Acta Oncologica Vol. 34, No. 4, pp. 538–542, 1995

Short Communications

Comments on published articles, short communications of a preliminary nature, technical notes and the like are accepted under this heading. The articles should be short and concise and contain a minimum of figures, tables and references.

A SUBDURAL HEMATOMA FROM METASTATIC ESOPHAGEAL CANCER

Esophageal cancer accounts for 15% of all cancers and 7% of all gastrointestinal malignancies in the United States (1). The most common sites of metastases are lymph nodes, lungs, liver and bone (2). Intracranial metastases are less frequently encountered. Subdural hematomas from metastatic carcinomas have been reported infrequently; but, there are no reports in the literature of subdural hematoma from metastatic squamous cell esophageal carcinoma. The present case report describes what we believe to be the first case of subdural hematoma secondary to metastatic squamous cell esophageal cancer, with a review of the relevant literature.

Case report. A 57-year-old man was admitted with acute pancreatitis and a four-month history of epigastric pain, odynophagia and dysphagia. There was also weight loss of 30 lbs. in the 6 months prior to admission. The patient was a chronic alcohol abuser who had smoked one pack of cigarettes per day for 40 years. A barium swallow revealed a long segment of irregular narrowing in the mid-esophagus. Endoscopic biopsy of the narrowed area revealed infiltrating moderately differentiated squamous cell carcinoma. Staging included computed tomography scans (CT) of the chest and abdomen, and a bone scan; all were normal except for thickening of the mid-esophageal wall. Combined-modality therapy was given consisting of chemotherapy with 5-fluorouracil 400 mg/m^2/d, and radiotherapy. Treatment was complicated by severe mucositis which required a 2-week interruption of therapy with a reduction of the dose of 5-fluorouracil to 300 mg/m^2/d. He completed 6 weeks of therapy receiving 51.5 Gy of radiation. Two weeks after completing therapy he was admitted to the hospital, complaining of malaise, fatigue, dysphagia and fever. A Groshong catheter infection was found and treated by catheter removal and intravenous antibotics. One week later, he complained of a left-sided headache. His blood pressure at this time was moderately elevated and so anti-hypertensive medication was prescribed, resulting in a reduction of his blood pressure; but, the headache persisted. Neurology was consulted and recommended CT scan of the brain which revealed a left parietal subdural hematoma. He was evaluated at the National Naval Medical Center for neurosurgical consultation and was diagnosed with a chronic subdural hematoma that was deemed to be stable. The patient recalled 6 months before falling down steps at work and hitting his head. He was seen at the emergency room of a local hospital and was sent home as he was feeling fine. He was discharged from the VA Medical Center after neurosurgical consultation but was readmitted 2 weeks later with lethargy. CT scan at this time showed an increase in the size of the hematoma with a midline shift with multiple areas of destruction involving frontoparietal and parieto-occipital region of the calvarium (Fig. 1). Burr hole drainage with subsequent drain placement was performed. At the time of surgery, the fluid was noted to be dark and liquefied, consistent with chronic subdural hematoma. No acute blood or clot was noted. The patient's neurologic condition improved and postoperative CT scan showed a resolution of the low density area and midline shift. On postoperative day 4 the patient was agitated and combative and a CT scan now showed a reaccumulation of a mixed attenuation of extra-axial collection of fluid. The patient was taken back to the operating room where he underwent a formal craniotomy for evacuation of the subdural hematoma. Again, chronic liquefied subdural hematoma fluid was evacuated and there was no evidence of acute blood or clot. The

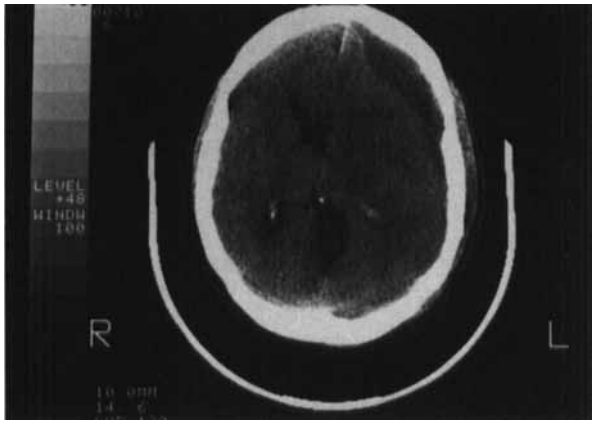

Fig. 1. Non-contrast axial computerized tomography scan of the head showing a large, low attenuation extra-axial collection in the left frontoparietal region with significant shift of midline structures to the right.

