# CASE REPORT

**Cardiac metastasis and tumor embolism in a patient with adenocarcinoma of the colon presenting with paraneoplastic polymyositis**

E. MIHALI¹, MIHAELA MUREŞAN², M. L. RUSU¹, DANIELA FODOR¹

¹²nd Department of Internal Medicine
²Department of Anatomopathology
“Iuliu Hațieganu” University of Medicine and Pharmacy, Cluj-Napoca

## Abstract

Colorectal cancer usually presents with alterations in the bowel habit. Less commonly, the presenting symptoms may be part of a paraneoplastic syndrome. Metastasis can occur by lymphatic or hematogenous spread, most frequently to the lungs and liver. We present the case of a 56-year-old man admitted with paraneoplastic polymyositis due to a poorly differentiated colon adenocarcinoma. The evolution was unfavorable with death on the 19th day following admission due to pulmonary thromboembolism and subsequent pulmonary edema. Autopsy showed micrometastases with lymphatic and venous emboli to the heart, liver, kidney, adrenal gland and lung. The case highlights the diversity of manifestation that can occur in a colon adenocarcinoma.

**Keywords:** colon adenocarcinoma, polymyositis, cardiac metastasis, tumor embolism.

## Introduction

Colorectal cancer is the third most commonly encountered cancer [1]. Distant metastases from this type of cancer occur most frequently at the lymph nodes, the liver and the lungs. Uncommon metastatic sites to the spleen, breast, thyroid gland, spermatic cord or skeletal muscle have also been reported, generally as a distinctive feature of end-stage disease [2, 3]. Based on autopsy studies, the heart is an extremely rare site for metastasis with only less than 2% of all cases [4, 5]. More than 90% of all cardiac metastasis are clinically silent [6]. In reported cases, the right atrium was mainly involved [2, 3, 7].

The association of malignancies with dermatomyositis (DM) or polymyositis (PM) has been extensively studied [8, 9] but the relationship between the colorectal cancer and the inflammatory myopathies is unusual, especially if the cutaneous lesions are absent [10].

We report the case of a 56-year-old patient with colon cancer, heart metastases and multiple tumor embolism, presenting with paraneoplastic polymyositis.

## Patient, Methods and Results

A 56-year-old man was admitted for proximal muscle weakness, which was more pronounced in the lower limbs. He also had continuous pain in the thoraco-lumbar spine region. Dyspnea after minimal effort, constipation (onset three month before admission – one episode of stool every 3–4 days with no macroscopic blood), loss of appetite and weight loss (10 kg in the last three months) were his other complaints. He has also been having meteorism and left abdominal flank pain that gradually progressed during the preceding month. His medical history included pulmonary tuberculosis eight years prior to this admission, cured after a six months course of specific medications, with residual left upper lobe fibrotic lesions. No signs of recurrence for the disease were found after a recent pulmonary examination.

Clinical examinations revealed impaired general condition, pallor, temperature of 37.4°C and a body mass index of 25.8 kg/m². Muscle palpation was slightly painful in the thighs and arms. The erect position and walking were very difficult. The patient even required assistance to stand from a seated position. The abdominal right upper quadrant and flank were tender to palpation but no enlargement of the liver, spleen or peripheral lymph nodes were noted. Cardiovascular and pulmonary systems were in the normal range.

Routine laboratory tests (Table 1) showed important inflammatory syndrome, mild anemia, leukocytosis, elevated serum levels of muscle enzymes, cholestasis, hepatic cytolysis, hyperuricemia and dyslipidemia. Elevated carcinoembryonic antigen level was detected (52.6 ng/mL, normal <10 ng/mL) while antinuclear antibodies were negative.

<table>
<thead>
<tr>
<th>Laboratory test</th>
<th>Normal values</th>
<th>Values at admission</th>
<th>19th day after admission</th>
</tr>
</thead>
<tbody>
<tr>
<td>ESR</td>
<td>3–10 mm/h</td>
<td>110 mm/h</td>
<td>80 mm/h</td>
</tr>
<tr>
<td>Hb</td>
<td>11.5–17.5 g/dL</td>
<td>10.3 g/dL</td>
<td>7.7 g/dL</td>
</tr>
<tr>
<td>Leukocytes</td>
<td>5000–10 000/mm³</td>
<td>12 300/mm³</td>
<td>8400/mm³</td>
</tr>
<tr>
<td>Platelets</td>
<td>150 000–450 000/mm³</td>
<td>333 000/mm³</td>
<td>109 000/mm³</td>
</tr>
<tr>
<td>C-reactive protein</td>
<td>&lt;0.6 mg/dL</td>
<td>19.2 mg/dL</td>
<td>9.6 mg/dL</td>
</tr>
<tr>
<td>CK</td>
<td>24–195 U/L</td>
<td>582 U/L</td>
<td>815 U/L</td>
</tr>
<tr>
<td>CK-MB</td>
<td>&lt;24 U/L</td>
<td>undetectable</td>
<td>170 U/L</td>
</tr>
</tbody>
</table>

ISSN (print) 1220–0522 ISSN (on-line) 2066–8279
to be the most appropriate solution. Irinotecan together administrated. Palliative chemotherapy was considered antibiotics and prophylactic anticoagulation were (500 mg/pulse, three consecutive days) along with towards lower values. Three Methylprednisolone pulses with impossibility of walking and standing, and of the general status, aggravation of the muscle weakness metastasis (stage IV) and paraneoplastic polymyositis. of the thoracic lymph nodes enlargement. Electrocardiogram had no spine, diffuse pulmonary fibrosis and right mediastinal irregular thickening in the distal third of the descending abdominopelvic computed tomography examination found and synaptophysin markers were positive. The chest and (G3). At immunohistochemistry, CK AE1/AE3, chromogranin and the muscular biopsy were postponed. to the patient’s altered condition, the electromyography to be the tumoral expression of high levels of myositis autoantigens with subsequently cross-reaction in genetically predisposed patients [13]. The association between colorectal cancer and PM has rarely been reported [10]. Our patient presented with typical symptoms and laboratory data for PM and the aspect of myositis was confirmed by post-mortem histopathological examination. It is known that following cancer removal, the clinical and biological abnormalities usually resolve [12] but in our case, the rapid and unfavorable evolution made the surgical resection to be unattainable. The colon adenocarcinoma being poorly differentiated with multiple metastases probably explain the intensity of paraneoplastic PM.

