EJGO European Journal of Gynaecological Oncology

Case Reports

Peritoneal tuberculosis associated with adrenocorticol primitive neoplasm mimicking a peritoneal carcinosis: a case report

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Summary

The authors report a case of women, 51-year-old that presented with peritoneal tuberculosis, with concomitant adrenocortical primitive neoplasm, mistaken as peritoneal carcinomatosis, due to the failure of correct histological analysis. In fact, the critical life status, associated to increases of CA 125, typical imaging of peritoneal carcinomatosis, and presence of 75% atypical cells in ascitic fluid, induced to begin chemotherapy.

Key words: Peritoneal carcinosis; Tuberculosis; CA 125; Chemotherapy; Immunosuppression.

Introduction

Very little literature describes the capacity of peritoneal tuberculosis to mimic a peritoneal carcinomatosis [1, 2]. An elevation of CA 125 has been observed in tumoral peritoneal carcinomatosis, but has also been observed in cases of peritoneal tuberculosis. If CA 125 monitoring is well known as a means to assess response to anti-cancer therapies [3], its interest is not appreciated for the monitoring of peritoneal carcinomatosis associated with mycobacterium tuberculosis. CT-scan is not specific enough for the diagnosis of peritoneal carcinomatosis and does not allow to distinguish its origin. PET scan can be useful for diagnosis of peritoneal carcinomatosis [4] but does not prove the certitude of the diagnostic. Laparoscopy with histological analysis is the only diagnostic instrument able to provide a clear diagnosis [5].

Case Report

In July 2008, a 51-year-old women who came from Madagascar and had been living in France for about ten years, presented an important abdominal volume increase associated only with abdominal pain. The clinical history of the patient showed that a Bartholin cyst surgery had been performed in 2003, arterial hypertension controlled with drugs, and that the patient was a heavy smoker (15–20 cigarettes/day). The biological marker CA 125 was highly elevated (717 KU/L), however, no other biological anomaly was observed. Ultrasound exploration showed abundant ascites with peritoneal carcinomatosis and pleural effusion with dilatation of the mediastinum. In order to deepen the diagnosis and to expedite treatment, a CT-scan and ascitic liquid analysis were performed. The CT-scan showed a peritoneal and bilateral pleural carcinomatosis (Figure 1a), pelvic mass with uterus myomatous confirming the previous ultrasound diagnosis (Figure 1b), and a right adrenal mass of six cm, compatible with adrenocortical primitive neoplasm (Figure 1c). The biochemical, bacteriological, and cytological ascitic liquid analysis were diagnostically non-contributory, except for the presence of 75% atypical cells.

Because the clinical life status of the patient deteriorated rapidly, no coelioscopy was performed and a chemotherapy was established with carboplatin and paclitaxel (six cycles), beginning in August 2008 and ending in November 2008. During chemotherapy, a primary treatment of neutropenia (granulocyte colony stimulating factor, GCSF) was administered.

After the sixth cycle of chemotherapy, a control CT-scan showed a disappearance of abdominal ascites, important reduction of abdominal and pleural carcinomatosis (Figure 1e), and stability of size of the right adrenal mass (Figure 1f). However, bilateral pulmonary embolism was diagnosed (Figure 1d). The patient was immediately treated with anticoagulation therapy (tinzaparin sodium). A PET-scan realized in December 2008 revealed a suspected abdominal and pleural fixing, compatible with the conclusion of the CT-scan, excluding the right adrenal mass (Figure 2a). Monitoring of the biological marker CA 125 was performed prior to during and following chemotherapy (Figure 3).

Revised manuscript accepted for publication January 26, 2015

Eur. J. Gynaecol. Oncol. - ISSN: 0392-2936
XXXVII, n. 4, 2016
doi: 10.12892/ejgo3557.2016

