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Overview

Pseudomyxoma Peritonei

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ABSTRACT:
Pseudomyxoma peritonei is a rare form of mucinous ascites associated with peritoneal and omental implants. The origin is controversial, and recent immunohistochemical and molecular genetic evidence suggests the appendix to be the likely site. The condition often presents as an incidental finding at laparotomy. Ultrasonography, computed tomography and magnetic resonance imaging aid in preoperative diagnosis. Treatment remains controversial, surgery being the mainstay. The role of intraperitoneal and systemic chemotherapy is poorly defined. We review the literature on the pathology, clinical features and treatment options in pseudomyxoma peritonei. Harshen, R. *et al*. (2003). *Clinical Oncology* **15,** 73–77

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Introduction

Pseudomyxoma peritonei (PMP) is a rare condition characterized by mucinous ascites associated with peritoneal and omental implants. The term was introduced in 1884 by Werth [1], and literally means a false mucinous tumour of the peritoneum. The basic pathologic process is thought to be seeding of the peritoneal cavity by mucin-producing epithelial cells. The condition is often an unexpected surgical finding occurring in approximately two of 10,000 laparotomies. The mean age at presentation is 58 years [2] with a wide range. Many reports suggest a female preponderance (75% of patients) although others report equal incidence in men and women [3]. Recent immunohistochemical and molecular genetic advances have contributed significantly to resolving the controversy surrounding the site of origin of PMP, with the appendix and ovary being the main contenders [4]. Surgical debulking remains the mainstay of treatment, and the role of other modalities such as chemotherapy and radiotherapy is undefined. This article reviews the current literature on pseudomyxoma peritonei including recent reports on treatment.

Pathological Origin

Morphological Evidence

PMP is commonly thought to arise from either an appendiceal or an ovarian neoplasm. Both these organs are found to be involved in the majority of female patients, thereby making the primary site of origin controversial [5].

Appendiceal lesions reported in association with PMP include mucosal hyperplasia, benign cystadenoma and cystadenocarcinoma. Ovarian lesions including primary ovarian cystadenocarcinoma or mucinous borderline tumours were previously thought to be the site of origin. However, several recent reports have suggested that involvement of the ovaries is secondary to a primary origin in the appendix [6,7]. This theory is supported by the fact that 25–50% of the patients in different series are male, and that ovarian lesions are often bilateral or predominantly right sided. In addition, implants are found on the surface of the ovary rather than within it, in 75% of the patients.

Another theory suggests that the two sites represent multifocal primary neoplasia. This is supported by morphological and immunophenotypical differences between synchronous lesions [8].

Immunohistochemical Evidence

Immunohistochemistry supports the theory of appendiceal origin of PMP. Ronnet *et al*. [9] found that the ovarian lesions were positive for cytokeratin 7, 18 and 20, carcinoembryonic antigen (CEA) and human alveolar macrophage 56. This pattern was almost identical to that of appendiceal lesions, but distinct from a control group of ovarian mucinous tumours without PMP.

Molecular Genetic Evidence

Recent molecular genetic studies also suggest that simultaneous ovarian and appendiceal lesions are clonal

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and likely to originate in the appendix. Szych *et al.* [4] analysed K–ras mutations and allelic loss of chromosomes 18q, 17p, 5q and 6q in 17 cases of PMP with synchronous ovarian and appendiceal mucinous tumours. Identical K–ras mutations were identified in the appendiceal and ovarian tumours in all cases. Loss of heterozygosity was demonstrated in the ovarian tumours in all but one case, with both alleles retained in the appendiceal lesion. This supports the theory that the appendiceal tumour precedes spread to the ovary.

Rare Sites of Origin

In addition to the appendix and the ovary, rare sites of apparent origin of PMP have been reported. These include colon, stomach, gall bladder, pancreas, urachus, urinary bladder, uterine corpus, fallopian tube, breast and lung [10,11].

Histological Classification

Ronnet *et al.* [3] analysed the clinicopathological features of 109 patients with PMP and classified them into two categories. Disseminated peritoneal adenomucinosis (DPAM) was characterized by abundant extracellular mucin containing scant simple or focally proliferative mucinous epithelium, with little cytological atypia or mitotic activity. The other group, peritoneal mucinous carcinomatosis (PMC), demonstrated peritoneal lesions with more abundant mucinous epithelium showing cytological features of carcinoma. DPAM was more common, accounting for 59.7% of cases. Twenty-seven point five percent were classified as PMC and 12.8% had intermediate or discordant features. The classification was found to have prognostic significance, with five year age adjusted survival rates of 84% for DPAM, 37.6% for tumours with intermediate or discordant features and 6.7% for classical PMC.

A recent paper by Jackson *et al.* [12] suggested that cytological features of epithelial cells in the peritoneal washings could accurately categorize patients into DPAM or PMC. The validity of the prognostic classification was further confirmed by this study.

Clinical Features and Complications

The commonest presentation is as an unexpected finding at laparotomy. Common symptoms include abdominal distension, abdominal pain (often mimicking acute appendicitis), or a palpable mass. Nausea, vomiting, urinary symptoms and a scrotal or hernial mass are less frequent symptoms [13,14].

