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ASSESSMENT OF ANTIOXIDANTS STATUS IN LUNG CANCER PATIENTS RECEIVING RADIOTHERAPY

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Abstract:

Introduction: *Lung cancer (LC) is the leading cause of cancer-related deaths in the Western world. LC is divided into two main groups: non-small cell lung cancer (NSCLC) and small cell lung cancer (SCLC).*

Objectives of the study: *The main objective of the study is to analyze the level of antioxidants status in lung cancer patients receiving radiotherapy.*

Material and methods: *This descriptive study was conducted in Holy family hospital, Rawalpindi during April 2018 to July 2018. The data was collected from 50 patients of lung cancer who visited the OPD of the hospital regularly. Patients with any other serious medical or psychiatric condition or those who had received any prior chemotherapy were excluded from the study. All patients underwent laboratory testing at baseline.*

Results: *The data was collected from 50 patients of lung cancer. The diagnosis of LC was confirmed histologically in 27 of the patients and for the remaining nine patients the diagnosis was based on class V BAL cytology and imaging results. Twenty of the LC patients received radiotherapy, 15 of whom underwent a second bronchoscopy during RT. There were no significant differences in glutathione (p = 0.636), vitamin E (p = 0.264), TRAP (p = 0.751), TBARS (p = 0.855), Nox (p = 0.482) or proteins (69.0 vs 72.0 g/L, p = 0.054) between the two groups at baseline.*

Conclusion: *It is concluded that LC is associated with increased oxidative stress. The findings suggest that antioxidant responses may serve as a protective mechanism against production of ROS during RT.*

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INTRODUCTION:

Lung cancer (LC) is the leading cause of cancer-related deaths in the Western world. LC is divided into two main groups: non-small cell lung cancer (NSCLC) and small cell lung cancer (SCLC). Treatment is based on the staging of the cancer and on the performance status of the patient. Surgery is the main treatment for limited disease. Advanced NSCLC is mainly treated with radiotherapy (RT) and chemo-radiotherapy [1].

Radiation generates primary radicals by transferring energy to certain cellular components, e.g. water. Reactive oxygen species (ROS) are formed in this process and they mediate the anti-tumour effects of RT. A delicate situation occurs at the tissue level when oxidative stress is a desired effect against malignant cells; yet the amount of oxidative stress should be kept in balance to prevent permanent damage to normal cells [2].

An increasing number of studies have been published where bronchoalveolar lavage (BAL) has been used as a window to assess the oxidative status of the lungs, e.g. in asthma, pulmonary fibrosis, chronic obstructive pulmonary disease (COPD) and after exposure to ozone and diesel [3]. However, there are only a limited number of clinical studies involving BAL in LC patients. Lung cancer is the most common malignancy worldwide and the most common cause of cancer-related death. Oxidant-antioxidant imbalance is proposed as a possible mechanism for the development and propagation of lung cancer [4]. Elevated levels of thiobarbituric acid reactive substances (a marker of oxidative stress) and reduced activity of antioxidant enzymes have been detected in the blood of lung cancer patients. In addition, surgical removal of the lung tumor has led to increased total body antioxidant status, implying a possible association between the presences of lung cancer cells and circulating antioxidant levels [5]. The administration of chemotherapy is also associated with increased free radicals and decreased antioxidants. This oxidant-antioxidant imbalance plays a role in the side effects of chemotherapy drugs as well, for example, cisplatin-induced nephrotoxicity [6].

Objectives of the study

The main objective of the study is to analyze the level of antioxidants status in lung cancer patients receiving radiotherapy

MATERIAL AND METHODS:

This descriptive study was conducted in Holy family hospital, Rawalpindi during April 2018 to July 2018. The data was collected from 50 patients of lung cancer who visited the OPD of the hospital regularly. Patients with any other serious medical or psychiatric condition or those who had received any prior chemotherapy were excluded from the study. All patients underwent laboratory testing at baseline.

