Ann Surg Oncol (2020) 27:1793–1797
https://doi.org/10.1245/s10434-020-08319-7

Annals of
SURGICAL ONCOLOGY
OFFICIAL JOURNAL OF THE SOCIETY OF SURGICAL ONCOLOGY

ORIGINAL ARTICLE – PERITONEAL SURFACE MALIGNANCY

The Chicago Consensus on Peritoneal Surface Malignancies: Management of Desmoplastic Small Round Cell Tumor, Breast, and Gastrointestinal Stromal Tumors

Chicago Consensus Working Group

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ABSTRACT The Chicago Consensus Working Group provides multidisciplinary recommendations for the management of desmoplastic small round cell tumor, breast, and gastrointestinal stromal tumor specifically related to peritoneal surface malignancy. These guidelines are developed with input from leading experts including surgical oncologists, medical oncologists, pathologists, radiologists, palliative care physicians, and pharmacists. These guidelines recognize and address the emerging need for increased awareness in the appropriate management of peritoneal surface disease. They are not intended to replace the quest for higher levels of evidence.

DESMOPLASTIC SMALL ROUND CELL TUMOR

This article provides multidisciplinary recommendations for the management of peritoneal surface malignancies of desmoplastic small round cell, breast, and gastrointestinal stromal origin and constitutes 1 article in a series composed by the Chicago Consensus Working Group for the Management of Peritoneal Surface Malignancies.[1–10] Information regarding formation of the Chicago Consensus Group and explanation of the working group's consensus methodology is discussed elsewhere.[1,2]

The collaborators for the Chicago Consensus Working Group are listed in the acknowledgments.

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First Received: 4 July 2019;
Published Online: 13 April 2020

INTRODUCTION

Desmoplastic small round cell tumor (DSRCT) is an aggressive, rare form of sarcoma that primarily affects children, adolescents, and young adults. Its tissue of origin is unknown. It is characterized by the translocation t(11;22)(p13;q12), which results in a fusion protein joining EWS with WT1.

Patients with suspected DSRCT are evaluated for their performance status and hepatic and renal function and are considered for locoregional therapies only if they have no extra-abdominal disease. Although the presence of hepatic metastases does not preclude surgical resection, complete cytoreduction with complete resection of these metastases is essential. Preoperative imaging modalities include cross-sectional imaging such as computed tomography (CT) and magnetic resonance imaging (MRI). Positron emission tomography (PET) with FDG is also indicated for this disease. Although the presentation of disease could be synchronous or metachronous, it is very often synchronous. Management algorithms usually apply to patients with synchronous disease. (See *Peritoneal Metastasis of DSRCT Management Pathway*, Fig. 1).

Principles of Pathology

The diagnosis of DSRCT is usually made with core-needle tissue biopsy specimens obtained under image guidance or via a laparoscopy or laparotomy. Immunohistochemistry and translocation analysis are essential to make the diagnosis and distinguish DSRCT from Ewing sarcoma and other sarcomas that have a similar appearance. DSRCTs are characterized by solid nests of round or oval cells surrounded by cellular desmoplastic stroma. Necrosis, cystic degeneration, glandular arrangements, and occasional signet ring–like cells are often present. Staining with EWS-WT1 fusion protein is diagnostic.[11]

