Editorial

Personalized Medicine: The Future of Cancer Treatment

No two individuals are same, macroscopically, microscopically, psychologically and genetically. Similarly, not all the cancers either of different categories/types/nature or of the same category/type/nature are same. Microscopically, they may look similar to each other but there are significant differences at molecular level. This is especially true for oral squamous cell carcinoma (OSCC), as it has got varied carcinogenesis mechanism by virtue of its multifactorial/multietiologic nature.[1,2] Moreover, its association with different oral potentially malignant disorders, which have different carcinogenesis mechanisms, also contributes to the intertumor heterogeneity.[2] This is well-known in oral submucous fibrosis associated carcinogenesis in which tumor cells often show better differentiation.[3]

This molecular heterogeneity in a category of cancer is responsible for upregulation and/or downregulation of myriads of signaling pathways required for cell survival, invasion, migration, angiogenesis and so on. Specific sets of molecules are overexpressed and/or underexpressed and ultimately activate a unique group of signaling events. Blocking of these molecules therapeutically can stop or reverse the process of carcinogenesis. In this regard, kinase inhibitors evolve as important molecules as 'targeted therapy' in cancer.

In OSCC, cyclooxygenase 2, epidermal growth factor receptor, phosphoinositide 3-kinase, protein kinase B, mammalian target of rapamycin, signal transducer and activator of transcription pathways, peroxisome proliferator-activated receptor gamma and so on have been reported to be involved in carcinogenesis. Nowadays inflammation medicated carcinogenesis is also linked with OSCC.[4] The different drugs under trials for OSCC treatment are cetuximab, panitumumab, erlotinib, sorafenib, sunitinib malate, imatinib mesylate, bevacizumab, trastuzumab, lapatinib and mechanistic target of repamycin.

As discussed previously, it is well-known that tumors are 'personalized' in terms of upregulation and/or downregulation of specific signaling pathways. With this knowledge, born the era of 'oncogenomics' and 'personalized cancer medicine'. This is only possible because of new advent in the high-throughput gene sequencing methods, which helps in molecular profiling of tumor tissue. With this diagnostic companion, it is possible to achieve the targeted prescribing of therapeutics. Unlike other cancer therapeutics, this approach is also facing some problems and limitations. The most important being polygenic drug resistance caused by mutational, nonmutational or epigenetic mechanism. Identification of all exploitable molecular abnormalities and full range of resistance mechanism could be answer to this limitation. Other most worrisome aspect is extraordinary 'intratumoral' genetic heterogeneity, which could confuse us in identifying accurate combination of targeted drugs. Moreover, the extent of drug metabolism and its availability at tumor site is the major issue for calculation of therapeutic dosage. Recently, it has been identified that OSCC is a unique form in terms of metabolism of targeted drug, thus making issue of 'personalized cancer medicine' more complex.[5] On the top of it, unpredictable behavior, such as tumor cell cannibalism[6,7] and microbial interactions with signaling pathways[8] could be big challenge for development of this unique therapeutic approach.

In conclusion, oncogenomics and personalized cancer medicine hold good promise for cancer patients. Good genomic analysis of tumor tissue and perfect combination targeted drugs tailored for particular case is key to the success. Future research should be directed to find out and minimize the drug resistance mechanism by cancer cells. Because of the cost and complexity of technology, still time is required to reach this therapeutic approach to every patient. Till then, prevention and early detection may hold the key to success.[9]

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