The blood samples for oxidative stress markers were taken as follows: Peripheral venous blood samples were collected using a Venoject blood collection system (Terumo, Leuven, Belgium). Two tubes (10 ml each) of blood were obtained, one for serum preparation and one for plasma analysis. The serum samples were collected into sterile tubes and the samples intended for plasma analysis were collected to cooled, sterile tubes containing ethylenedia-minetetraacetic acid (EDTA). Blood samples were collected of all patients at baseline. The second set of blood samples coincided with second bronchoscopy during RT at 18–22 Gray and the third 3 months after start of RT.

Statistical analysis

The data was collected and analyzed using SPSS version 21.0. All the values were expressed in mean and standard deviation.

RESULTS:

The data was collected from 50 patients of lung cancer. The diagnosis of LC was confirmed histologically in 27 of the patients and for the remaining nine patients the diagnosis was based on class V BAL cytology and imaging results. Twenty of the LC patients received radiotherapy, 15 of whom underwent a second bronchoscopy during RT. There were no significant differences in glutathione (p = 0.636), vitamin E (p = 0.264), TRAP (p = 0.751), TBARS (p = 0.855), Nox (p = 0.482) or proteins (69.0 vs 72.0 g/L, p = 0.054) between the two groups at baseline.

Table 01: Level of Antioxidant variables in patients and control group

			Patients vs Controls			
	Patients	**Controls**	**M (95% CI)**	***p***	**M (95% CI)**	***p***
Alphatocopherol	7.39 (6.01–8.76)	NA				
Gammatocopherol	0.80 (0.55–1.04)	NA				
Ascorbate	73.3 (61.9–84.8)	78.3 (67.0–89.7)	−5.0 (−20.8 to 10.8)	0.529	6.8 (−37.1 to 50.7)	0.753
Thiols	303 (283–322)	364 (338–390)	−61 (−92 to −31)	<0.001	20.6 (−73.3 to 114.6)	0.651
Nitrite	0.591 (0.548–0.634)	0.181 (0.136–0.225)	0.41 (0.35 to 0.47	<0.001	0.32 (0.18 to 0.46)	<0.001
Nox	28.6 (23.8–33.4)	26.5 (22.8–30.2)	2.1 (−3.9 to 8.1)	0.482	7.0 (−10.2 to 24.2)	0.410
Vitamin E	26.7 (22.1–31.3)	29.8 (26.3–33.2)	−3.0 (−8.5 to 2.4)	0.264	−14.4 (−33.1 to 4.3)	0.121

DISCUSSION:

Lung cancer is associated with enhanced circulating concentrations of urate and nitrite ($p < 0.001$ for both), which is in accordance with previous findings. Urate, which is the end product of purine metabolism, is one of the major antioxidants in human plasma and it may play an essential role in protecting cells against free-radical induced damage [6]. Thus, elevated levels of urate may signify a compensatory mechanism to oxidative stress. It is also shown that both cancer and RT are associated with increased oxidative damage to DNA and thus hyperuricemia might be also partly due to increased purine metabolism through the effects of xanthine oxidase as a consequence of RNA-DNA breakdown [7].

Nitric oxide (·NO) is involved in many physiological processes and has an extremely short half-life. However, the blood ·NO level does not necessarily reflect the ·NO status of the tissues. In plasma and other physiological fluids, NO is oxidized to nitrite, whereas in the whole blood [8]. NO and nitrite are oxidized to nitrate. Increased production of nitric oxide may protect the cells from oxidative stress and this might explain the elevated levels of nitrite among LC patients compared to controls [9]. On the other hand, production of a potent oxidant and cytotoxic molecule, peroxynitrite in the reaction of ·NO with the superoxide anion may lead to increased biochemical reactivity and a wide range of damaging effects [10].

CONCLUSION:

It is concluded that LC is associated with increased oxidative stress. The findings suggest that antioxidant responses may serve as a protective mechanism against production of ROS during RT. The cellular damage caused by RT may also result in release of intracellular antioxidative substances.

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