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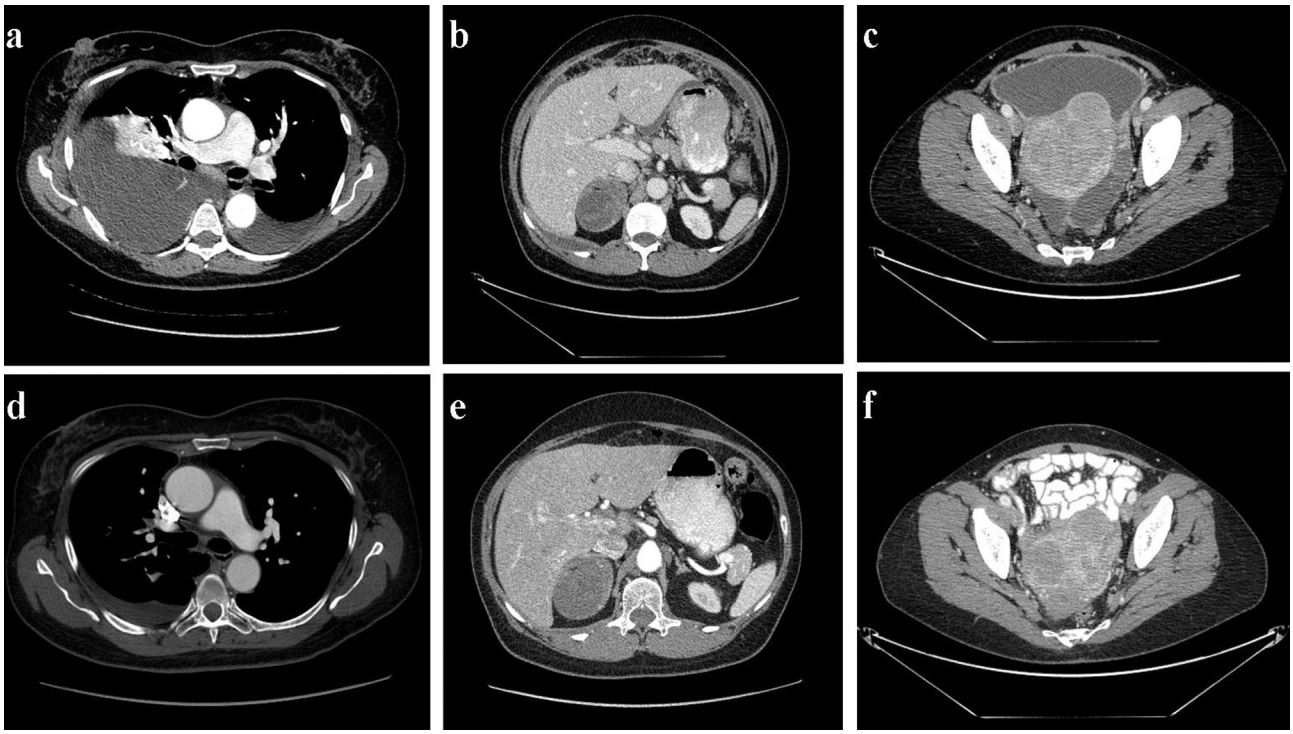

Figure 1. — A CT-scan performed July 2008, prior to chemotherapy, showing: (a) peritoneal and bilateral pleural carcinomatosis, (b) an adrenal mass of six cm and (c) pelvic mass with ascites misinterpreted as carcinomatosis. After chemotherapy, the CT-scan of November 2009 reveals: (d) bilateral pulmonary embolism, (e) unconformity of disappearance of abdominal ascites and reduction of abdominal and pleural carcinomatosis, and (f) size stability of the right adrenal mass.

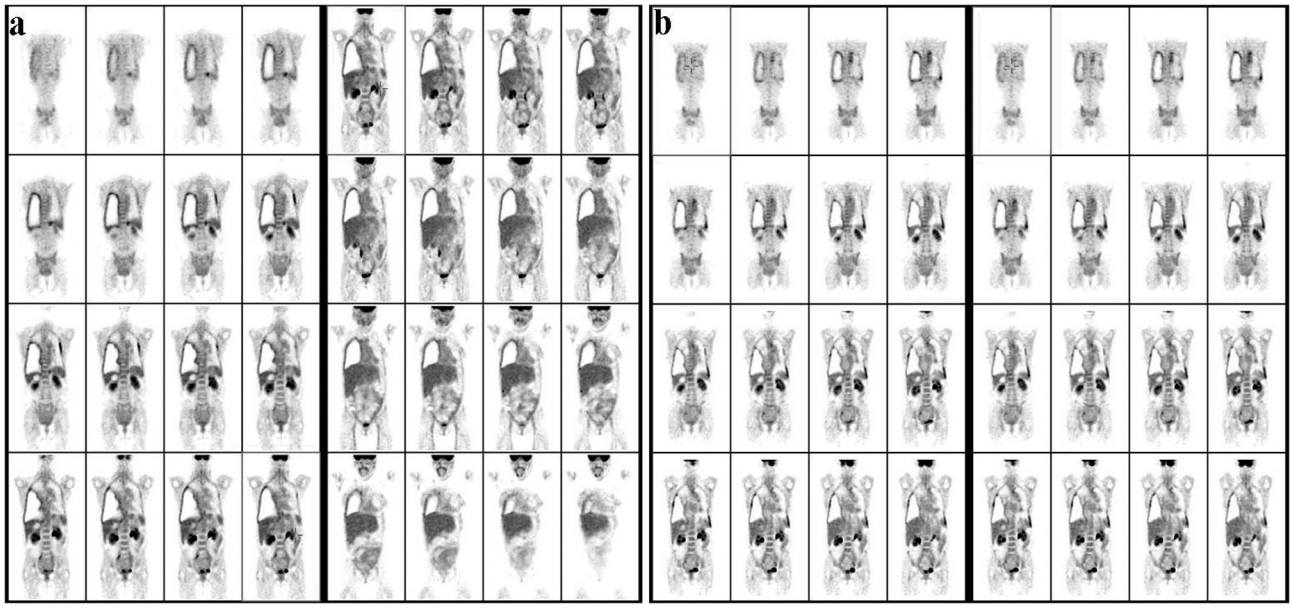

Figure 2. — (a) PET-scan taken in December 2008 showing a suspected tumor abdominal and pleural lesions. This is compatible with conclusions from the CT-scan, but no suspected fixing imagine corresponds to a right adrenal mass location. (b) PET-scan performed in February 2009 showing an evident response to anti-tubercular therapy comparatively to the one first taken in December 2008.

