Low dose ionizing radiation induced acoustic neuroma: A putative link?

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ABSTRACT

Although exposure to high dose ionizing radiation (following therapeutic radiotherapy) has been incriminated in the pathogenesis of many brain tumors, exposure to chronic low dose ionizing radiation has not yet been shown to be associated with tumor genesis. The authors report a case of a 50-year-old atomic reactor scientist who received a cumulative dose of 78.9 mSv over a 10-year period and was detected to have an acoustic neuroma another 15 years later. Although there is no proof that exposure to ionizing radiation was the cause for the development of the acoustic neuroma, this case highlights the need for extended follow-up periods following exposure to low dose ionizing radiation.

Key words: Acoustic neuroma, ionizing radiation, oncogenesis, occupational hazard

INTRODUCTION

There are various case reports and short series regarding radiation-induced CNS tumors following high dose therapeutic radiotherapy.[1-9] Most of these tumors are gliomas, sarcomas, meningiomas,[2,3,5,8,10] and acoustic neuromas are comparatively very rare.[1,4,6,7] On the other hand, exposure to chronic low dose ionizing radiation which occurs as an occupational hazard of working in a controlled radiation environment has never been shown to be linked to carcinogenesis.

CASE REPORT

A fifty-year-old male patient, an atomic energy scientist by occupation, working at the Regional Atomic Reactor Centre in India, presented in 2008 to our Gamma Knife department with a history of left sided hearing loss for one year. Patient also had tinnitus for the same duration. No features of raised intracranial pressure were present. Patient was exposed to ionizing radiation during his stint at the Atomic Energy Reactor from 1982 to 1992 and his total cumulative radiation exposure was 78.9 mSv (63.6 mSv external and 16.3 mSv internal) (1 mSv = 1 mGy). On examination, patient was found to have left sided sensorineural hearing loss. Rest of the central nervous system examination was within normal limits. No neurocutaneous markers were noted. Pure tone audiometry (PTA) revealed a 30 decibel sensorineural hearing loss in left ear. Magnetic resonance imaging (MRI) of brain revealed a left sided cerebello-pontine angle tumor originating from the left internal acoustic meatus, isointense on T1W and hyperintense on T2W images with homogenous post-contrast enhancement. The tumor was 1.6 × 1.4 cm in size [Figure 1]. The patient subsequently underwent radiosurgery (Gamma-knife) and is presently on follow up.

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DISCUSSION

Radiation-induced carcinogenesis is on a continuous rise because of increased environmental and occupational radiation exposure. Radiation causes early as well as late damage to the body. In order to classify radiation-induced tumors, Cahan et al.\( ^2 \) proposed certain criteria for radiation-induced carcinogenesis as follows: the tumor should arise in the field of irradiation, it must be histologically different from the disease that occasioned the radiotherapy; a suitable duration must elapse between the radiation and clinical onset of tumor; no other obvious predisposing conditions for oncogenesis be present. Radiation-induced tumors may further be distinguished into low dose radiation (<10 Gy) and high dose radiation (>24 Gy) tumors. Low dose radiation usually induces benign or malignant tumors, which usually arise after long interval; whereas high dose radiation usually induces malignant tumors.\(^3\) Low dose radiation usually seem to progress to oncogenesis, while high dose cause direct cellular damage. However, chronic exposure to very low dose ionizing radiation which occurs usually as an occupational hazard in medical and radiation fields may also lead to oncogenesis although this is not well documented due to long interval periods and lack of proof of a definitive link between the cause and effect. Our case also suffers from the same limitation. Other studies have, however, documented that effects of chronic exposure to ionizing radiation can be detected on the group level using translocation analysis after chromosome painting, although the mean cumulative dose needed was approximately 100 mSv.\(^{11,12}\) A significant association between translocation frequency and cumulative dose was also observed in workers receiving this dose. Our case nevertheless shows that very long follow up periods (two decades or more) are required to detect oncogenesis in these workers.

Our case also raises an interesting question for studies such as the ‘INTERPHONE’ study\(^3\) which have attempted to assess the risk of mobile phone use with brain tumors. As widespread use of mobile phones started only in 1990’s, the oncogenic effect of this ionizing radiation (if any) may only become apparent after 2010 or even later. We therefore believe that such studies need to be repeated after every decade to detect such putative links.

CONCLUSION

Acoustic neuroma may develop decades after chronic exposure to low dose ionizing radiation. Workers exposed to ionizing radiation over a prolonged period of time should ideally undergo lifetime periodic surveillance to detect any evidence of radiation-induced tumors.

REFERENCES


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