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Influence of Cobalt (60) radiation on the Rat Lung

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ABSTRACT

This study we tried to investigate the effect of Cobalt radiation in lung of Wistar rats weighing 160 to 180 gr. A total of 60 rats were divided into 4 groups with 15 rats each.T1, First group as control group did not receive any radiation, T2) was irradiated with 5 Gy, T3) was irradiated with 7.5 Gy, T4) ws irradiated with 12 Gy. These findings demonstrate dosage d.ependent changes in different parts of the lung tissue. Histopathological studies of the lung revealed radiation-induced Edema, hyperemia can be seen in all the 3 groups and goes up as the dosage increases. Also, the thickening of alveolar walls through penetration of unicellular e.g. lymphocytes and macrophages are observed in animals of groups 3 and 4.

Key words: Cobalt, Irradiation, rat, necrosis, Lung.

INTRODUCTION

In patients with breast cancer, the combination of lumpectomy and radiation therapy as primary treatment has become more commonplace. Studies from exposed human and animals indicate that radiation from cobalt can affect a wide variety of tissues particularly those with greater levels of cellular turnover and divisions also expose to the Moderately low doses of cobalt-60 radiation has resulted in decreased body weight and organ weight [1-3]. *Radiation damage is generally divided into early and late stages. The early stage, occurring 1 to 4 months after treatment, is that of radiation pneumonitis. The severity of radiation pneumonitis increases with increased volume of tissue radiated [4], increased radiation dose, and faster rate of administration [5-7]. Radiation pneumonitis is generally not radiologically apparent after doses less than 20 Gy [8]. A second course of radiotherapy is more likely to induce significant pneumonitis than the initial course [9]* Radiation injury to the lungs does not follow anatomic boundaries. It has sharp, well defined areas of air space consolidation with borders that conform to the radiation pneumonitis may present as patchy consolidation in the

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irradiated field and when the damage is very early or minimal in extent, manifests as indistinctness of the pulmonary vasculature. Radiation fibrosis is generally seen in all patients who received therapeutic doses of radiation. [10-11]

The purpose of the present tudy was to study the impact of cobalt irradiation on the lung tissue response to cobalt irradiation.

MATERIALS AND METHODS

Sixty adult male and female Wistar rats were used in this experiment. Animals were housed under standardized conditions for light and temperature. A commercially prepared diet and clean drinking water were provided ad libitum. The rats were anesthesized with an intraperitoneal injection of mixture of ketamin (80mg/kg) and xylazine (8 mg/kg) prior to irradiation. Rats were randomly divided into four groups (n=15/group) and three groups were irradiated with 5 Gy, 7.5 Gy and 12 Gy, on the whole body for 10 to 15 minutes. The first group served as normal control. Irradiation was performed through the use of cobalt 60 rays with the device from a Canadian company Tretron, model Phoenix, belonging to the Cancer treatment center of Omid hospital, in Urmia. The cobalt radiation was administered to the body using a 250 kv orthovoltage system. A custom designed positioning device based on the standard steriolactic frame was used so that 15 animals could be simultaneously irradiated. Dosimetry was performed by implanting lithium fluoride thermoluminescent dosimeters into various areas. The corrected dose rate was determined to be 205/69c GY/min and irradiated with a distance of 7.5 cm on the field of 35x35 in the dorsoventrl axis.

During30days after irradiation behavioural changes and other changes, mainly on the body surface, and lethality were recorded. The surviving rats at the end of experiment were sacrificed with carbon dioxide.

Samples for histological analysis were processed by commonly used methods. Whole lungs were fixed in %10 formaline.

RESULTS AND DISCUSSION

On the first day after the irradiation the animals were very lethargic. However the apparent lack of gross evidence of any severe effect for several days was unusual. About the fifth to sixth day following exposure weight loss and ruffled fur were observed in group one and by eight day some of them died. Daily mortality increased by 10th to 14th day then subside. When the radiation increased to 7.5 GY somewhat different pattern of illness and death occurred. The animals in this group were severely injured by the day 3rd to 4th. Loss of weight were pronounced and by the fifth day some of them were dead. There was a sharp peak of mortality. Non were survived beyond the 2nd week. The animals which received the highest dose of irradiation were severely prostrated, very wet with sweat and by the second day some of them were already dead by the 3rd day. All the rats in this group died in the 1st week period. Result of quantitative analysis of structural disorders in the lungs revealed radiation produced damages early after exposure which correspond to vascular responses, and edema and hyperemia can be seen in all the 3 groups and goes up as the dosage increases. Also, the thickening of alveolar walls through penetration of

unicellular e.g. lymphocytes and macrophages are observed in animals of groups 3 and 4. There are hyperplasia of lymphatic follicles around respiratory tract. Bleeding, wide necrosis in lung tissue and pleural pnemonia could be seen in animals group 4 which received the highest dosage. Other changes in different groups were showed in table1. (Fig1)

