EPIDURAL SPINAL CORD COMPRESSION FROM METASTATIC CANCER: CLINICAL FEATURES AND MANAGEMENT

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ABSTRACT

We retrospectively analyzed thirty-three patients (21 males, 12 females) with malignancy induced spinal cord compression (SCC). The mean age of the patients was 42.8 years and almost half (51%) of them presented with SCC. Mean duration of symptoms was 4.5 months and the mean interval between the original diagnosis of cancer and the development of SCC was 14.6 months. Back pain was the most frequent (97%) symptom with an equal number of patients having subjective or objective evidence of lower limb weakness. Majority (73%) of the patients were non-ambulatory at the time of diagnosis. Spinal level involvement was mostly thoracic (62%) followed by lumber (38%). Breast cancer was the commonest underlying malignancy (21%). Lung (12%), prostrate (12%), multiple myeloma (9%), and carcinoma with unknown primary (12%) were also frequently encountered. There was an overall response rate of 22% to the therapeutic interventions: mostly observed in the ambulatory patients. Only 7% of the non-ambulatory patients regained ability to walk. None of the responders had bladder or bowel dysfunction. Twenty-two percent of the responders are still ambulatory with a mean follow-up of six months (JPMA 41: 60, 1991).

INTRODUCTION

Spinal cord compression (SCC) is one of the commonest neurologic complications seen in patients suffering from cancer. At autopsy, its occurrence is documented in approximately 5% of patients with malignancy[1]. SCC is an oncologic emergency because delay in recognition and without urgent treatment, permanent neurologic damage may occur. SCC usually occurs in the setting of previously diagnosed malignancy but a sizeable proportion (8% - 47%) may present with SCC as the initial clinical manifestation of underlying cancer[1-3]. Management and the results of treatment depend upon the duration of symptoms prior to the diagnosis, degree of neurologic deficit, promptness of intervention, type of treatment and the underlying malignancy. At the Aga Khan University Hospital, we performed a retrospective analysis of thirty-three patients with malignancy induced SCC. We studied the clinical features, site of vertebral involvement, type of underlying malignancy, subsequent management and survival. We also compared these data with the study from Memorial Hospital[2].

PATIENTS AND METHODS

All patients developing SCC who had a prior diagnosis of cancer or those presenting with SCC in whom malignancy was subsequently found to be the cause of SCC were eligible for this study. On a pre-set format, .information was collected regarding the clinical features at the time of diagnosis. Origin of the primary neoplasm was ascertained from review of the clinical findings and the available pathologic material. Level of the cord compression was determined on the basis of clinical findings, plain x-rays, CT scan and myelogram when available. Details of management, response to treatment and follow-up data were obtained from the case records.

RESULTS

Thirty three patients were eligible for the study. The clinical features are presented in Table I.

Table I. Retrospective Analysis of SCC.

Clinical Features	AKUH (n=33)	Memorial (n=130)
Sex		
Male (%)	64	61
Female (%)	36	39
Age		
Mean (years)	42.8	58
Range (years)	18-60	4-85
SCC as Presenting Feature (%)	51.5	10.0
Duration of Symptoms		
Mean (months)	4.5	2.0
Range	1 day - 2 years	5 days - 2 years
The Interval between Original Tumor and SCC		
Mean	11.8 months	
Range	0-5 years	
Signs and Symptoms (%)		
Pain	97	96
Weakness	97	87
Sensory loss	45	51
Autonomic Dysfunction	45	57
Ataxia	6	3
Ambulatory	27	49
Non-ambulatory	73	51

Almost two-thirds of the patients were male. Median age was 42.8 years. Slightly more than half (52%) presented with SCC as the initial manifestation of underlying malignancy; in the remainder it developed after a mean interval of 11.8 months (range 0-5 years) after the original diagnosis of cancer. Patients had symptoms related to SCC for an average of 4.5 months (range 1 day -2 years). Back pam was the commonest finding. Sensory deficit and autonomic dysfunction were frequently observed (45.5% each), whereas ataxia was uncommon (6%). Most of the patients (73%) were non-ambulatory at the time of diagnosis. Level of spinal involvement was mainly thoracic (62%), followed by lumbar (38%). One patient had compression at more than one site. Primary tumours causing SCC are listed in Table II.

TABLE II. Retrospective Analysis of SCC.

Primary Tumors causing SCC	AKUH (%)	Memorial (%)
Breast	21	21
Lung	12	16
Prostate	12	11
Unknown	12	3
Myeloma	9	6
Kidney	6	9
Sarcoma	6	5
Others	22	29

Other include Hodgkin's disease, ALL, melanoma, parotid, esophageal and germ cell cancers.