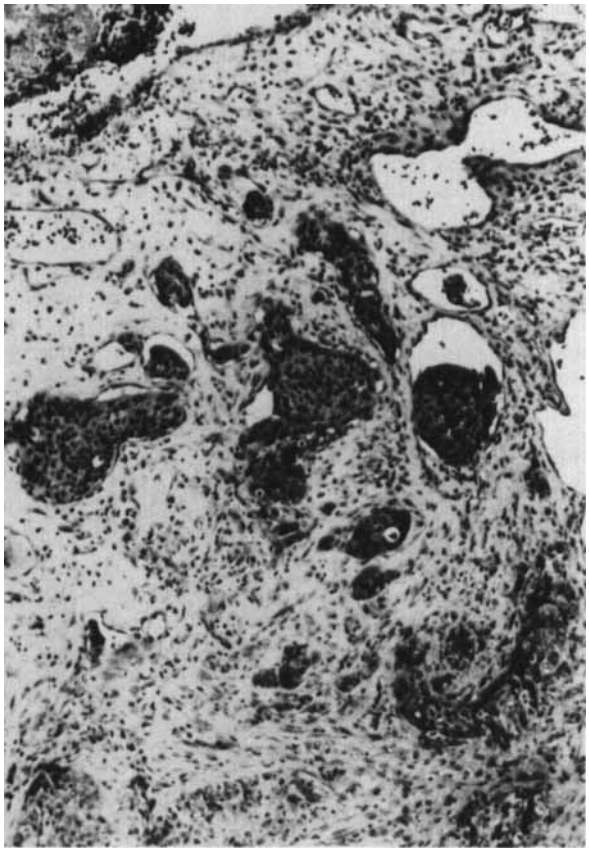

Fig. 2. Meningeal wall at edge of hematoma showing stromal infiltration and permeation of venous channels by metastatic squamous cell carcinoma (HE 160 ×).

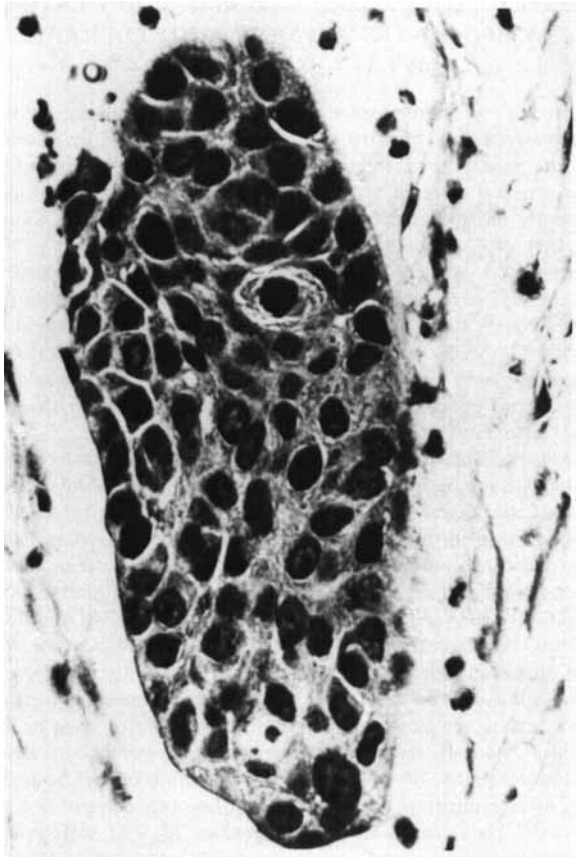

Fig. 3. Embolus of squamous cell carcinoma adherent to wall of meningeal vein (HE 400 ×).

membranes appeared abnormal and were biopsied. Pathologic examination of this specimen revealed metasatic poorly differentiated squamous cell carcinoma, infiltrating part of the meningeal wall of a granulating hematoma (Fig. 2). The venous channels in the specimen were permeated by carcinoma (Figs. 2 and 3).

