

# Summary And Conclusions

Summary

## SUMMARY AND CONCLUSIONS

Recurrent aphthous stomatitis is chronic inflammatory disease of the oral mucosal tissue. Its aetiology still unknown exactly. also its treatment and methods of prevention are unknown as well.

- The present study aims to find if there are changes in the minor labial salivary glands which may predispose these patients to ulceration more than others.
- This study has included 10 biopsies from cases of minor recurrent aphthous stomatitis (Mi RAS) during the ulceration phase, 10 biopsies from these cases during the ulcer free period and 5 biopsies from healthy volunteers never complained of RAS before.
- The biopsy is taken from the lower labial salivary glands under local infiltration anaesthesia.
- Every one of these biopsies has been examined under light microscope with haematoxylin and eosine stain, subjected also for histochemical reactions for succinate dehydrogenase enzyme, acid phosphatase enzyme and alkaline phosphatase enzyme and the biopsy specimens are also subjected for electron microscopic examination.
- By means of light microscope we found no gross differences between diseased group, patients during ulcer free period or control group.

### **As regards histochemical reactions:**

- Succinate dehydrogenase enzyme showed no reaction in both secretory and excretory units and this reaction is weaker than that in the control group and patients during the ulcer free period.
- Acid phosphatase enzyme, the reaction exhibited more stronger reaction than the control group in patients during the ulcerative period and during the ulcer free period.
- Alkaline phosphatase enzyme, the reaction was weaker than control group in both patients during ulcerative phase and during the ulcer free period.

### **By electron microscope:**

#### ***In the diseased group during ulcerative period:***

- Many of the secretory acini revealed destructed microvilli and even the apical borders of some acinar cells were broken down and could be observed detached in the lumen.
- The light acinar cells revealed high destruction of mitochondria than in the cases in between attacks.
- The Golgi apparatus exhibited highly expanded cisternae and vacuoles.
- The endoplasmic reticulum tubules were over distended and mostly lost their granular intracisternal contents.
- In the dark cells a fewer destructed mitochondria were observed, the nuclei showed more euchromatin than heterochromatin and the laters were adherent to the inner nuclear membrane.

- In general the mucigen granules of secretory end - pieces were decreased than control but contained concentrated material, the excretory ducts, revealed more electron dense secretory granules than in control.
- The mitochondria of the dark cells mostly lost their cristae or attacked by primary lysosomes and formed secondary lysosomes (Autophagosomes).
- Numerous lysosomes were present either primary or secondary.
- Cells junctional complexes of cells were resistant to these changes.
- Hence we can say that during severe attack the secretion of mucous acini was less in amount but concentrated so that its chemical nature becomes mainly of acidic mucopolysaccharides which give the secretion an acid nature which weakened the covering epithelium and cause the ulcerative condition moreover this ulcerative eruptions were enhanced by proteolytic enzymes secreted from the excretory duct cells where the latter secrete serous fluid rich in these proteolytic enzymes.

#### *In the diseased group in between attacks of RAS*

The picture appears to be lesser than in severe attacks but more exaggerated than control revealing the following:

- Fewer destructed mitochondria than severe cases.
- Golgi apparatus cisternae and vacuoles were slightly distended than severe cases.
- Presence of lysosomes is more than normal but lesser than in severe cases.
- Junctional complexes were still intact.
- The acini revealed poor destruction of microvilli.

So we can conclude that RAS can be considered one of the diseases which is induced by hyperacidity. Its treatment had to be

modified to include antiulcer therapy especially for prevention of recurrences

More studies had to be done to verify these findings and to prove if there is only the secretion in the buccal mucosa playing role in hyperacidity.