CONCLUSION

8. Conclusion
Purpose of the present study was to assess the role of FXIIIa, MMP-9 and VEGF in fibrosis in the entire spectrum of disease pathology in OSMF. It was found that FXIIIa is strongly expressed in OSMF and the expression decreases with the increasing grades of OSMF. Our study results suggest that FXIIIa expressing cells increase in number in early grade that is before fibrosis develops and the expression goes down in advanced grade which may suggest that the role of FXIIIa diminishes in advanced fibrosis. Hence, we can conjecture that FXIIIa may play role in initiating fibrosis in OSMF. Increase in expression of MMP-9 with increasing grades of OSMF may suggest pro-fibrotic role of MMP-9 in OSMF. Increase in VEGF expression in early grade of OSMF may be related to inflammatory vascular response. VEGF expression increased from early to moderate and there was persistence of VEGF expression in advanced grade. This may suggest the shift of role of VEGF from angiogenesis to fibrosis (angio-fibrotic switch) in OSMF. Positive correlation between FXIIIa and VEGF supports this finding. However, cell culture based studies are required to validate this. Thus, this study may help us to understand the real pathophysiology of fibrosis in OSMF by exploring the role of FXIIIa, MMP-9 and VEGF in initiation and progression of OSMF. This study also helps us to interpret that different factors play role in pathogenesis of fibrosis in OSMF and the role of these factors may change with the progression of fibrosis. Targeting these factors at different stages in OSMF can form the basis of anti-fibrotic treatment modality to treat this progressive disorder.