Discussion. Esophageal cancer most frequently metastasizes to the lung, liver and bone with reported incidences of 48%, 28% and 12% respectively (2, 3). Metastasis to the central nervous system (CNS) is rare with an incidence of 2% with only one case in the literature reporting meningeal matastasis (4). The incidence of intracranial metastases from neoplasms located outside the central nervous system ranges from 18 to 35% with meningeal carcinomatosis having been reported in 8% of autopsy series (5). Subdural hematoma associated with dural metastasis is rare with approximately 30 cases having been reported (6). It occurs in 0.4–5% of metastatic neopolasms of the central nervous system (5, 7–11). Ambiavagar & Scher (12) in an autopsy study of 2 408 cases of non-CNS malignancies found 437 cases (17%) with CNS metastases. Of these there were 166 cases with meningeal metastases of which two also had a subdural hematoma. One of the cases of subdural hematoma was a patient with a cervical carcinoma while the other was a patient with prostate cancer. The first case of subdural hematoma, associated with a metastatic malignancy, was reported by Westerhoeffer (13) in 1904 in a patient with metastatic stomach cancer. This report was followed by Wolhild's (14) report in 1913 of two cases of subdural hematoma from metastatic breast and penile carcinomas. Metastasis to a subdural hematoma may also occur as reported by McKenzie et al. (5). Russell & Cairns (15) were first to postulate a mechanism for a subdural hematoma in the setting of meningeal meta-stasis. They pointed out that the dura consisted of a dense outer layer and a loose inner layer which contained veins which drained into the veins of the dense outer layer and then into the calvarial veins. They found that the veins of the dense outer layer became occluded with metastatic tumor and postulated that this led to rupture of the inner layer veins resulting in a subdural hematoma. Head computed tomography is essential in diagnosis, showing enhanced dural tumor extensions and destructive calvarial changes (16). Our case showed no enhancing lesion but did show effacement of the sulci due to pressure effect and extensive bony destruction involving the left half of the calvarium. The present case demonstrates two extremely rare features of esophageal cancer. The first is dural metastasis which has been reported once previously (4) and the second is an associated subdural hematoma (17). In the prior case of subdural hematoma with esophagus cancer the histology was 'microcellular carcinoma' (17). This, to our knowledge, is the first report of a case of subdural hematoma secondary to metastatic squamous cell esophageal cancer. Patients with cancer who have persisting unexplained headache or mental status changes should have serial imaging of the central nervous system with computed topography or magnetic resonance which is more sensitive than CT since abnormalities may not initially appear or may change over time (18) which happened in this case. Finally, any suspected abnormality found during neurosurgery must be biopsied as it may show a malignancy as was also exemplified in this case.

PATRICK A. CROSS[1]
LOUIS G. ORTEGA[2]
NICHOLAS THEDORE[3]
ROBERT G. WADLEIGH[1]

Division of Oncology[1]
Department of Medicine
Pathology and Laboratory Service[2]
Veterans Affairs Medical Center
Washington, D.C.
Department of Neurosurgery[3]
National Naval Medical Center
Bethesda, Maryland
USA

December 1994

Correspondence to: Dr. Robert G. Wadleigh, Oncology Division, 2CN, Veterans Administration Medical Center, 50 Irving St. NW, Washington D.C. 20422, USA.

REFERENCES

1. Cancer Statistics Review Division of Cancer Prevention and Control. NIH Publication No. 87-2889. Bethesda, Department of Health and Human Services, 1988.
2. Anderson LL, Lad TE. Autopsy findings in squamous cell carcinoma of the esophagus. Cancer 1982; 50: 1587–90.
3. Ayiomamitis A. Epidemiology of cancer of the esophagus in Canada: 1931–1984. Gastroenterology 1988; 94: 374–80.
4. Yonetani KA, Ueki K, Okubo K, Toyohira K, Ito Y, Shinohara S. A case report of esophageal cancer with meningeal carcinomatosis. Rinsho Hoshasen 1987; 32: 857–60.
5. McKenzie CR, Rengachary SS, McGregor DH, Dixon AY, Suskind DL. Subdural hematomas associated with metastatic neoplasms. Neurosurgery 1990; 27: 619–25.
6. Cheng CL, Greenberg J, Hoover LA. Prostatic adenocarcinoma metastatic to chronic subdural hematoma membranes. Case report. J Neurosurg 1988; 68: 642–4.
7. Bergmann M, Pushkas Z, Kuchelmeister K. Subdural hematoma due to dural metastasis: case report and review of the literature. Clin Neurol Neurosurg 1992; 94: 235–40.

