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Non-organic Somatic Symptoms in Cancer

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Somatic symptoms considered as unrelated to cancer or grossly out of proportion to the known pathology were systematically assessed in a group of 98 consecutively referred cases. Subjects with prominent somatic symptoms (somatisers) had depression (53%), anxiety (12%) and atypical somatoform disorder (27%). During follow-up, somatisers with depression showed clinical improvement whereas those with atypical somatoform disorder showed no improvement or deteriorated. Methods of distinguishing the cause of somatic symptoms in cancer patients are discussed.
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INTRODUCTION

SOMATISATION REFERS to physical symptoms that cannot be explained by organic disease or are considered grossly in excess of what would be expected given the objective medical findings [1]. Clinical and epidemiological studies [2–5] have shown an association with depression and this appears to be the commonest cause.

Depression occurs in a substantial proportion of patients with cancer [6–11] but the nature and frequency of somatisation in cancer patients has not been studied. This may be because of the difficulty in deciding whether certain physical symptoms are due to cancer [12–14], treatment by cytotoxic drugs [15, 16], radiotherapy [17], psychiatric disorder or a combination of these, for somatic symptoms of depression [4, 5] overlap those of cancer [12–14]. The aim of this study was to determine the frequency and nature of somatisation in cancer patients referred for psychiatric consultation.

PATIENTS AND METHODS

98 patients consecutively referred to the first author over a 6-month period by the medical oncologists for a psychiatric consultation were included. Symptoms considered unrelated to cancer, or grossly out of proportion to the known pathology by the oncologists were systematically evaluated. Assessment included a careful perusal of the clinical notes and the opinion of the medical oncologists was taken into account for all cases. Physical symptoms were assessed in order to ascertain whether the cause was purely organic, mainly psychological or emotional in origin, or arising out of physical as well as psychological factors. Psychiatric diagnosis was made when appropriate, according to the International Classification of Diseases, 9th revision [18]. Patients who had prominent somatic symptoms without an organic basis, anxiety state or depressive illness were diagnosed as "atypical somatoform disorder" using the criteria in the DSM IIIR [19]. Patients who demonstrated somatic preoccupation (repetitive and minute details of bodily symptom) and somatic concern (excessive worry about dysfunction, recurrence or progression) were identified during the clinical evaluation. Patients who reported these prominent somatic symptoms were considered as "somatisers". All the patients were assessed and followed-up by the first author. All cases were followed-up for periods of 2 weeks ($n=3$), 4 weeks ($n=10$), 6 weeks ($n=9$) and 2 months ($n=5$).

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Table 1. Psychiatric diagnosis of the somatisers

Psychiatric diagnosis	*n*	%
Depression	14	53
Anxiety	4	12
Atypical somatoform	7	27
Adjustment disorder	1	4
Nil psychiatry	1	4

RESULTS

27 (28%) of the 98 patients were considered to be somatisers. The commonest somatic symptoms reported as the chief complaint were pain ($n=14$, 52%), tiredness or exhaustion ($n=8$, 30%) weakness ($n=6$, 22%), reduced energy ($n=5$, 19%), lethargy ($n=4$, 15%) and tremors ($n=3$, 11%). Somatic concern and preoccupation was noted in 11 (41%) of these cases. The psychiatric diagnoses of the somatisers were depression (53%), anxiety (12%) and atypical somatoform disorder (27%). 1 case had an adjustment disorder and in one no psychiatric disorder could be identified (Table 1). Table 2 presents features of the cases diagnosed as atypical somatoform disorder. Many patients found it difficult to give an accurate description of their bodily sensation or symptom. Some somatisers had multiple somatic complaints.

Table 2. Presentating features of somatoform disorder cases

Case	Age (years)	Sex	Diagnosis	Main presenting feature
1	54	M	Ca Lung	Abnormal pulsations in neck
2	46	M	Lymphoma	Flu symptoms, muscular pain
3	55	F	Ca Ovary	Persistent tremors hands, body
4	60	M	Myeloma	Bodily functioning
5	30	M	Myeloma	Tightness over face, chest pain
6	62	M	Ca Lung	Sensation over chest
7	50	M	Ca Lung	Sensations over body

M, male; F, female; Ca, carcinoma.

Antidepressants were prescribed for 17 (63%) of the cases, who either had moderate to severe depression (11 cases), atypical somatoform disorder (4 cases) or anxiety (2 cases). Beta-blockers and benzodiazepines were given to 3 cases each. Other treatments given were relaxation exercises (4 cases) and counselling (4 cases) with or without psychotropic medications. The nature of somatic symptoms were explained to the patients but it was difficult to assess how much insight they gained regarding their symptoms. During follow-up 14 of the somatisers showed clinical improvement whereas 13 remained the same or deteriorated. Improvement was noted in the subjects with anxiety (3 out of 4) and depression (9 out of 14), whereas 6 of the atypical somatoform disorder cases remained the same or deteriorated.

DISCUSSION

Somatic symptoms have been found in association with physical diseases like neurological [20] and cardiovascular diseases [21]. In the present study 28% of the cancer patients referred for psychiatric consultation had prominent somatic presentation and multiple somatic symptoms were common. The main reasons for referral were the persistence of physical complaints despite appropriate therapy and/or suspicion of psychological factors in causation or maintenance of the physical symptoms. Previously, a few studies have described somatic symptoms in survivors of childhood cancer [22, 23], and survivors of Hodgkin's disease [24, 25]. However, in these studies the symptoms were considered to be residual symptoms caused by cancer or its treatment. In contrast, the somatic symptoms observed in this study were not related to cancer or its treatment.