The association between acquired inflammatory myopathies (DM, PM and inclusion body myositis) occurring as a paraneoplastic syndrome, and malignancies is well known but the individual risk for cancer development in these cases is less established [11]. This association is more frequently encountered for DM. The most common types of cancer associated with paraneoplastic inflammatory myopathies were found to be non-Hodgkin lymphoma, lung, ovarian, renal and bladder carcinoma [8, 12]. The pathogenic link between cancer and inflammatory myopathies was demonstrated to be the tumoral expression of high levels of myositis autoantigens with subsequently cross-reaction in genetically predisposed patients [13]. The incidence of cardiac metastases from any malignancy is highly variable, ranging from 2.3% and 18.3% (more frequently in melanoma and mediastinal primary tumors) [14] but cardiac metastases from colorectal cancer are extremely rare [2]. The cardiac metastases can be found in any structures of the heart (pericardium, epicardium, myocardium, endocardium or vessels). The spread of malignant cells into the heart can be done by direct extension, through the bloodstream or the lymphatic system and by intracavitary diffusion from venous system [14]. In our case, the myocardium was the site of metastasis and this can be explained as the result of retrograde lymphatic spread. The presence of lymphatic emboli suggests this mechanism. It is known that if the intramural lymphatics are obstructed by neoplastic emboli, the lymphatic wall may break, leading to interstitial tumor spreading [14].

The presence of widespread neoplastic emboli (heart, liver, kidney, adrenal gland, and lung) is another particularity of our case. The tumor embolism, especially in low-grade malignancies, correlates with tumor type and site, the incidence being higher for large cell carcinoma (lung, ovary, kidney and

<table>
<thead>
<tr>
<th>Laboratory test</th>
<th>Normal values</th>
<th>Values at admission</th>
<th>19th day after admission</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDH</td>
<td>240–480 U/L</td>
<td>1884 U/L</td>
<td>7044 U/L</td>
</tr>
<tr>
<td>ASAT</td>
<td>0–37 U/L</td>
<td>80 U/L</td>
<td>160 U/L</td>
</tr>
<tr>
<td>ALAT</td>
<td>0–40 U/L</td>
<td>63 U/L</td>
<td>135 U/L</td>
</tr>
<tr>
<td>GGT</td>
<td>7–52 U/L</td>
<td>332 U/L</td>
<td>345 U/L</td>
</tr>
<tr>
<td>ALP</td>
<td>98–279 U/L</td>
<td>1174 U/L</td>
<td>1340 U/L</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>109–202 mg/dL</td>
<td>242 mg/dL</td>
<td></td>
</tr>
<tr>
<td>Triglycerides</td>
<td>40–145 mg/dL</td>
<td>183 mg/dL</td>
<td></td>
</tr>
<tr>
<td>Uric acid</td>
<td>3.5–7 mg/dL</td>
<td>8.2 mg/dL</td>
<td></td>
</tr>
</tbody>
</table>

ESR – Erythrocyte sedimentation rate; HB – Hemoglobin; Ht – Hematocrit; CRP – C-reactive protein; CK – Creatine kinase; CK-MB – Isoenzyme of creatine kinase; LDH – Lactate dehydrogenase; ASAT – Aspartate aminotransferase; ALAT – Alanine aminotransferase; GGT – y-Glutamyltransferase; ALP – Alkaline phosphatase.
Cancer is recognized as a major factor for thrombotic pulmonary embolism but tumor embolization via lymphatic or venous system is generally increased after chemotherapy, radiation or surgical treatment, by promoting the fragmentation of the tumor mass [15]. In this setting, the palliative chemotherapy administrated in our patient could be an explanation for multiple tumor embolizations. The atypical precordial chest pain reported by patient and the sudden increase in cardiac enzymes before his death could be the results of the tumor embolization.

**Figure 1** – (a) Intramyocardial micrometastasis (HE stain, ob. 4×); (b) Intramyocardial tumor embolus (HE stain, ob. 10×); (c) Lung – tumoral emboli, alveolar edema (HE stain, ob. 2×); (d) Kidney – lymphatic tumoral emboli (HE stain, ob. 4×); (e) Muscle fiber – size inequalities of the muscle fibers, nuclear hyperplasia with nuclei internalization, rare lymphocytes (HE stain, ob. 10×); (f) Muscle fibers – discontinuity of sarcolemma, homogenization of the sarcoplasm (HE stain, ob. 10×).

**Conclusions**

The diversity of manifestations of the colon adenocarcinoma can be extremely large. In our case, the paraneoplastic PM symptoms were the most important clinical feature of the patients. Unusual and extensive metastatic disease (including the heart metastases), tumor emboli in various organs and the short time from diagnosis to death were another particularities of the case.
References


Corresponding author
Daniela Fodor, MD, PhD, 2nd Department of Internal Medicine, “Iuliu Hatieganu” University of Medicine and Pharmacy, 2–4 Clinicilor Street, 400006 Cluj-Napoca, Romania; Phone +40264-591 942/442, e-mail: dfodor@umfcluj.ro

Received: January 23, 2013
Accepted: October 25, 2013