SUMMARY AND CONCLUSION

Chronic periodontitis is a complex disease which is chiefly caused by Dental plaque, a biofilm that harbors variety of periodontal pathogens. This is a commonest cause of tooth loss in adults due to damage of periodontal ligament and the alveolar bone. The development of periodontitis and its progression varies from person to person and depends on modifiable and non-modifiable risk factors. Modifiable risk factors are usually environmental or behavioral in nature whereas non-modifiable risk factors are usually intrinsic to the individual and therefore not easily changed. Non-modifiable risk factors are also known as determinants.

Depression is the most commonly diagnosed disease in the practice of psychiatry which includes disruptive mood dysregulation disorder, major depressive disorder and persistent depressive disorder, which may occur at any age. Dental consequences of depression are high and are usually associated with chronic facial pain and bruxism. There are many studies which describe a causal relationship between the periodontitis and the depression.

Stress and depression reduce the immune system function and facilitate chronic inflammation which is mediated through the hypothalamic-pituitary-adrenal axis and the production of cortisol. This leads to increased plaque load with various types of bacteria and reduced ability to prevent connective tissue invasion. Additionally, after periods of chronic elevation, cortisol loses its ability to inhibit inflammatory responses initiated by immune reactions, which leads to sustained inflammatory destruction within the periodontium. Recent studies have also confirmed positive correlations between stress, depression and periodontal disease by demonstrating convincing linkages between depression and tooth loss; stress and attachment loss; stress/depression and neglect of oral hygiene; and elevated cortisol levels and pocket depth/tooth loss, frequently consuming an unhealthy diet and neglecting their oral hygiene. This leads to increased oral biofilm burden and decreased resistance of the periodontium to inflammatory break down.
CONCLUSION

We could not prove that the patients taking antidepressants like venlafaxine or fluoxetine protect the periodontium due to their anti-inflammatory properties, but these drugs may be considered as a risk factor of periodontal disease. Although it is not necessary for the treating dentist to diagnose a depressive conditions but familiarity with the patient’s medical history, current prescriptions, and general indicators of depression could alert the dentist to possible problems and possibly facilitate an appropriate referral for evaluation of the depressive symptoms.

Further studies are required to confirm the role of antidepressants as a risk factor for periodontal disease focusing on drug intake over specified time period and also on the role of individual drugs.

SIGNIFICANCE OF STUDY: To our knowledge this is first study in which the effects of fluoxetine and venlafaxine, on periodontal status in depressed patients have been studied, although these drugs are being prescribed since long. This study reveals, although these drugs possess potent anti-inflammatory effects, but fail to prevent periodontal inflammation.

FUTURE WORK: The results of this study should be interpreted keeping in mind that the study was performed with parameters recorded at one point in time. A cohort study with long follow up should be done. Further, to evaluate the effect of these drugs, prospective randomized clinical trials to be undertaken and they should include the study of changes that might be occurring at cellular or molecular level.