Depression was diagnosed commonly and this association between depression and somatisation is similar to that documented in psychiatric populations [2–5]. Most patients (of depression) were treated with antidepressants (mainly dothiepin) and responded well. The favourable clinical improvement in somatic symptoms in those depressed could be due to the antidepressant as well as the analgesic properties of the tricyclic antidepressants. The response of those with atypical somatoform disorder was poor. Though a number of measures were attempted the clinical response was not very encouraging, similar to the experience with somatising psychiatric patients.

Table 3. Common somatic symptoms reported as chief complaints

Somatic symptom	*n*	%
Tiredness	8	30
Weakness	6	22
Multiple pains	6	22
Muscular pain	6	22
Reduced energy	5	19
Lethargy	4	15
Headache	3	11
Pain (heightened)	3	11
Backache	3	11
Tremors	3	11
Pain chest	2	6
Pain abdomen	2	6

Other symptoms reported were tingling, sense of wind, hot and cold sensations, tightness over face, odd sensations, pulsations on neck, feelings of bearing down (in 1 case each).

The findings confirm an association between somatisation depression and cancer patients, though they may be of little diagnostic significance in identifying depression in cancer patients [26]. It needs to be appreciated that somatic symptoms do pose a clinical difficulty as regards their management. In advanced cancer, depression and somatic symptoms have been known to be due to endocrine and metabolic brain syndromes, cerebral metastasis, neuroinfections, nutrition deficits, and antitumour therapies [14, 15, 26, 27]. Somatic manifestations in the present study did not have these disturbances, and seemed to be caused by psychological distress.

Certain characteristics were noted in our patients with somatisation. Excessive somatic preoccupation and somatic concern were obvious in 40% of these cases. The somatic symptoms varied with mood state and worsened during emotional distress. Past psychiatric morbidity and family psychiatric history could also be important in identifying somatisation, but there were not enough cases in this study to look into this aspect. Also, it should be noted that these somatic symptoms were studied in cancer patients referred for psychiatric consultation and that non-organic somatic symptoms in other cancer populations have not yet been investigated. The follow-up period in this study was brief and a longer follow-up interval would have been more useful in allowing evidence of metastatic disease or other causes related to cancer to become more obvious.

In summary, this study demonstrates that somatisation can manifest in cancer patients, mainly in association with a depressive disorder. Somatisation in cancer patients without depression was found to be difficult to treat and tended to persist. More studies are needed to understand the occurrence and treatment of such somatic symptoms in cancer patients. Identification of somatisers has importance in clinical practice also for their appropriate management. Patients who have physical symptoms grossly out of proportion to the underlying pathology, or if the site of somatic symptom is not understandable, or if there is evidence of psychological factors and symptoms along with the physical symptoms, such patients are likely to be somatisers. Moreover, somatisers show a marked variability in the intensity of somatic symptoms with changes in mood, though to a certain extent mood can affect the intensity of physical symptoms of cancer as well. It would, therefore, be useful to refer such patients for careful psychiatric assessment.

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Trends in Neuroblastoma in Great Britain: Incidence and Mortality, 1971–1990

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Incidence and mortality rates for neuroblastoma in Britain from 1971 onwards were examined using data from the population-based National Registry of Childhood Tumours. Incidence throughout 1971–1990 was within the range previously reported from Europe, North America and Oceania. The age-standardised rate rose, however, by 26% between 1971–1975 and 1986–1990, and there were increases of 36% both among infants aged under one year and also among children aged 1–9. There was a pattern of increasing risk with more recent years of birth up to 1985. It is implausible that improved diagnosis could explain the increase in rates since 1971, though it may account for a marked decrease in recorded incidence at the age of 10–14. Age-standardised mortality fell by 27% between 1971–1975 and 1981–1985, but rose again during 1986–1990. This was the result of a halt in the improvement in survival rates for neuroblastoma combined with a substantial and as yet unexplained increase in incidence.

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INTRODUCTION

NEUROBLASTOMA HAS a poor prognosis compared with most of the more common childhood cancers and the outlook for older children with advanced disease at diagnosis is particularly poor. Survival rates have improved substantially during the past 20 years, but survival from late stage neuroblastoma is even now only achieved in a minority of cases, and at the cost of very intensive treatment which is debilitating whilst in progress and carries a substantial risk of a range of late effects.

An alternative strategy for reducing mortality from neuroblastoma is population screening, in the expectation that most cases could be diagnosed at an earlier age and stage, allowing a high cure rate with less intensive treatment. Screening is technically feasible, since the great majority of children with neuroblastoma have increased levels of catecholamine metabolites in their urine, which can be detected in samples by chromatographic methods.

As with any cancer, some clues to the degree of success of screening for neuroblastoma may be obtained by observing changes in the distribution of cases by age and stage and in their survival rates, but the ultimate test is whether screening has any effect on mortality [1]. This is particularly important in the case of neuroblastoma for two reasons. The first is that the most malignant forms may not have a sufficiently long presymptomatic period to be reliably detected by screening [2]. The second is the well known phenomenon of spontaneous regression in neuroblastoma, which has recently been reported in a case detected by screening at age 6 months [3]. Although the screen-

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