

The Spontaneous Regression of Primary Gastrointestinal Malignancies: An Observational Review

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Abstract

The spontaneous regression or remission (SR) of cancer, often described as the partial or complete disappearance of a malignant tumor in the absence of all medical treatment and therapy, is a well-documented phenomenon. With efforts ongoing to establish cancer treatments that limit undesirable outcomes and adverse effects, these uncommon occurrences of SR carry significant implications for novel therapies and warrant further investigation. While several case studies have reported instances of SR in gastrointestinal (GI) malignancies, a comprehensive review of previous manifestations of SR in the GI tract remains lacking. The inclusion criteria for the rare phenomenon are also in need of an appropriate update that takes recent scientific advancements and emerging new medical technologies into account. Our analysis of 390 cases of SR in the GI tract focuses primarily on neoplasms of the hepatobiliary system and proposes an updated version of the older inclusion criteria for spontaneous regression.

Categories: Internal Medicine, Gastroenterology, Oncology

Keywords: hepatobiliary system, neoplasms, oncology, gastroenterology, hepatology, gastrointestinal cancers, hepatocellular carcinoma, spontaneous remission, spontaneous regression

Introduction And Background

In 2021 alone, 372,470 new cases of primary gastrointestinal (GI) cancer were reported worldwide, and approximately 124,348 deaths occurred as a result, comprising 19.6% and 20.4% of total new cancer cases and deaths, respectively [1]. Nonetheless, with the introduction of various novel diagnostic, therapeutic, and antineoplastic modalities, the mortality rates of GI cancers have declined significantly over the past several years [2]. These new modalities have led to a greater understanding of the pathogenesis of cancer and of achieving remission. Spontaneous regression (SR) is defined as the complete or partial disappearance of a primary and/or disseminated lesion of a histologically diagnosed metastatic disease in the absence of any medical treatment or therapy known to have antitumor effects. Spontaneous regression has been found to occur throughout the entire body, including the GI tract [3]. But it is not equivalent to a cure, as cancer may reappear or spread elsewhere in the body. The frequency of spontaneous regression varies based on the type of cancer, as it is most commonly reported in renal cell carcinoma, melanoma, and neuroblastoma [3-5]. Occurring at a rate of about one out of every 60,000 to 100,000 cases of all cancers, these extremely rare occurrences of SR have the potential to serve as an instructive *in vivo* model of biological tumor regulation and control [6].

A number of putative mechanisms have been proposed for the observed spontaneous disappearance of malignancies, including inflammation, apoptosis, ischemia, and immunological responses [7]. Other mechanisms proposed to cause SR include epigenetic modifications, hormonal responses, oncogenes, tumor suppressors, cytokines and growth factors, and psychological mechanisms. Unfortunately, several of these postulated mechanisms are based on association and speculation alone, with the exact mechanistic modalities surrounding the SR of GI cancer yet to be elucidated. Regardless, an immunological anti-tumoral response of a patient's body to specific malignancies is among the most prevalently described mechanistic hypotheses for the observed spontaneous disappearance of neoplasms. Since the very inception of the term, spontaneous regression has historically been speculated to be a dynamic interplay of immunological anti-tumor responses. In 1956, Everson and Cole defined the criterion for SR as the partial or complete disappearance of a malignant tumor in the absence of all treatment or in the presence of therapy that is considered inadequate to exert a significant influence on neoplastic disease. At that time, they theorized that the phenomenon must be an opportunistic by-product of an activated immune response. Cases of SR linked to infections have significantly influenced the discovery of several different anticancer therapies that facilitate the targeting of cancer cells by the host's immune system. For example, immune checkpoint inhibitors have revolutionized modern cancer treatment by targeting inhibitory receptors (e.g., PD-1, CTLA-4, LAG-3), ligands (e.g., PD-L1) expressed on T cells, antigen-presenting cells, and tumor cells, which result

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in an anti-tumor response by stimulating the host immune system.

Focusing chiefly on malignancies of primary GI origin, this observational review of the literature hopes to bring further attention to the phenomenon of SR while also identifying some potential mechanisms that have been purported to contribute to this largely unreported phenomenon. Secondly, this comprehensive review aims to introduce a revised and up-to-date version of the older inclusion criteria for SR throughout the body. This updated criterion has been modified in a way that takes into account recent scientific advancements and emerging new medical technologies, with the intent that it will also be easy to follow for physicians and clinical researchers alike. Finally, this study seeks to broaden the scope of how SR is perceived by clinicians and members of the medical community by encouraging a holistic view of the exceptionally rare phenomenon as a dynamic interplay of various modalities.