Breast cancer was the commonest cause of SCC in females. In males, lung and prostrate were frequently encountered. The results of treatment are summarized in Table III.

TABLE III. Retrospective Analysis of SCC.

Management and Response to Treatment	
Treatment (n=27)	%
Steroids	85
Radiotherapy	33
Surgery	15
Chemotherapy	74
Response Rate	22
Survival (mean period of follow-up = 6 months)	
Alive and ambulatory	22.5
Alive and non-ambulatory	5.5

Only eighty-two percent of the patients received treatment. Rest were felt to have SCC for too long a time period to benefit from any treatment. Majority (85%) received steroids, followed by chemotherapy (74%) and radiotherapy (33%). Decompression laminectomy was performed in fifteen percent of the patients. Follow-up is available on 67% of patients, 22% of them are still fully ambulatory. Best results were achieved in those who were ambulatory at the time of presentation and did not have bladder or bowel dysfunction. Fifty percent of the patients who were ambulatory remained so, however only 9% of the patients who were non-ambulatory were able to walk again. Of those who remained or regained ambulatory status, none had bladder or bowel dysfunction. Seventy-two percent of the patients died without resolution of SCC, 22% are alive and ambulatory, rest alive but non-ambulatory. The median duration of follow-up is six months.

DISCUSSION

SCC can be a devastating neurologic complication of cancer. Delay in diagnosis and initiation of therapy may result in jrreversible neurologic deficit[4]. Hence early recognition and prompt intervention is essential for a favourable clinical outcome. Autopsy studies indicate that approximately 5% of the cancer patients develop extra-dural spinal metastases, many of them remain asym-ptomatic during life[1]. Vast majority of the cases of SCC are caused by extra-dural rather than intra-medullary compression of the spine[2]. Primary tumours causing SCC most commonly arise from lung, breast, prostrate and kidney[3]. In this study, two-thirds of the patients were males. This is similar to Memorial study[2]. However, our patients had a mean age of 42.8 years which is much lower than 58 years reported in that study. Reason for this observation remain unknown although it may be related to lower life expectancy. Almost half of our patients presented with SCC as compared to less than ten percent in the Memorial study[2]. However, others have observed this mode of presentation in upto 47% of their patients[1,3]. The earliest and the most important symptom of SCC is back pain. It has been observed in 96% of cases in the Memorial study[2] and 97% of our patients. It precedes the onset of SCC by 7 weeks to almost 7 months[5]. Hence the development of back pain in patients with cancer should be taken very seriously and may possibly be an early indicator of SCC. Motor dysfunction (subjective or objective weakness) was observed equally frequently in ours and Memorial studies[2] (97% vs 87%). Distribution of sensory dysfunction (45% vs 59%) and ataxia (6% vs 3%) is also very similar. Duration of the symptoms due to SCC prior to the establishment of the diagnosis varied from 1 day to 2 years. Similarly, time interval between the initial diagnosis of cancer and development of SCC varied from o to 5 years, the longest interval was observed in patients with carcinoma of breast. Level of spinal involvement waâthoracic in 62% and lumbar in 38% of the cases. In the Memorial series[2] 15% of the blocks were cervical, 69% thoracic and 16% lumbosacral. Absence of cervical SCC in our series remains unexplained although small size of this study and lower number of lung cancer patients may be partly responsible for this finding. Commonest cause of SCC in our study is breast cancer which differs from the Memorial study[2]. Since Aga Khan Hospital is not a radiation oncology centre and lung cancer more often requires radiotherapy, the pattern of referral of patients may explain this difference. It would be interesting to look at data from Jinnah Postgraduate Medical Centre, Karachi or other radiotherapy centres in the country. Distribution of other tumours is quite similar to the Memorial study[2]. Outcome of the treatment depends upon the degree of neurologic deficit already present and its duration prior to the diagnosis[6-10]. Fifty percent of our patients who were ambulatory remained so for a period of 2 months to over one year. Only seven percent of the non-ambulatory patients became ambulatory (50% vs 7%). This is similar to the Memorial study[2] (60% vs 7%). Our study also demonstrates that development of autonomic dysfunction is a poor prognostic factor and none of our patients with bladder or bowel dysfunction responded to any measures. This has been shown in other studies as well where two-thirds of the patients with autonomic dysfunction failed to achieve any benefit[4]. This is in marked contrast to those without autonomic dysfunction where more than fifty percent remain ambulatory[4]. Steroids are commonly used for the SCC to decrease vasogenic edema in the compressed cord[11-14] and results in stabilization of the neurologic deficit. Decompression laminectomy may not offer any additional advantage over radiotherapy alone[2,11,15]. Its role may be confined to the patients who have received prior radiation, require a diagnostic procedure and those with pathologic compression fracture, known radioresistant tumours (melanoma, sarcoma etc) or with destruction of the spine by a paraspinous tumour. However, more recent introduction of vertebral body resection, tumour excision and immediate stabilization of spine has shown excellent responses[16-18]. Most of our patients had dense paraplegia at the time of diagnosis. Vast majority of these patients did not respond to steroids

