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Case Report

Multiple primary neoplasms developing in a case of prostate cancer

D Kulkarni, G A B Miller

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Carcinoma of prostate is the second most common cause of cancer death in men in the UK. It is known that patients with carcinoma of prostate are at significant risk of developing a second primary neoplasm.[1] These second primary are mainly colorectal, stomach or urinary bladder. The histopathology of gastrointestinal tumours is usually adenocarcinoma. We report a case of prostate cancer that developed an unusual combination of atrial myxoma, small bowel carcinoid, basal cell cancer on shoulder skin, rectosigmoid adenocarcinoma and caecal adenoma over a period of five years.

CASE REPORT A 73 years old man presented with significant bladder outlet obstruction in 1996. On clinical examination he had a smooth enlarged prostate. The postate specific antigen (PSA) was elevated at 38.5ng/1. Transurethral resection of prostate was performed; 21gm of this was resected. The histopathology report was adenocarcinoma of prostate (Gleason score 3, in 90% of resected tissue). The staging CT scan was normal, and bone scan revealed a suspicious area of increased uptake near the right femoral metaphysis. The patient was started on hormonal treatment in the form of Goserelin injections. The serum PSA level returned to normal within 1 year and has remained within the normal range for 5 years. A bone scan repeated after 3 years was also normal.

In June 2000 the patient underwent an emergency left brachial embolectomy. Histology showed only blood clot. Subsequent 2D echocardiography confirmed the presence of a lesion with features of atrial myxoma almost filling the entire right atrium. After discussion with the patient, conservative treatment of the myxoma was decided upon and anticoagulation therapy was started.

In March 2001 the patient presented with vomiting and abdominal distension. X-ray showed features consistent with small bowel obstruction. An exploratory laprotomy was carried out. A small bowel tumour was identified with extensive mesenteric lymphadenopathy and was resected. Histopathology confirmed small bowel carcinoid. All the mesenteric lymph nodes were negative for metastases.

In July 2001 the patient presented with bleeding per rectum. Colonoscopy showed a rectosigmoid pedunculated polyp more than 2 centimetres and moderate divericular disease of the sigmoid colon. The rectosigmoid polyp was snared and excised and was confirmed to be an adenoma with a focus of adenocarcinoma.

In 2001 a 1cm lesion on the left shoulder was biopsied and found to be a basal cell cancer. It was treated with flurouracil cream and colistin sulphate and polymyxin B sulphate cream.

On routine follow-up colonoscopy in November 2001, a flat growth in the rectum was found along with a large polypoid lesion in the caccum and a small polyp in the transverse colon. The histopathology confirmed the presence of a rectal adenocarcinoma and a tubulovillous adenoma of the caecurn with no dysplasia.

Tyrone County Hospital, Hospital Road, Omagh, Co. Tyrone, BT79 0AP.

D Kulkarni, MB, BS, MS, Senior House Officer.

G A B Miller, FRCS, Consultant Surgeon.

Correspondence to Dr Kulkarni, Breast Unit, Department of Academic Oncology, 3rd Floor, Thomas Guy House, Guy's Hospital, London, SE1 9RT.

The patient was also on medications for hypertension and gout.

DISCUSSION

This patient developed multiple primary neoplasms of prostate, small bowel, heart, rectum, caecum and skin. The pathology was as varied as adenocarcinoma, myxoma, basal cell carcinoma and carcinoid.

The patient gave no family history of any disease or malignancy. His only risk factor was heavy smoking, which he had stopped 5-6 years before his first diagnosis. Apart from the lesion seen on bone scan (which was not apparent on subsequent bone scan) there was no other evidence of metastasis from any of the primary neoplasia.

The clinical picture in this case does not fit into any described multisystem genetic syndromes like Carney or Gorlin syndrome where more than one malignant tumour occurs.

The interval between the first two primary neoplasms detected was 4 years. The subsequent intervals were 2 years, 6 months, and 3 months. This is keeping with other reported cases. For reasons unknown, the mean interval between diagnosis of first two primaries is always greater than the subsequent neoplasms detected.[2]

Multiple primary neoplasms in a single individual is a known clinical entity. It is not yet fully understood but it is blamed on chromosomal instability, genetic predisposition, and environmental risk factors.

11q13 is a homogenously mutated gene in the majority of MEN 1 tumours, whereas BRCA1 and BRCA2 mutations might be associated with increased susceptibility for developing more than one neoplasm in cancer of breast or ovary. This suggests that a single mutation can bring about malignant change in more than one organ.[9, 10]

A patient with heritable cancer syndromes often develops multiple primary cancer (MPCs) suggesting hereditary predisposition.

Out of 50,000 cancer cases recorded at National Cancer Center Japan, 2000 had MPCs. This frequency is rising in Japan probably due to exposure to carcinogens.[3] Thus environmental factors, carcinogens, genetic predisposition, mutations, and even heredity and familial syndromes are thought to play a role in developing MPCs.

It has been noted that patients with carcinoma of prostate are at a significant risk of developing second malignancy (as high as 15.2% in a Japanese study).[2, 5] Stomach, urinary bladder, colon and lungs are the commonly involved organs.[8] It has also been reported (as seen in this case report) that overall survival of patients with prostate cancer was not significantly reduced by the association of MPCs,[2, 4] and chances of developing MPCs increase with the tumour grade,[5] and the fact that a patient already has a malignancy makes him prone for MPCs.[6]

CONCLUSION

Patients with cancer prostate are at high risk of developing multiple primary neoplasms. The mechanism however is not fully understood. The mean interval between detection of second primary neoplasm after carcinoma of prostate is usually longer than subsequent detection of neoplasms. Multiple primary cancers however do not seem to significantly affect the overall survival of patients with prostate cancer.

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