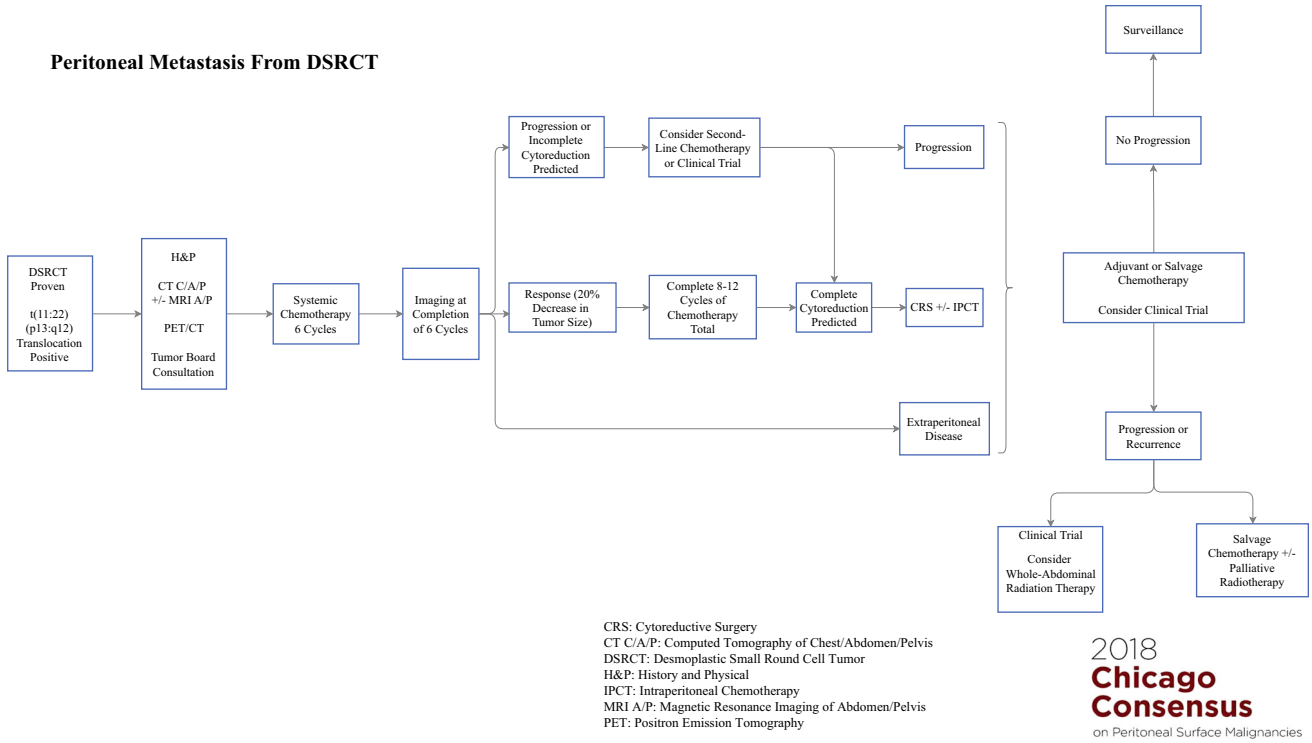

FIG. 1 Peritoneal metastasis from DSRCT management pathway

Principles of Chemotherapy

DSRCT frequently presents as advanced disease. It is remarkably chemosensitive, so chemotherapy is the first line of treatment for most patients. Chemotherapy used to treat Ewing sarcoma is recommended and usually consists of a combination of vincristine, doxorubicin, and cyclophosphamide alternating with ifosfamide and etoposide every 2–3 weeks. Chemotherapy is administered for 6 cycles, and if response is achieved (> 20% decrease of tumor size), a total of 8–12 cycles is completed before attempting surgery, or until there is a plateau of response.[12] Because this regimen was developed primarily in pediatric patients, older adults may not tolerate this intense regimen. In older patients, vincristine with doxorubicin and ifosfamide is a reasonable alternative with excellent response. Most regimens include combinations of vincristine, dactinomycin, cyclophosphamide, doxorubicin, ifosfamide, and etoposide. Second-line regimens include temozolomide/irinotecan, cyclophosphamide/topotecan, and high-dose ifosfamide.[13]

Salvage regimens include cyclophosphamide/vinorelbine, gemcitabine/docetaxel, and dacarbazine. Many DSRCTs overexpress vascular endothelial growth factor receptor 2, and some patients' cancers respond to sunitinib, sorafenib, or pazopanib.[14]

After complete cytoreduction, adjuvant chemotherapy (irinotecan/temozolomide) for 3 cycles may be considered (Fig. 1).

Principles of Radiotherapy

Whole-abdominal radiation (28–30 Gy) has been applied to patients with DSRCT with peritoneal disease after complete cytoreductive surgery. Recent multivariate analysis has shown that whole-abdominal radiation does not improve overall survival, but it appears to delay extrahepatic intraabdominal tumor recurrence and may improve quality of life for selected patients.[15] These end points must be balanced by the potential associated adverse effects.

Principles of Surgery and Intraperitoneal/Adjuvant Chemotherapy

Cytoreductive surgery for patients with DSRCT involves meticulous planning and coordination with anesthesia considerations. Burden of disease is measured by standard peritoneal staging scores such as the peritoneal cancer index (PCI) or simplified PCI score. Standard peritonectomy techniques and visceral resection procedures are employed. The CC or R score, with an intention to leave less than 2.5 mm of disease (CC1 or R1), is used to document the completeness of cytoreduction.

Cytoreduction of DSRCT is strikingly different from cytoreduction of other peritoneum-based tumors. DSRCT is very bulky and almost always has a high burden of disease in the pelvis and the pouch of Douglas. Unlike other peritoneal malignancies, DSRCT can be successfully dissected away from the ureters, bladder, and rectum without resection of these structures. Therefore, attempts should be made to complete the pelvic dissection before abandoning complete cytoreduction or proceeding with pelvic exenteration. Surgical experience with DSRCT in particular is important to achieve complete cytoreduction (CC0, R0), which should be the preferable goal for surgery in these patients. Unresectable periportal disease does not preclude cytoreduction but can be addressed with focal radiation postoperatively (Fig. 1).

