Cancer Chemother Pharmacol (1983) 10: 223

Cancer Chemotherapy and Pharmacology

Short Communication

Reversible Microangiopathic Hemolytic Anemia after Mitomycin C

David J. Perry

Hematology-Oncology Service, Walter Reed Army Medical Center, Washington, DC 20012, USA

Table 1. Laboratory values at various dates

Date	HGB	Retic	Schisto cytes	Plat	PT	PTT	Cr	Treatment
11/79	11.7	0.4	0	305 K			1.0	FAM
5/80	9.1		+	430 K			1.2	
6/80	7.6		+++	280	12	29	1.3	4 Units PRBC
8/80	6.8	3.9	+++	175	11	32	1.6	2 Units PRBC
9/80	7.3		+++				2.0	4,000 r to stomach
10/80	6.3		++	140	12	36	2.3	
1/81	9.2		+	165			3.3	
10/81	9.9		0	275			3.1	

The patient was a 71-year-old man who presented in November 1979 with gastric adenocarcinoma metastatic to the colon, bone, and retroperitoneal nodes. His admission hemoglobin was 11.7, WBC 6.2, and platelets 305,000. The reticulocyte count was 0.4%. He was treated with 5-fluorouracil, adriamycin, and mitomycin C (FAM). His hemoglobin gradually dropped to the 9–10 g/dl range by May 1980 after nine cycles of FAM (total mitomycin dose of 50 mg/m^2). In May 1980 the hemoglobin was 9.1; the peripheral smear showed infrequent schistocytes. Chemotherapy was stopped at the patient's request; there was no regression of measurable disease. Table 1 shows the relevant laboratory values. In August 1980 tests for fibrinogen and fibrin-split products were done and were negative. Local radiotherapy to the stomach was begun. By Januar 1981 the hemoglobin rose to 9.2. The peripheral smear showed only rare schistocytes. In October 1981 the hemoglobin was 9.9; there were no schistocytes on peripheral smear. The patient died in November 1981; at autopsy there was microscopic evidence of tumor in the stomach, lung, diaphragm, and abdominal lymph nodes.

Microangiopathic hemolytic anemia (MAHA) is a well-known complication of gastric adenocarcinoma and of treatment with mitomycin C [1–3). Evidence of disseminated intravascular coagulation and acute renal failure are usual concomitants. This patient developed MAHA while receiving mitomycin C. During a year of subsequent follow-up the microangiopathic process disappeared in spite of the presence of metastatic gastric adenocarcinoma.

This case illustrates that mitomycin C can produce a MAHA that reverses spontaneously. Patients with gastric adenocarcinoma who develop MAHA have a life expectancy of weeks to months. This very poor prognosis may not apply to those who develop MAHA while receiving mitomycin C treatment for gastric adenocarcinoma.

References

1. Kresssel BR, Duong AT, Berenberg J, Schein PS (1981) Microangiopathic hemolytic anemia and renal failure in patients with adenocarcinoma. Cancer 48: 1738
2. Pavy MD, Wiley EL, Abeloff MD (1982) Hemolytic-uremic syndrome associated with mitomycin therapy. Cancer Treat Rep 66: 457
3. Rabadi SJ, Khandekar JD, Miller HJ (1982) Mitomycin-induced hemolytic uremic syndrome. Case presentation and review of literature. Cancer Treat Rep 66: 1255

Received November 29, 1982/Accepted December 14, 1982

The opinions or assertions contained herein are the private views of the author and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense