Chapter 13

Aging and Cancer: Intervention Strategies

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ABSTRACT

This chapter describes aging and the resultant effects of aging. According to WHO report 8.8 million people died from cancer worldwide in 2015. The incidence of cancers increases with age. This increase incidence may be due to biological factors, prolonged exposure to carcinogens and incidence of mutations etc. Cancers in elderly may have poor biological vulnerability, presence of various co-morbidities and poor tolerance of therapy hence treatment got compromised. Elderly cancer patients also are neglected in various trials and strong data lacks for optimal management. Other important aspect is psychosocial state of these patients. Discipline of Psycho-oncology deals with patients with cancer, their lifestyle related difficulties, negligence by self, family members and society, Lack of emotional support, poor financial assistance and treatment monitoring etc. leading to various psychological problems. This chapter will address issues of cancers in elderly including disease biology, disease characteristics, management, their quality of life etc. with reference to elderly patients suffering from cancer.

INTRODUCTION

Aging and cancers are closely interrelated. Incidence of most cancers increases with age. Most of the cancer patients are in there 60’s or 70’s. As the average life span of a population increases with availability of better healthcare, more and more elderly population and more cancers are expected in this group. Recent SEER data estimates that 60% of all cancers and 69% of all cancer related deaths occur above 65 years of age in US (Ries et al., 1996). Both solid and haematological malignancies increase with aging. To understand it deeply we should be able to understand biology of aging and cancer at cellular and molecular level and see their interrelation.

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AGING AT CELLULAR AND MOLECULAR LEVEL

Aging is a complex process many theories have evolved over a time period in quest of explanation to the aging process. None of the theories proposed till date could explain it in a holistic way. It is increasingly becoming apparent that the molecular and subcellular processes of aging are also relevant in cancer and tumorigenesis.

Genetics and Aging

Experiments have shown that there are defined genes in low evolutionary animals like yeast, nematodes and drosophila which affect the life span of these organisms (Orr et al., 1994; Sun et al., 1994). Probably certain critical genes also relate to aging and life span in humans. Genetic regulation of aging comes from analysis of clinical syndromes with accelerated aging like progeria. Accelerated aging is seen in early onset progeria, late onset progeria and Down syndrome (Yu et al., 1996). Identification of genes in these patients is at least a beginning towards understanding of molecular mechanisms involved in aging process. Werner syndrome is a disease of adolescent onset characterised by short stature and probability of development of cancers of mesenchymal origin. Yu et al. (1996) published a data on etiology of Werner syndrome caused by mutation in a single gene located on human chromosome 8 that encodes a helicase-like protein (Yu et al., 1997). Functional characterisations of these genes may be of great significance in understanding physiology of aging.

Telomere Biology and Aging

Cells have their genetic code in form of DNA which is a part of the chromosome. Telomeres are part of our chromosomes which cap their terminal ends and protect chromosomes from damage. With each cell division the telomere gets shortened. Ultimately it comes to a critical level where cell senescence occurs and cell dies. This process gives a defined life span to an individual. Telomerase is an enzyme which tries to protect the telomeres from shortening (Harley & Villeponteau, 1995). During this process cancers can arise due to chromosomal instability. If telomere shortening can be prevented life can be extended which is area of current research too. Many cells of our body like stem cells, sperm cells and T lymphocytes have high replicative power and express telomerase.

There is evidence that many key genes may be involved in maintenance of telomeres. TERC (Telomere RNA complex gene) and TERT (Telomere reverse transcriptase gene) mutations have been implicated in rapid shortening of telomeres in inherited and acquired aplastic anaemia, a process where bone marrow fails producing hematopoietic stem cells. Dyskeratosiscongenita (DKC) is an inherited bone marrow failure syndrome characterized by extremely short telomeres. It has X-linked, autosomal recessive and autosomal dominant patterns of inheritance. Mutation in a gene called DKC1 which codes dyskerin protein is responsible for X-linked type of DKC. Autosomal dominant type of DKC is associated with mutation in TERC gene. This syndrome presents with bone marrow failure (cytopenias), dystrophic nails and skin changes. Patients with DKC are at increased risk myelodysplastic syndrome, leukemia, primarily acute myeloid leukemia and squamous cell carcinomas. This is a classic example of a group of relatively recent entity i.e. telomeropathy (Dokal, 2000).