8. Turner DM, Graf CJ. Nontraumatic subdural hematoma secondary to dural metastasis: case report and review of the literature. Neurosurgery 1982; 11: 678–80.
9. Tasaki K, Shima T, Matsumura S, et al. A case of subdural effusion secondary to dural metastasis of prostatic cancer: case report. No Shinkei Geka 1989; 17: 763–8.
10. Kinjo T, Mukawa J, Takara E, Horikawa K. Chronic subdural hematoma following advanced cancer: report of three cases. No Shinkei Geka 1989; 17: 763–8.
11. Hirashima Y, Kamiyama K, Endo S, Takaku A. Subdural hematoma due to metastatic dural carcinomatosis associated with DIC—A case report. No Shinkei Geka 1983; 11: 651–6.
12. Ambiavagar PC, Scher J. Subdural hematoma secondary to metastatic neoplasm: report of two cases and a review of the literature. Cancer 1978; 42: 2015–8.
13. Westenhoeffer M. Pachymeningitis carcinomatosa haemorrhagica interna productiva mit Colibacillosis agonalis. Virchows Arch 1904; 175: 364–97.
14. Wohlhild U. Pachymeningitis haemorrhagica interna. Virchows Arch 1913; 214: 388–407.
15. Russell DS, Cairns H. Subdural false membrane or haematoma (pachymeningitis interna haemorrhagica) in carcinomatosis and sarcomatosis of the dura mater. Brain 1934; 57: 32–48.
16. Naheedy MH, Kido DK, O'Reilly GV. Computed tomographic evaluation of subdural and epidural metastases. J Comput Assist Tomogr 1980; 4: 311–5.
17. Vonofakos D, Zieger A, Marcu H. Subdural hematoma associated with dural metastatic tumor. Neuroradiology 1980; 20: 213–8.
18. Snoey ER, Levitt MA. Delayed diagnosis of subdural hematoma following normal computed tomography scan. Ann Emerg Med 1994; 23: 1127–31.

CHROMOSOME ABERRATIONS IN CIRCULATING LYMPHOCYTES AFTER BRACHYTHERAPY FOR UTERUS CARCINOMA

Studies on chromosome aberrations in circulating blood lymphocytes from patients treated for cancer disease by external or internal exposure can serve two purposes: a) they can simulate the situation after an accidental inhomogeneous irradiation and thus aid the assessment of biological damage in such situations, b) they can help to evaluate the response of the patient to the therapy and, possibly, discern patients of abnormal radiosensitivity. Several such studies were carried out in our laboratories in the past, investigating patients with mammary cancer (1, 2), pelvic cancer (3), brain cancer (4) or thyroid cancer (5)—the latter after treatment with ^{131}I. The present study deals with chromosome aberrations in ten patients before and after ^{137}Cs brachytherapy for uterus cancer.

Material and Methods. The investigation was carried out on 10 patients, aged from 37 to 91 years, suffering from cervical cancer, corpus uteri cancer stage 1 or 2a or cervical dysplasia (Table). The patients had not been treated previously. The intracavitary ^{137}Cs treatment involved one cylindrical applicator in the uterine cavity (consisting from the fundus downward of 3 sources of 1.4, 0.814 and 0.814 GBq) and two olive-shaped applicators (1.34 GBq each) placed laterally in the vaginal vault. The duration of exposure and the calculated dose at point A (a point defined by the brachytherapy protocol as being situated 2 cm above the lateral applicator and 2 cm to the side from the cervical midline) are given in the Table. Obviously, this treatment yields very inhomogeneous doses at low dose rates. Blood samples (0.5 ml) were taken before and 1 day after termination of the treatment; they were cultured in 5 ml of F-10 Ham's medium in the presence of fetal calf serum, phytohemagglutinin and antibiotics at 37°C. One ml of 10^{-5} M

Table

Cytogenetic observations in patients before and after treatment

Patient No.	Sampling	Dose Gy (hours exposed) tumor & stage)	No cells analyzed	No abnormal cells	Type and Number of aberrations		
					Chromatid		
					Gaps	Breaks	Exchanges
1	Before	52.5 (70 h)	200	2	0	0	0
	After	Cerv. C. T1	300	20	2	0	0
2	Before	52.5 (70 h)	200	1	1	0	0
	After	Cerv. C. T1NXM0	300	23	3	0	0
3	Before	60 (80 h)	200	5	0	1	0
	After	Copr. C. T1NXM0	300	14	0	2	2
4	Before	30 (50 h)	200	2	2	0	0
	After	Cerv. D.	300	10	2	0	0
5	Before	50 (67 h)	200	8	4	0	0
	After	Cerv. C. T1NXM0	200	10	3	0	0
6	Before	50 (67 h)	200	1	0	1	0
	After	Cerv. C. T1NXM0	200	22	1	9	0
7	Before	50 (67 h)	185	4	0	0	0
	After	Cerv. C. T2aNXM0	200	16	2	1	0
8	Before	50 (67 h)	200	6	2	0	0
	After	Cerv. C. T1NXM0	200	16	2	3	0
9	Before	50 (83 h)	200	5	1	2	0
	After	Cerv. C. T1NXM0	200	18	3	0	1
10	Before	50 (50 h)	200	7	3	1	0
	After	Copr. C. T1NXM0	200	11	1	0	0

Abbreviations: Cerv. C. = cervix carcinoma; Cerv. D. = severe cervix dysplasia; Corp. C = corpus two olive-shaped applicators in the vaginal vault