Typically, there is early, transient radiation pneumonitis that occurs within 4–12 weeks after completion of radiation therapy. Radiation fibrosis usually develops within 6–12 months after completion of radiation therapy and can progress for up to 2 years before stability occurs [12-15] *Radiation induced pneumonitis typically develops approximately 6–8 weeks after treatment with doses of 30–40 Gy and is a well known early expected effect of therapy that is related to total dose and fractionation [16].Radiation pneumonitis is most extensive 3–4 months following the end of therapy and eventually becomes radiation fibrosis becomes a stable finding approximately 9–12 months after therapy[17].*

Table1	The	pathologic	wastages of	radiation in	different	dosages on	lung
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second group(5 GY)	Third group(7.5 GY)	Forth group (12 GY)
-edema	-edema,	-edema,
-hyperemia	-hyperemia	-hyperemia
	-thickening of alveolar wall,	-thickening of alveolar wall
	-lymphatic hyperplasia follicles	-lymphatic hyperplasia follicles
	around respiratory tract	around respiratory tract
		-wide necrosis in lung tissue

The pathologic wastages of radiation in different dosages on lung



Various factors influence the degree of injury sustained by the lung after irradiation of a thoracic malignancy, including patient age, prior or concomitant chemotherapy, and irradiation technique (18-20). Three of the most important irradiation technique factors that affect injury are the volume of lung irradiated, the total dose of radiation delivered, and the fractionation of the dose. The total dose of radiation delivered is important, as radiologic manifestations of radiation pneumonitis rarely appear at doses below 20 Gy and are almost always present in patients who receive doses greater than 40 Gy (18). In addition, chemotherapeutic agents such as actinomycin

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D, adriamycin, bleomycin, and busulfan can potentiate the effects of radiation (21). Although steroids ameliorate radiation pneumonitis, abrupt termination of administration can unmask latent radiation injury to the lung (22).

REFERENCES

[1] DeAngelis LM, Gutin PH, Leibel SA. Murtin Dunitz Ltd. **2002**. Interacranial tumors. Dignosis and Treatment.

[2] Rubin P, Gash DM, Hansen JT, Nelson DF, Williams JP. Radiother Oncol 1994; 31:51-60

[3] Schultheiss TE, Stephens LC. Br J Radiol 1992; 65: 737-753.

[4] Rubin P,CasarettOW. Philadelphia: Saunders, 1968

[5] WebbWR, MOller NL, Naidich DP.High reso!u tion CTofthe lung, 2nd ed. Philadelphia: Lippincott-Raven, **1996**

[6] Fraser RS, Pare JAP, Fraser RG, Pare PD. Diseases of the thorax caused by external physical agents. In: Synopsis of diseases of the chest, 2nded. Philadelphia: Saunders,**1994**:781-814

[7] Libshitz HI. Semin Roentgeno, **1993**;28:303-320

[8] Jennings FL, Arden A. Arch Pat/sol1962;74:351-360

[9] Kaplan HS, Steward JR. Natl Cancer Inst Monogr 1973;36:439-444.

[10] Libshitz HI, Shuman LS. J Comput Assist Tomogr 1984; 8: 15–19.

[11] Ikezoe J, Takashima S, Morimot S et al. Am J Roentgenol 1988; 150: 765–70.

[12] Evans WA, Leucutia T. Am J Roentgenol Radium Ther Nucl Med 1925; 13:209-220.

[13] Desjardins AU. Am J Roentgenol Radium Ther Nucl Med 1926; 16:444-453.

[14] Chu FC, Philips R, Nickson JJ, McPhee JG. Radiology 1955; 64:642-653.

[15] Warren S, Spencer J. Am J Roentgenol Radium Ther Nucl Med 1940; 43:682-701.

[16] Wallgren A. Acta Oncol 1992; 31: 237–42.

[17] Libshitz HI. Semin Roentgenol 1993; 28: 303–20.

[18] Rubin P, Casarett GW. Clinical radiation pathology Philadelphia, Pa: Saunders, 1968.

[19] Smith JC. Am Rev Respir Dis 1963; 87:647-655.

[20] Gross NJ. Ann Intern Med 1977; 86:81-92.

[21] Sostman HD, Putnam CE, Gamsu G. AJR Am J Roentgenol 1981; 136:33-40.

[22]Castellino RA, Glastein E, Turbow MM, Rosenberg S, Kaplan HS. Ann Intern Med 1974; 80:593–599.