

BREAST CANCER RADIOTHERAPY ASSOCIATED DIABETES MELLITUS CASE REPORTS.

By

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INTRODUCTION

Primary breast cancer when detected early, can be treated by radical mastectomy alone. However, 20-30% of women treated as such later on, develop local or regional recurrence(1). This leads to an additional treatment with radiotherapy by the oncologist for the sake of the 20-30% of patients that may need it postoperatively. The additional treatment by the use of radiotherapy has its own side effects. These include, among other things radiation fibrosis of the lungs which is said to be very rare and has been reported to affect the apices of the lung with excessive fibrous tissue formation. Regional radiotherapy can affect the cellular mechanism of immunity leading to low lymphocyte count. This depression of immunity lead to increase in micro metastases(2). Therefore, because of the above reasons as side effects of radiotherapy, some oncologist do not give radiotherapy in immediate postoperative period.

Diabetes mellitus has never been reported as a complication of therapeutic radiology of the breast in breast cancer, so we have decided to report these cases to keep clinicians on the alert of the possibilities of Diabetes mellitus after radiotherapy.

CASE 1:

A 64-year old retired female teacher, who was brought to the emergency unit of our hospital on account of drowsiness, incoherent speech and weakness of the body. There was no history of fever but patient was noticed to have increased frequency of micturition particularly at night. Her day to night ratio was 5/3. this was noticed 6 weeks after she had received chest radiotherapy as adjunct therapy to mastectomy for the treatment of her left breast cancer. She was not hypertensive nor a diagnosed diabetic. She did not have any family history of hypertension nor diabetes mellitus. She was only on Tamoxifen for her breast cancer chemotherapy after she had had a course of radiotherapy.

On examination, she was found to be an elderly-looking woman, with acetone odour of the breath. She was restless, afebrile, not pale, not jaundiced, no pedal oedema, no peripheral lymphadenopathy. She was dehydrated. Her chest moved with respiration and a left total mastectomy scar had broken down on the upper part of the breast area. Breath sounds were vesicular and of normal intensity. There were no added sounds. They only abnormal finding in the cardiovascular system was a pulse of 124/min, regular but small volume. There were no abnormal findings in the abdomen. The central nervous system revealed a delirious woman, restless with a Glasgow coma scale of 7/15. there were no signs of meningeal irritation and no focal neurological deficit. A urinalysis done in the emergency unit showed 4+ of glucose but no protein was present. A random blood sugar done at the same time was 44.2mmol/L. a provisional diagnosis of Type 2 diabetes mellitus with

Hyperosmolar coma was entertained. Patient was started on intravenous fluids low dose soluble insulin hourly and serum potassium was corrected by the addition of potassium chloride into the fluids. She also had antibiotic therapy although we found no focus of infection. She made some improvement about 4 days after admission and this was maintained until the 7th day. Thereafter, she lapsed into unconsciousness and all efforts at resuscitating her failed. She died on the 11th day.

CASE 2

A 58 year old house wife was brought by relatives to the emergency unit of our hospital with a one day history of coma. Patient had been noticed to be drinking a lot of fluids recently and was also urinating frequently. No history of fever. No other symptoms referable to other systems of the body. She had had bilateral mastectomy and had received radiotherapy as adjunct therapy for breast cancer 10 weeks earlier. She was not a diabetic nor was she hypertensive. There was no known family history of diabetes mellitus or hypertension. She was on Tamoxifen chemotherapy for breast cancer.

On examination, she was an elderly looking woman, in coma with a Glasgow coma scale of 4/15. She was afebrile, not pale, not jaundiced, no pedal oedema, no lymphadenopathy. She was dehydrated. Her chest had scars of bilateral mastectomy, while other chest findings were not remarkable. In the cardiovascular system, there was a tachycardia of 128/minute and a low normal blood pressure of 90/50mmHg supine. No remarkable findings were seen in the abdomen. The central nervous system revealed a comatose woman, responding only to sternal pressure by slight movement of the upper limbs. She had no signs of meningeal irritation. A urinalysis done at the same time showed a serum glucose of 587/mg/dl.

A provisional diagnosis of Type 2 diabetes mellitus with hyperosmolar coma was made. Patient was started on intravenous fluids (Normal saline) with low dose intravenous soluble insulin, hourly. She received only 3 doses of the insulin with 4 litres of fluids before she stopped breathing. Her serum electrolytes and urea (which came out after patient had died) were markedly deranged and her calculated serum osmolality was 396mOs/l.

CASE 3:

A 38 year old female business woman was referred from a private hospital with a diagnosis of diabetes mellitus for follow up in our diabetes clinic. Clerking the patient further, revealed that she had no family history of diabetes, but she had received radiotherapy after she had had mastectomy of the right breast. Her symptoms of diabetes started shortly after a course of radiation therapy for breast cancer. She was being managed on oral hypoglycaemic agents.

The only abnormal finding on physical examination was a

right mastectomy scar. Her fasting blood sugar level was not very high and it was at 9.7mmol/l. She has been continued on oral hypoglycaemic agents and her serum glucose level now is under control.

DISCUSSION

Diabetes mellitus is a disorder that results from relative or absolute lack of insulin³. The Pancreas produces this insulin which regulates carbohydrate metabolism. When the Pancreas is damaged by noxious agents, it may result in glucose intolerance or frank diabetes mellitus. The agents known to damage the Pancreas ranged from microorganisms (Viruses, particularly hepatitis B virus, mumps virus and Coxsackie's B virus) to chemicals particularly alcohol (and its metabolites) and excessive iron leading to Bronze's diabetes⁴. There has been no reports of diabetes occurring in patients who have had radiation therapy for breast cancer. These three patients all had breast cancer and all had radiotherapy. It therefore suggest that radiotherapy must have had something to do with the pancreatic Islet cell damage that brought about the lack of insulin manifesting with the symptoms of diabetes mellitus which were not overt, hence the first and the second patients were not brought for medical attention early but were brought in hyperosmolar coma.

It is possible that the radiation therapy that leads to fibrosis of the apex of the lung as a complication of radiotherapy can also damage the Islet cells. These Islet cells are more in the tail of the Pancreas than in the body and head. The tail of the Pancreas seem to be easily damaged as seen from other conditions that bring about diabetes from Pancreatic damage. The conditions (haemochromatosis, alcoholism

And hepatitis B infection) may lead to overt diabetes mellitus but not to maldigestion from Pancreatic exocrine dysfunction as Pancreatic enzymes are not affected. It may therefore be that the Islet cells become damaged and fibrotic leading to diabetes mellitus in the process of treating breast cancer by chest irradiation.

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