Colorectal cancer

Colorectal cancer starts in the colon or the rectum. These cancers can also be called colon cancer or rectal cancer, depending on where they start. Colon cancer and rectal cancer are often grouped together because they have many features in common. Cancer starts when cells in the body start to grow out of control.

Colorectal adenocarcinoma is a type of cancer that starts in the large intestine. Colorectal stands for your colon and rectum, which make up the large intestine.

BST2 played a vital role in CRC progression and might be a predictable marker for immunotherapy [1)]

CT-defined sarcopenia is an independent predictor for worse OS in patients with rectal cancer. Future studies with a more stringent definition of sarcopenia are required to further confirm these findings [2)].

Several groups have reported cases of rectal cancer with carcinomatous involvement of the lumbosacral plexus and sciatic, obturator, pudendal, or spinal nerves.

Imaging studies demonstrated distinct features of tumor spread from the organ to the plexus and beyond, histological specimens demonstrated perineural involvement.

Once the tumor reaches the lumbosacral plexus, it can continue to spread proximally or distally [3)].

Types

There are different types of colon and rectal cancer, but adenocarcinoma is the most common.

The transcriptomic classification of primary colorectal cancer (CRC) into distinct consensus molecular subtypes (CMSs) is a well-described strategy for patient stratification. However, the molecular nature of CRC metastases remains poorly investigated. To this end, this study aimed to identify and compare organotropic CMS frequencies in CRC liver and brain metastases. Compared to reported CMS frequencies in primary CRC, liver metastases from CRC patients were CMS4-enriched and CMS3-depleted, whereas brain metastases mainly clustered as CMS3 and rarely as CMS4. Regarding overall survival rates, CMS4 was the most favorable subtype for patients with hepatic lesions, followed by CMS1 and CMS2. The survival of patients with brain metastases did not correlate with CMS. However, we identified a CMS3-related metabolic gene signature, specifically upregulated in the central nervous system (CNS)-infiltrating CRC, as a negative prognostic marker and potential tumor progressor. In summary, subtyping of CRC metastases revealed an organotropic CMS distribution in liver and brain with impact on patient survival. CNS-infiltrating CRC samples were enriched for CMS3 and predictive metabolic biomarkers, suggesting metabolic dysregulation of CRC cells as a prerequisite for

metastatic colonization of the brain [4].

Colorectal cancer intracranial metastases

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1)
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