At laparotomy a variable amount of mucinous ascites is found, with lesions of appendix and one or both ovaries. The tumour deposits commonly involve the right hemidiaphragm, paracolic gutters and retrohepatic space. The peritoneal surfaces of the bowel are usually free of the tumour [15].

Primary PMP rarely causes complications even in presence of large volume disease. Rarely, ureteric obstruction and lower limb oedema secondary to venous obstruction have been reported. Recurrent disease, however, may occur on bowel surfaces and can cause fibrosis and intestinal adhesions. This often leads to intestinal obstruction or obstructive jaundice, which may prove fatal [16,17].

Imaging

Preoperative diagnosis by imaging helps in planning the operative procedure. Imaging is also vital in diagnosis of recurrent disease.

Plain Radiography

Plain X-rays of the abdomen are unhelpful in the diagnosis of PMP, but may be useful in assessing complications, particularly intestinal obstruction [18].

Ultrasonography

Reported findings in PMP include non-mobile echogenic ascites, homogeneous tumour deposits, scalloping of liver and spleen due to adjacent peritoneal implants, and rarely small mucinous vesicles [19,20]. Ultrasound guidance to identify less viscous areas aids in diagnostic paracentesis [11].

Computed Topography (CT)

This is the most widely used technique in diagnosis and defining extent. The mucinous material is of fat density. Scalloping of liver and spleen, ascites, septations, and central displacement of bowel loops are typical. Curvilinear calcifications and omental thickening are other highly suggestive features [18].

A recent paper suggests that DPAM and PMC have characteristic features on CT scans [21]. Although there is considerable overlap between the two, certain features such as lack of a dominant mass or lymph nodes, with superficial peritoneal involvement, suggests a diagnosis of DPAM.

Magnetic Resonance Imaging

Magnetic resonance imaging demonstrates the morphological features shown by CT scanning. In addition, T2-weighted images enable differentiation between mucinous and fluid ascites [22]. However, there have been very few published reports of the use of MRI scans, and application is limited by the cost and limited availability.

Treatment

Treatment of PMP remains controversial. Friedland *et al.* [23] suggested that no treatment is necessary, and described one patient who remained asymptomatic for five years without intervention. However, most authors recommend active treatment as summarized below.

Surgery

Surgical debulking is currently regarded as the primary treatment approach [2,24–26]. All operable gross disease is removed, with appendicectomy for histological examination. Sugarbaker *et al.* [27] published a series of 69 patients suggesting that aggressive surgery improves survival in PMP. Patients treated with cytoreductive surgery and intraperitoneal chemotherapy had a five year survival of 92% for minimal postoperative residual disease, compared to 48% for moderate and 20% for gross residual disease. The authors also published an updated series of all patients treated prior to 1999 [28]. Multivariate analysis of prognostic factors for survival showed completeness of surgical cytoreduction and histopathologic character to be highly significant.

Gough *et al.* [29] reviewed the Mayo Clinic experience of PMP. In a series of 56 patients treated over 26 years, removal of all gross tumour was possible only in 34% with limited disease. However, they concluded that surgical debulking should be considered in all patients along with postoperative intraperitoneal chemotherapy or radio-isotope therapy. This paper also discussed the high complication rates associated with aggressive surgery (overall complication rates of 36% with 2.7% mortality).

Smith *et al.* [30] described the MSKCC experience of 34 patients treated over a 37 year period and suggested that long-term survival can be achieved by surgery alone, with chemotherapy reserved for relapse. The lack of randomized data and the absence of a valid control group make it difficult to evaluate the role of aggressive cytoreductive surgery in PMP.

Recent Surgical Advances

Laparoscopic surgery

Raj *et al.* [31] described a laparoscopic approach to PMP. Laparoscopy allowed the thorough exploration of the abdomen, as well as irrigation and aspiration of thick mucinous material and instillation of mucolytic agents. Appendicectomy can be performed laparoscopically with minimal morbidity. In addition, the procedure allows easy placement of catheters for intra peritoneal chemotherapy.

Ultrasonic surgical aspiration

The ultrasonic surgical aspirator has been described as a safe and efficient method of removing residual gelatinous tumour adherent to the abdominal viscera [32]. This avoids the use of diathermy, which may lead to peritoneal burns and ileus.

Chemotherapy

Intraperitoneal chemotherapy

The commonly used agents are mitomycin C, 5-fluorouracil, cyclophosphamide, and cisplatin [33–36]. Sugarbaker *et al.* [28] described the use of perioperative intraperitoneal chemotherapy in patients with optimal surgical debulking. Patients with DPAM received intraperitoneal mitomycin C at surgery. Patients with PMC received in addition, five consecutive days of intraperitoneal 5-fluorouracil. Patients with complete cytoreduction of DPAM had a five year survival of 86%. Patients with incomplete cytoreduction received systemic chemotherapy and had a five year survival of 20% and a ten year survival of 0%.

