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Pathological Analysis of Kidney Transplantation in Cancer Patients

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Abstract

Colorectal cancer (CRC) is a serious health problem that can happen after receiving a solid organ transplant. The objective of our study was to describe the clinical and pathological characteristics of CRC in kidney and liver transplant recipients at a tertiary and reference center. Our cohort consisted of 12 patients, 10 men and 2 women, with a mean age of 60. Ten of the patients underwent CRC resection. In five patients, the transplanted organ was the liver, and in seven, the kidney. Concerning endurance, patients submitted to renal transplantation were completely expired 5 years after CRC analysis, while those exposed to hepatic transplantation had an endurance of 60% at the fifth year.

Keywords: Colorectal cancer; Tumour microenvironment; Transplantation

Introduction

Seven patients had advanced disease (stage III/IV) and a significant amount of necrosis, as revealed by pathology. The tumor microenvironment was disturbed, with no PD-L1 expression, natural killer cells, or any inflammatory infiltrate. 40 percent of CRC patients had microsatellite instability and expressed cancer stem cell markers (CD133, CD44, and ALDH1), as well as P53 and KRAS mutations in 50 percent of cases [1].

After kidney and liver transplantation, CRC cancer is a rare, aggressive, and fatal condition. These patients should be monitored on a regular basis. Colorectal cancer (CRC) is a major health concern because it is the third most common type of cancer, accounts for 10.2% of all cancer cases, and is the second leading cause of cancer-related death. Despite all the advancements in clinical and biological knowledge, CRC remains a significant problem despite the availability of effective treatment and prevention methods, and the mortality rate associated with this kind of cancer is on the rise [2]. The pathophysiology of CRC has been extensively studied, and four genetic pathways have been identified as risk factors for the disease: chromosomal insecurity, mutations of DNA bungle fix qualities, proto-oncogene and PI3K ways. Solid organ transplantation (SOT) comes with some risks because cancer is one of the leading causes of death, especially hematopoietic tumors, most of which are caused by oncogenic viruses being activated by immunosuppression [3]. Immunosuppression has been argued to increase the risk of CRC in recent years, particularly when induced by medication. This is due to the immunosuppressive drugs' resistance to chemotherapy as well as the diminished immune system activity.

Methods

The purpose of this study was to describe the clinical and pathological characteristics of kidney and liver transplant recipients at our tertiary and reference center. Patients with CRC diagnosed at CHUC between January 2004 and December 2016 who had previously undergone liver and kidney transplantation were examined clinically and pathologically. The patient had to be over the age of 18 and have a biopsy that showed CRC after the transplant. Excluded were patients who had CRC prior to transplantation [4].

There were 12 patients in the study, 10 of them men and 2 of them women, with a mean age of 60.54 13.41 years (range 34–78); Only three patients were younger than 50 (25%). One patient had kidney and liver transplants, but because the liver transplant was the first, it was included in the liver transplantation cohort for this study [5].

In accordance with the recommendations of the European Society for Medical Oncology (ESMO), the National Comprehensive Cancer Network (NCCN), and the American Society of Colon and Rectal Surgeons (ASCRS), colorectal carcinoma management, including neoadjuvant and adjuvant treatments (radiotherapy and chemotherapy) and follow-up, is carried out. Pathological analysis The patient material was reviewed with the analysis of CRC tissue that was available in paraffin blocks from surgical specimens. After a routine staining with hematoxylin and eosin (H&E), the samples were used. There was no prior knowledge of the patient's data (such as the type of transplant) or outcome during the blinded histopathologic review. Koelzer and Lugli's criteria for defining a histological growth pattern were as follows: pushing tumor growth and infiltrative tumor growth (dissection of the bowel wall structures by tumor tissue with little or no desmoplastic stromal response) are two types of tumor growth. This property could only be evaluated using surgical specimens because it was necessary to observe the interaction between the tumor and the host [6].

Discussion

Cancer rot was surveyed by Schneider and Langner central corruption (<10% of the growth region), moderate (10-30% of cancer region) and broad (>30% of the growth region). With the Klintrup criteria of low grade (absence or mild inflammatory infiltrate) and high grade (moderate or severe inflammatory infiltrate, with tumor destruction), the inflammatory response was also evaluated at the tumor's invasive border. According to the findings of Fernández-Aceero et al., tumor-associated osteophils were evaluated. Low eosinophil counts (less than 10/HPF) are considered to be of grade 1, while high eosinophil counts (more than 50/HPF) are considered to be of grade 3. The only specimens used for the evaluation were surgical ones.

According to the ESMO Clinical Guidelines for Familial Risk Colorectal Cancer, microsatellite instability (MSI) was tested: Primarily, an immunohistochemical panel consisting of PMS, MLH1,

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MSH2, and MSH6 was utilized. If MLH1 was absent, subsequent immunohistochemical testing for BRAF was carried out. All of the patients underwent the MSI examination. The P53 mutation was determined by performing an immunohistochemical analysis. Overexpression of P53 and the presence of P53 mutations were deemed to occur in more than 75% of tumor cells. Immunohistochemical staining for CD44, CD133, and ALDH1 was used to determine whether or not there were cancer stem cells (CSCs). When one of the markers was expressed in more than half of the tumor cells, this indicated a high number of CSCs.

Conclusion

The presence of normal executioner (NK) cells was assessed as expressed by The number of NK cells per mm2 was counted, and tumors were deemed positive when they had more than four NK cells per mm2. The only specimens used for the evaluation were surgical ones. In accordance with the guidelines established for non-small cell lung cancer, PD-L1 expression was graded as follows: score 0 for absence of staining; score 1: membranous and/or cytoplasmic staining of more than 1% and less than 50% of the tumoral cells staining less than half of the tumoral cells score 2 All of the patients completed the staining.

The following antibodies were used in indirect multimeric detection, biotin-free, peroxidase-conjugated immunohistochemistry on four-millimeter-thick tissue sections from a representative tumor block using a Ventana Marker Platform Bench Mark ULTRA IHC/ISH: CD68 (KP1, Ventana, Tucson, AZ-USA), P53 (DO7, Ventana, AZ-USA), PD-L1 CD44 (SP37, Ventana, AZ-USA), ALDH1 (EP1933Y, AbCam, Cambridge, UK), MLH1 (M1, Ventana, AZ-USA), MSH2 (G219-1129, Ventana, AZ-USA To get the best signal-to-noise ratio, adequate controls for each antibody were used in accordance with the instructions provided by the manufacturer. All stained slides were examined with a Nikon Eclipse 50i light microscope, and a Nikon-Digital Sight DS-Fi1 camera was used to take pictures. KRAS changes were assessed depending on Polymerase Chain Response (PCR) on a 10 µm thick cut segment of paraffin-inserted growth following an area of 25-300 mm2 with at least 20% of growth cells. All patients underwent the genetic investigation. Sequencing for NRAS and BRAF mutations was carried out if no KRAS mutation was found.

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