequently and not share eating utensils (such as forks or glasses). It is especially important that anyone with sore throat wash hands frequently and cover their coughs and sneezes. There is no vaccine to prevent strep throat (Aydın, et al., 2011).

**Scenario # 2**

**Contact Dermatitis**

Jack has been exposed to the chemical which causes Contact dermatitis. Knowledge of the pathophysiology of Allergic Contact Dermatitis (ACD) is derived chiefly from animal models in which the skin inflammation induced by exposure of the skin to the hapten is known as contact hypersensitivity (CHS). Two stages divided spatially-temporo are typically expected to attain ideal CHS response: the phases of sharpening and actuation. We depict here the decently acknowledged pathophysiological pathways of CHS and ACD. The sharpening stage (otherwise called afferent stage) is the first skin contact with the hapten and prompts "preparing" and the extension of hapten-particular T cells in lymph hubs (Nosbaum, et al., 2012).

Topically connected hapten is taken up by dendritic cells (DC) skin, particularly Langerhans cells (LC), which move from the epidermis into the paracortical range of emptying lymph hubs, where they display edifices of MHC atoms/ hapten-peptide conjugate antecedent's hapten-particular T cells. Particular T cells emigrate from lymph hubs and achieve the blood through the thoracic conduit and recirculate in the blood and auxiliary lymphoid organs (Nosbaum, et al., 2012). The impelling stage happens a few hours after an ensuing contact of the skin with the same hapten which actuates the creation of chemokines, enactment of endothelial cells and pole cells and the invasion of neutrophils, all important for the recruitment of particular T cells. Immune system microorganisms interface with cutaneous displaying cells convey the hapten antigen.

Initiated CD8 + cytotoxic T cells delivering sort 1 cytokines (IFN g) and incite the actuation of skin cells and apoptosis of keratinocytes, bringing about expanded skin aggravation through the creation of a scope of cytokines and chemokines, and these

recent permit the recruitment of a phone penetrate normal for CHS polymorph. The efferent period of CHS endures 72 h in people. The incendiary response holds on for a few days and logically diminishes upon physiological systems of negative regulation (Nosbaum, et al., 2012).

## Scenario # 3

### Hypertension

Martha is having difficulty in sleeping because of stress as her mother is also dependant on her for the well being. She has been diagnosed hypertension as per her conditions. Despite all the advances in cardiovascular physiology, the primary determinants of hypertension are still poorly understood; can be said that is a multifactorial syndrome, in which complex interactions between genetic and environmental factors cause sustained elevation of blood pressure (Hall, et al., 2011). A small number of patients (between 2% and 5%) have renal or adrenal disease as a cause of high blood pressure, leading to secondary hypertension. In approximately 90% to 95% of cases hypertension is unknown etiology (classified as primary or idiopathic), and the treatment done through changes in lifestyle and / or medication (Hall, et al., 2011).

There is no single cause of hypertension in all individuals. The factors that produce changes in cardiac output or alter the resistance to blood flow will interfere with blood pressure. Sympathetic hyperactivity, environmental factors (such as salt intake or stress), peripheral insulin resistance, with or without obesity, and the renin-angiotensin-aldosterone system are some of the elements that lead to primary hypertension. In recent years, other factors have been evaluated, including genetic, endothelial dysfunction, kinin-kallikrein system and neurovascular abnormalities. In

approximately 30% of cases, hypertension associated with obesity, dyslipidemia and glucose metabolism disorders, or metabolic syndrome (Hall, et al., 2011).

Blood pressure is the force exerted by the blood against the arterial walls for a cardiac cycle and is determined by a combination of processes related to the cardiac output and the strength peripheral vascular. The pressure control is complex and involves hemodynamic, neural and hormonal mechanisms that interact to regulate the pressure occurring when variations due to various stimuli. Different mechanisms are involved in the maintenance as much variation in time-to-time blood pressure, regulating vascular reactivity and caliber, distribution of fluid into and out of the vessels and cardiac output (Hall, et al., 2011).

**Mind Map of Hypertension**

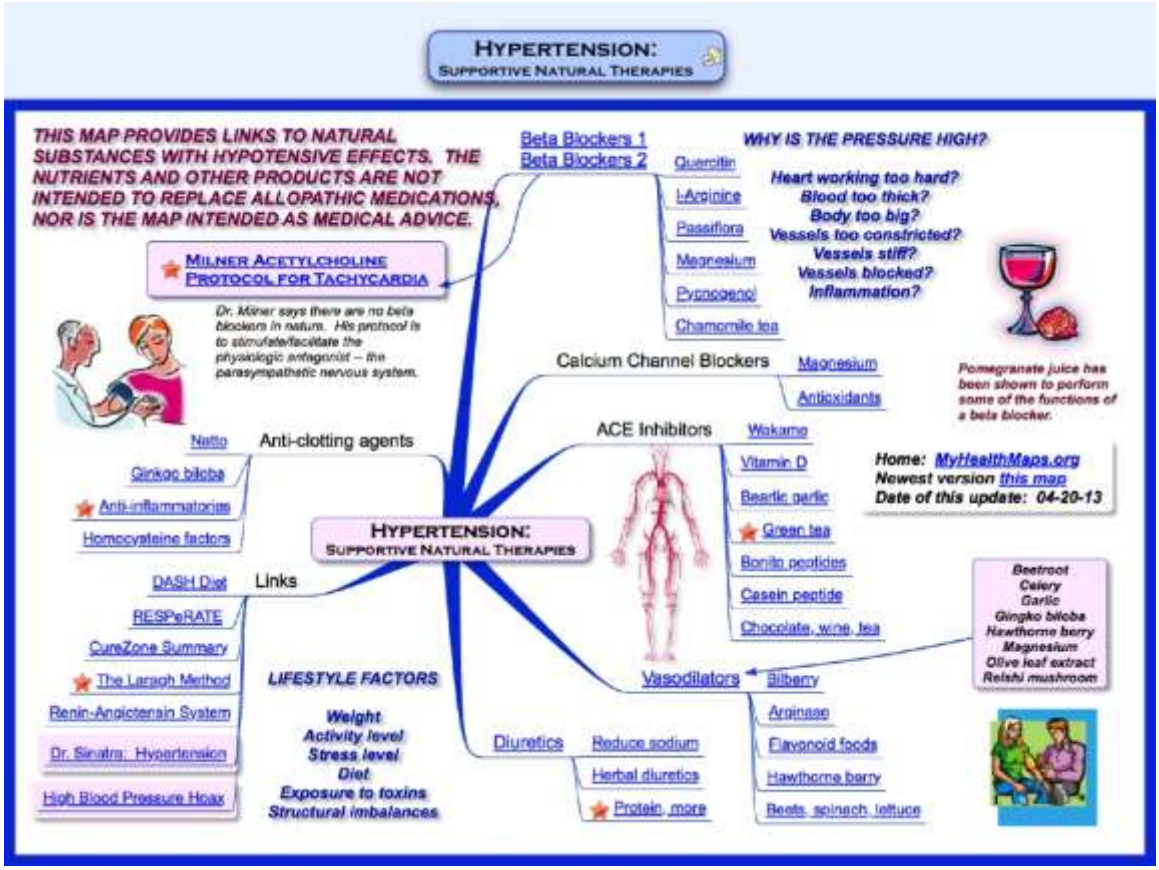


Fig. 1 Mind Map of Hypertension

# References

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