The Mayo Clinic series [29] suggested that the use of intraperitoneal chemotherapy significantly improved progression free survival over surgery alone. There have been a number of other reports of the use of intraperitoneal chemotherapy in PMP. All are retrospective, with most being case reports or small series [33–36]. As with surgery the lack of randomized or valid non-randomized data make it difficult to evaluate the role of this modality.

Systemic chemotherapy

Anecdotal reports describe the use of single agent systemic chemotherapy with 5-fluorouracil, cyclophosphamide, doxorubicin, hexamethylmelamine or cisplatin [2,28–30,37]. Most studies report very little improvement in disease free or overall survival with systemic chemotherapy [2,29,30]. The Mayo Clinic series [29] suggested that the use of systemic chemotherapy was an adverse predictor of survival on multivariate analysis. However, these are retrospective data, and no randomized comparisons exist between intraperitoneal and systemic chemotherapy.

In general, systemic chemotherapy is recommended for patients of good performance status with gross residual disease after surgery [28], although conclusive data on effectiveness are unavailable. Chemotherapy regimens such as ECF (epirubicin, cisplatin and 5-fluorouracil) need further investigation, in view of the likely origin from the appendix.

Intraperitoneal hyperthermic chemotherapy

This involves the intraperitoneal use of chemotherapeutic agents, usually mitomycin C and 5-fluorouracil, heated to temperatures of approximately 44°C following surgical cytoreduction. [38,39]. *In vitro* studies on hyperthermic solutions have suggested that the cytotoxicity may be increased compared to solutions at body temperature [40].

Witkamp *et al.* [38] reported a series of 46 patients treated with surgery followed by intraoperative hyperthermic chemotherapy with mitomycin C. Forty of the 46 patients had initial optimal surgical cytoreduction. The three year actuarial survival was 81%. There were

four treatment related deaths, and 22 patients (48%) developed bone marrow suppression. These survival figures suggest the need for further studies to evaluate this treatment modality.

Radiotherapy

External beam radiotherapy

El Sayed *et al*. [41] reported the use of megavoltage radiotherapy with large parallel opposed fields to treat the whole abdomen and pelvis in one patient. The intended dose was 35 Gy in 20 fractions over four weeks, but treatment had to be terminated at 14 Gy due to deterioration in general health. The patient remained well and free of symptoms for ten years following this treatment. She then required further surgery and radiotherapy to the right iliac fossa and whole abdomen. She survived for a total of 16 years from original diagnosis.

Intraperitoneal radioisotopes

Radioisotopes such as P^{32}, Au^{198} and I^{131} have been used postoperatively after surgical debulking [29,43,44]. The Mayo Clinic series [29] suggested that intraperitoneal radioisotopes significantly prolonged progression free survival in symptomatic PMP. However, as with other modalities, lack of controlled studies makes it difficult to assess its role.

Immunotherapy

There have been anecdotal reports of the successful use of adjunctive immunotherapy using intraperitoneal OK–432, a streptococcal preparation [45].

Mucolytic Agents

Dextrose solution and other agents have been suggested as a means of loosening mucinous deposits and aiding closed catheter drainage [46]. However, these are of doubtful benefit, and a recent study reported a potentially fatal incident of hyperglycaemia following the use of dextrose for peritoneal lavage [47].

Phototherapy

Laser therapy has been used with photosensitizers at laparotomy for peritoneal malignancies [48]. Reliable data on the efficacy of this modality in PMP are lacking.

Treatment of Recurrent Disease

Reports of multiple surgical procedures for recurrent PMP suggest that the morbidity is acceptable. As PMP is a slowly progressive process, most authors recommend that recurrence should be treated aggressively with further debulking, with or without adjuvant chemotherapy [13].

Treatment Outcome

Analysis of treatment outcomes is limited by the small number of patients in individual series, and the lack of uniform selection criteria for treatment. The majority of patients develop recurrence of PMP after primary treatment. The Mayo Clinic series [29] reported recurrence rate of 76%, of which 50% occurred within 2.5 years. The five year overall survival rate ranges from 10–75% in various reports (median 50%). Ten year survival rates are significantly lower than 5 year survival rates suggesting that late relapses are common. Histology appears to be the most significant factor for overall survival. Patients with borderline ovarian tumours show higher survival rates than adenocarcinoma [49]. Patients with noninvasive implants, especially from low-grade mucinous tumours, survive longer [50]. The prognostic significance of classifying patients into DPAM and PMC has been discussed earlier.

Conclusion

PMP is a condition characterized by mucinous ascites and peritoneal implants, now thought to be of appendiceal origin. Surgical debulking is the recommended treatment both for primary and recurrent tumours. Intraperitoneal chemotherapy appears to improve survival after surgical debulking of tumours with favourable histology. Mitomycin C or 5-fluorouracil may be used, with no evidence for higher efficacy of either agent over the other. The role of systemic chemotherapy continues to be poorly defined with no evidence of improvement in survival. Platinum-based ovarian cancer regimens are currently recommended in cases where systemic chemotherapy is used. Recent trends include the use of ECF, the results of which are awaited. The use of other modalities such as immunotherapy, intraperitoneal radioisotopes and hyperthermic chemotherapy, remains controversial.

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