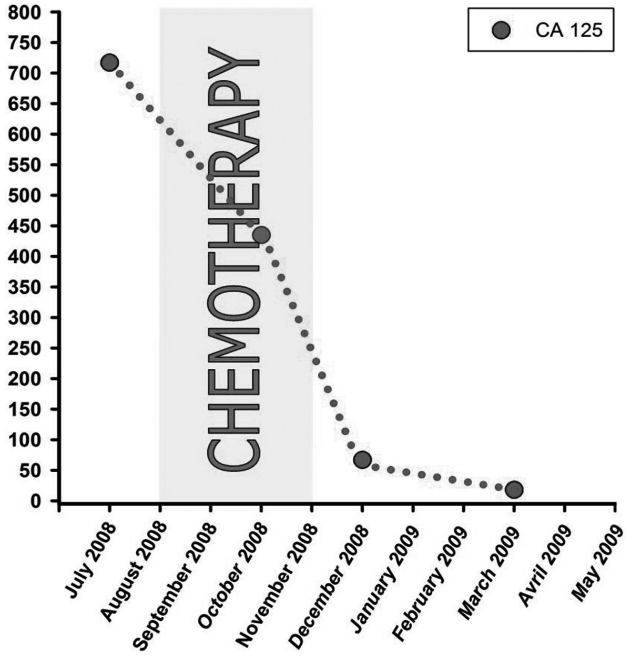

Figure 3. — Monitoring of the biological marker; CA 125 before and after chemotherapy.

As a result of the presence of pleural liquid and as a consequence of important respiratory symptomatology of the patient, it was decided to perform a pleuroscopy with biopsy in January 2009. The result of this test was surprising. No tumor cells were found, but the typical aspect of tuberculosis was revealed. Histological analysis described important necro-inflammation associated with the presence of numerous epithelioid granulomas and giant-cells; the Ziehl test was negative, finally no histological evidence of malignancy was identified. In light of this new diagnostic, an anti-tuberculosis treatment was initiated in February: isonazide 5 mg/kg, rinfapicine 9.6 mg/kg, ethambutol 15 mg/kg, and pyrazinamide. Therefore before the beginning of anti-tubercular treatment, the breath symptomatology improved . A PET-scan performed in February 2009 showed an evident response to anti-tubercular therapy comparatively to the one first made in December 2008 (Figure 2b).

To investigate the right adrenal mass, a study was performed: potassium 3.6 mmol/L, renin 995 µU/l, urinary aldosterone 144 pmol/L, hypercortisolism (cortisol at 8h 342 nmol/L and 12h 362 nmol/L) with urinary cortisol 936 nmol/24h, ACTH 4.1 ng/l, and DOC and 18-OH-progesterone were normal. In May 2009, the authors decided to precede with a adrenalectomy that confirmed a hypothesis of adrenocorticol primitive neoplasm. Furthermore, the abdominal exploration confirmed the absence of peritoneal carcinomatosis. After adrenalectomy and anti-tuberculosis therapy, the clinical status of the patient improved with reduction of pleural liquid.

Discussion

In this case, the critical condition of the patient associated with atypical cells present in cytological ascitic liquid motivated rapid initiation of chemotherapy. The fact that the patient initially responding to this treatment with improvement, was an additional aspect that led to the erroneous diagnosis of abdominal carcinomatosis. The decrease of clinical symptoms and of CA 125 after chemotherapy was misinterpreted as confirmation of peritoneal carcinosis. The authors did not observe a decrease in neutrophils during cytotoxic chemotherapy because the patient had received routine prophylaxis with GCSF: peritoneal tuberculosis of the patient has not worsened.

The authors believe that carboplatin and paclitaxel were efficient against adenocorticocarcinoma [6-8]. Thus, immunodeficiency related to adrenal carcinoma was probably reduced, possibly allowing greater local control of peritoneal tuberculosis.

Conclusion

This case reinforces the principle of histological diagnosis before beginning chemotherapy. The imaging, was not sufficient and can lead to therapeutic errors. Finally, this case illustrates that for certain types of peritoneal carcinomatosis associated with mycobacterium tuberculosis, disease progression is slow and can be better controlled for several months in case of efficient therapy and in situations favoring the tuberculosis.

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