What is the physiology that causes night sweats in tuberculosis?

One of the clinical features of tuberculosis is night sweats. Night sweats associated with active tuberculosis are responses in part to signaling molecules that are released by cells of the immune system as they react to the infectious organism.

The bacteria themselves may also be releasing fever-causing signals.

In response to these circulating chemical signals, the hypothalamus resets body temperature to a higher level for a while. Later, body temperature returns to normal, and the extra heat is lost by sweating or diaphoresis.

Tumor necrosis factor-alpha (TNF-?) is one of the peptide signaling molecules implicated in triggering night sweats.

Monocytes which are a type of white blood cells are a significant source of TNF-?. Monocytes leave the bloodstream and become migratory macrophages, homing in on the tuberculosis-causing mycobacteria.

Although the macrophages may be unable to eradicate the bacteria completely, in an immunocompetent person the macrophages and other cells are able to surround and contain the clusters of bacteria and prevent their further spread through the tissues.

The excess TNF-? released during this immune response appears to be linked with the fevers, weakness, night sweats, necrosis, and progressive weight loss that are characteristic of tuberculosis.

It is possible to reduce the TNF-? level. For example, thalidomide suppresses TNF-? production and helps to moderate the characteristic symptoms and signs of tuberculosis.

It is well tolerated by patients receiving anti-tuberculosis therapy and is associated with accelerated weight gain. However, care has to be taken when manipulating TNF-? levels to avoid reducing the person’s natural immunity to tuberculosis.

For example, it has been shown that when an antibody against TNF-? (Infliximab) is given in the treatment of Crohn's disease and rheumatoid arthritis, the patients have an increased risk of developing previously latent tuberculosis.

Infliximab reduces the numbers of macrophages undergoing apoptosis, and this may affect the integrity of the granulomatous tissue that they organize around clusters of tubercle bacteria.

It was observed in the case of night sweats associated with Hodgkin’s disease that the preceding fever may not be perceived by a sleeping patient, who is more likely to be awakened by the discomfort of the subsequent sweating. This may be the situation also with tuberculosis-linked night sweats.

It is not clear why tuberculosis-induced fevers occur at night. Normal human body temperature displays a circadian rhythm and is generally owest in the predawn hours at 36.1° C before rising to 37.4°C or higher in the afternoon, so the fever/sweating events are probably linked with this daily cycle.

There are three phases to fever.

In the *initiation phase*, cutaneous vasoconstriction promotes heat retention and shivering generates additional heat.

When the new (elevated) setpoint is reached, heat production balances heat loss and shivering stops. With the lowering of the set point to normal, cutaneous vasodilatation promotes heat loss in the form of sweating.

The cause of evening fever in tuberculosis is not yet settled. It could be due to the following two reasons:

1)Due to the tubercular process itself i.e. production of toxins.

2)Due to secondary inflammatory processes such as absorption of septic and toxic material from the decomposing contents of the bronchi and pulmonary cavities.