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Ear reconstruction using the Antia-Buch principles

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Purpose: The reconstruction of the ear following resection of part of it especially the helix, scapha and anti-helix is a confronting problem for plastic surgery. Many techniques had been described to approach this issue and to minimize the complications raised from resection of tumors as well. We present our experience with ear reconstruction using chondrocutaneous flaps and a modified Antia-Buch technique in order to obtain a more realistic result with least complications.

Methodology: The study is retrospective in design with accurate description of the ear reconstruction technique that was performed by the same surgeon on all patients. Twenty (20) patients included in this study. The collected data included demographics, any post-operative complications and post-operative measurements of both reconstructed and normal ears.

Results: There were no post-operative complications, all tumors were completely excised. No flap loss, wound dehiscence or haematoma has occurred. The mean residual defect was 21.8 mm ranging from 14 mm to maximum of 30 mm in size. There were differences noticed in height, width and projection.

Conclusion: This technique allows preservation of anatomical landmarks and contour of the ear and therefore maintaining normal overall 3D appearance of the reconstructed ear. Reconstruction of 10 mm defects are basically allowed through this technique with no loss in size. Due to the potential lobule distortion, we recommend applying this technique to defects up to 25 mm.

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Molecular pathology of oral cancer: Clinical implications

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ral cancer is a major health concern in India being the most common cancer in males and fifth most common cancer females and annual incidence of 77,003 new cancer cases, contributing 26% of the global oral cancer burden. Somatic mutations, aberrant expression, epigenomic regulation and genomic SNPs constitute specific alterations in oral cancer. The focus of our group investigating the molecular pathology of oral cancer is on identification of predictive biomarkers to indicate risk of oral cancer and molecular markers for early diagnosis, prognosis and as therapeutic targets. Somatic mutations in p53, H.ras, EGF-R and NOTCH1 have been observed in 30-60% patients and epigenetic deregulation via hypermethylation in p15/16, DAPK, MGMT, MLH1 and E-Cadherin in 36-50% patients; histone modification in H3 histone via methylation in 39-47% and acetylation in 37-80% and miRNA deregulation in 70% oral cancer patients, providing excellent targets for specific treatment. Several single nucleotide polymorphisms (SNPs) as genomic variants in genes associated with cell cycle, proliferation, differentiation, metastasis, oxidative stress and apoptosis were examined using allelic discrimination real-time PCR assay or high resolution melt-curve analysis. Oral cancer patients demonstrated increased risk with OR 2 to 6.73 and narrow confidence intervals in SNPs including rs4512367 (PREX2), rs1800734 (MLH1), rs34329 (p27), rs16944 (IL1-β), rs2071214 (Survivin), rs13026208 (GALNT13), rs3803300 (AKT1), rs187115 (CD44), rs1982073 (TGFβ), rs1229984 (ADH1B), rs187238 (IL-18) and rs189037 (ATM). Whereas 50% decreased risk was observed with alternate genotypes in PREX2 and TGFβ. Further, the somatic mutations in H.ras gene at codons 12/13/61 was used as a prototype target for identification of small molecules from Maybridge Hit Finder Library, for selective inhibition of constitutive activation of H.ras and consequent proliferation of oral cancer cells. The identified molecules may be potential single or combinatorial therapeutic agents. Thus, molecular biomarkers of oral cancer indicate clinical applications for better management of oral cancer patients.

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