A commonly used hyperthermic intraperitoneal chemotherapy (HIPEC) regimen includes cisplatin 100 mg/m^2 administered for 90 min at 41 °C. In a recently published phase 2 clinical trial, this regimen resulted in a 30-month overall survival rate of 78%.[16] Nephroprotection is provided with intravenous sodium thiosulphate given 30 min before the perfusion and for 12 h after the perfusion.[17,18] Other described but poorly studied HIPEC regimens include cisplatin 120 mg plus mitomycin 75 mg/m^2 for 30 min and oxaliplatin 300–460 mg/m^2 for 30 min, occasionally used in combination with irinotecan 200 mg/m^2. According to a retrospective analysis, these non-cisplatin-based regimens may or may not be as effective.[19] Early postoperative chemotherapy regimens are also poorly studied but include cisplatin 15 mg/m^2 and doxorubicin 0.1 mg/kg given for 5 days.

MISCELLANEOUS PERITONEAL DISEASE

Breast

Peritoneal carcinomatosis from breast cancer is generally associated with a history of high-grade lobular carcinoma, mostly tumors larger than 5 cm with positive lymph nodes. It can present from 5 to 10 years after diagnosis and in most cases is also associated with extraperitoneal metastasis. Generally, this presentation represents a life-threatening event with a very high mortality rate.[20] Evidence for cytoreductive surgery and intraperitoneal chemotherapy is limited,[21] so these procedures are not recommended except in a clinical trial. Treatment for these patients includes systemic chemotherapy, clinical trials, and best supportive care. Testing for *HER2* is recommended because the result might lead to treatment with pertuzumab and trastuzumab in addition to other chemotherapy agents, which include docetaxel, paclitaxel, doxorubicin, capecitabine, gemcitabine, cyclophosphamide, and epirubicin (single or combination agents). Tumor board consultation is highly advocated to choose the best regimen for each patient.

Gastrointestinal Stromal Tumor

Gastrointestinal stromal tumor has the highest incidence and prevalence of gastrointestinal tract sarcomas, accounting for approximately 5% of all mesenchymal tumors. Metastatic disease is not uncommon, and 20% of patients with metastatic disease have peritoneal involvement.[22] Since the introduction of tyrosine kinase inhibitors (TKIs) in 2002, a shift has occurred in the management of this disease. Peritoneal sarcomatosis has a poor prognosis, and most patients with this condition are considered to have terminal illness. However, in patients with sarcomatosis that originates from gastrointestinal stromal tumors, the scenario is slightly different because of the availability of TKIs for some of these patients. If sarcomatosis is suspected, a core-needle biopsy performed via interventional radiology or laparoscopy is required to identify *KIT* and *PDGFR* mutations. Patients should have a full-body assessment with abdominal CT, MRI, and/or PET-FDG/CT and chest CT. Recommended TKI agents are imatinib (400–800 mg), sunitinib (50 mg), and regorafenib (160 mg)[23] in an escalating manner if progression is assessed. For patients with a *PDGF* D842V mutation or an *NF1* or *SDH* deletion, imatinib is not indicated and a clinical trial or observation is offered. Only patients in whom response or stable disease is achieved with the former therapeutic agents and in whom a complete cytoreduction is predicted will be candidates for cytoreduction surgery. In these patients, CC0 resection is encouraged because of improved outcomes in this population.[24] Intraperitoneal chemotherapy has not shown a survival benefit for these patients and should not be recommended for patients not in a clinical trial.[25]

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FUNDING The Irving Harris Foundation and the University of Chicago

DISCLOSURES James C. Cusack reports grants from Lumicell Inc. outside the submitted work. Carla Harmath serves on the medical advisory council of Accumen. Hedy Kindler reports personal fees and non-financial support from Inventiva, AstraZeneca, Boehringer Ingelheim, Merck, and Paredox; personal fees from Aldeyra Therapeutics, Bayer, BMS, Erytech, Five Prime Therapeutics, Ipsen Pharmaceuticals, Kyowa, and MedImmune; and funds to support clinical trials at her institution from Aduro, AstraZeneca, Bayer, BMS, Deciphera, GSK, Lilly, Merck, MedImmune, Polaris, Verastem, and Blueprint, all outside the submitted work. Aytekin Oto reports grants from Philips Healthcare, Guerbet, and Profound Healthcare, and serves as a medical advisory board member for Profound Healthcare, all outside the submitted work. Nita Ahuja reports grant funding from Cepheid and Astex, has served as a consultant to Ethicon, and has licensed methylation biomarkers to Cepheid. Jesus Esquivel reports personal fees from Eight Medical. The remaining authors disclosed no conflicts of interest.

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