and no radiotherapy was given. However, almost half of the ambulatory patients remained so after treatment with steroids and radiotherapy. Twenty-two percent of our patients are still alive and ambulatory with a mean follow-up of six months. In conclusion, malignancy induced SCC must be part of the differential diagnosis ofa patient who presents with back pain and leg weakness. In those with prior cancer, back pain is usually the earliest sign. The condition requires prompt diagnosis and early intervention to prevent irreversible neurologic damage. Commonest tumour causing SCC is breast in females and lung in males. If diagnosed early, and treatment started while the patient is still ambulatory, the clinical outcome may be significantly improved.

REFERENCE

1. Barrons, K.D., 1-lirano, A., Araki, S. and Terry, RD. Experiences with metastalic neoplasms involving the spinal cord. Neurology, 1959; 9: 91.
2. Gilbert, R.W., Kim, J.H and Posner, J.B. Epidural spinal cord compression from metastatic tumor; diagnosis and treatment. Ann. Neurol., 1978; 3: 40.
3. Stark, R.J., Henson, R.A. and Evans, S.J.W. Spinal metastases. A retrospective sur from a general hospital. Brain, 1982; 105: 189.
4. Delaney,T.F. and Oldfield, E.H. Spinal cord compression, in cancer principlesand practice of Oncology. Edited by Devita, Heliman, Rosenberg. Philadelphia, Lippincott, 1989, p. 1978.
5. Rodriguez, M. and Dinapoli, R.P. Spinal cord compression; with repeated reference to metastatic epidural tumors. Mayo Clin. Proc., 1980; 55:442.
6. Posner, J.B. Spinal cord compression; a neurologic emergency. Clin. Bull., 1971; 1: 65.
7. Tarlov, LM., Klinger, H. and Vitale. S. Spinal cord compression studies. 1. Experimental techniques to produce acute and gradual compression. Arch. Neurol. Psychiatry, 1957; 70: 813.
8. Tarlov, l.M. and Kiinger, H. Spinsl cord compression studies. II. Time limits for recovery after acute compression in dogs. Arch. Neurol. Psychiatry, 1954: 71: 271.
9. Tarlov, LM. Spinal cord compression studies, Ill. Time limits for recovery after gradual compression in dogs. Arch. Neurol. Psychiatry, 1954; 71: 588.
10. Martenson, J.A. Jr., Evans, R.G., Lie, M.R., llstrup. D.M.. Dinapoli, R.P., Ebersold, M.J. and Earle, J.D. Treatment outcome and complications in patients treated for malignant epidural spinal cord compression (SCC).). Neurooncol., 1985; 3: 77.
11. Ushio, Y., Posner, R., Posner, J.B. and Shaprio, W.R. Experimental spinal cord compression by extradural neoplasms. Neurology, 1977; 27: 422.
12. Ikeda, H., Ushio, Y., Hayakawa, T. and Mogami, H. Edema and circulatory disturbances in the spinal cord compressed by epidural neoplasms in rabbits. J. Neurosurg,, 1980; 52:203.
13. Kato, A., Ushio, Y., Hayakawa, T., et al. Circulatory disturbances of the spinal cord with epidural neoplasms in rats.). Neurosurg., 1985; 63: 260.
14. Weissman, D.E. Glucocorticoid treatment for brain metastases and epidural spinal cord compression; a review.). Clin. Oncol., 1988; 6: 543.
15. Young. R.F., Post, EM. and King. GA. Treatment of spinal epidural metastases; randomized prospective comparison of laminectorny and radiotherapy. 3. Neurosurg,, 1980; 53: 741.
16. Harrington, K.D. Anterior cord decompression and spinal stabilization for patients with metastatic lesions of the spine.). Neurosurg., 1984; 61: 107.
17. Siegel. T., Siegel, T. Surgical decompression of anterior and posterior malignant epidura! tumors compressing the spinal cord; a prospective study. Neurosurgery, 1985; 17: 424.
18. Sundaresan, N., Galicieb, J.H., Lane, 3M., et al. Treatment of neoplastic epidural cord compression by vertebral body resection and stabilization.). Neurosurg., 1985; 63: 676.