Review

Materials and methods:

Search Strategy

A literature search across five databases (PubMed, Medline, Google Scholar, Semantic Scholar, and Jstage) was performed employing the following main keywords: gastrointestinal cancer, spontaneous regression, spontaneous remission, spontaneous necrosis, and abscopal effect. A full list of searched keywords is included in Appendix A. The clinical characteristics of each occurrence of SR within the GI pathway and the related long-term outcomes were then extracted. Articles were excluded if any systemic treatment was used or if any treatment directed at the lesion was utilized before the documented regression. All diseases were limited to the GI tract, spanning the oral cavity, esophagus, stomach, liver, bile duct, gallbladder, pancreas, mesenteries, peritoneum, small intestine, colon, and rectum. A manual search of each work's citations was performed, utilizing additional published works listed in the supplementary materials or reference sections of each of the aforementioned studies. No restriction was applied to the date of publication, the form of publication, or the primary language of the publication.

| Patient characteristics* | Oral (n=46) | Esophageal (n=11) | Gastric (n=38) | Peritoneal (n=3) | Hepatobiliary (n=212) | Pancreatic (n=10) | Small bowel (n=10) | Colorectal (n=60) | Total (n=390) |
|-----------------------------------|-------------|-------------------|----------------|------------------|-----------------------|-------------------|--------------------|-------------------|---------------|
| Age (Mean (SD)) | 61.2 (17.4) | 59.0 (16.2) | 60.1 (19.0) | 47.7 (15.9) | 64.7 (13.5) | 50.4 (18.2) | 51 (16.2) | 62.1 (15.0) | 63.1 (14.7) |
| Age group (n (%)) | | | | | | | | | |
| <19 | 1 (2.2%) | 0 | 1 (2.6%) | 0 | 0 | 0 | 0 | 1 (1.7%) | 3 (0.8%) |
| 19-34 | 3 (6.5%) | 1 (9.1%) | 2 (5.3%) | 0 | 6 (2.8%) | 2 (20.0%) | 1 (10.0%) | 1 (1.7%) | 16 (4.1%) |
| 35-44 | 1 (2.2%) | 1 (9.1%) | 7 (18.4%) | 2 (66.7%) | 6 (2.8%) | 1 (10.0%) | 2 (20.0%) | 7 (11.7%) | 27 (6.9%) |
| 45-54 | 11 (23.9%) | 1 (9.1%) | 2 (5.3%) | 0 | 21 (9.9%) | 1 (10.0%) | 1 (10.0%) | 7 (11.7%) | 44 (11.3%) |
| 55-64 | 8 (17.4%) | 4 (36.4%) | 10 (26.3%) | 0 | 49 (23.1%) | 2 (20.0%) | 5 (50.0%) | 17 (28.3%) | 95 (24.4%) |
| 65-74 | 9 (19.6%) | 2 (18.2%) | 5 (13.2%) | 1 (33.3%) | 82 (38.7%) | 1 (10.0%) | 1 (10.0%) | 13 (21.7%) | 114 (29.2%) |
| 75-84 | 11 (23.9%) | 2 (18.2%) | 10 (26.3%) | 0 | 43 (20.3%) | 1 (10.0%) | 0 | 12 (20.0%) | 79 (20.3%) |
| 85+ | 2 (4.3%) | 0 | 1 (2.6%) | 0 | 4 (1.9%) | 0 | 0 | 2 (3.3%) | 9 (2.3%) |
| Sex (n (%)) | | | | | | | | | |
| Male | 25 (54.3%) | 8 (72.7%) | 23 (60.5%) | 2 (66.7%) | 168 (79.2%) | 6 (60.0%) | 5 (50.0%) | 35 (58.3%) | 272 (69.7%) |
| Female | 21 (45.7%) | 3 (27.3%) | 15 (39.5%) | 1 (33.3%) | 44 (20.8%) | 4 (40.0%) | 5 (50.0%) | 25 (41.7%) | 118 (30.3%) |
| Site of Regression (n (%)) | | | | | | | | | |
| Primary tumor/Recurrence | 41 (89.1%) | 8 (72.7%) | 35 (92.1%) | 0 | 194 (91.5%) | 10 (100.0%) | 7 (70.0%) | 48 (80.0%) | 288 (73.8%) |

| | | | | | | | | | |
|--------------------------------------|------------|-------------|------------|------------|-------------|-----------|-------------|------------|-------------|
| Lung metastases | 2 (4.3%) | 3 (27.3%) | 0 | 1 (33.3%) | 28 (13.2%) | 0 | 1 (10.0%) | 2 (3.3%) | 37 (9.5%) |
| Liver metastases | 0 | 0 | 1 (2.6%) | 1 (33.3%) | 2 (0.9%) | 2 (20.0%) | 2 (20.0%) | 10 (16.7%) | 18 (4.6%) |
| Lymph metastases | 5 (10.9%) | 2 (18.2%) | 1 (2.6%) | 0 | 2 (0.9%) | 0 | 3 (30.0%) | 2 (3.3%) | 15 (3.8%) |
| Other metastases | 1 (2.2%) | 1 (9.1%) | 1 (2.6%) | 1 (33.3%) | 13 (6.1%) | 0 | 1 (10.0%) | 6 (10.0%) | 24 (6.2%) |
| Extent of regression (n (%)) | | | | | | | | | |
| Complete | 43 (93.5%) | 9 (81.8%) | 32 (84.2%) | 2 (66.7%) | 156 (73.6%) | 8 (80.0%) | 10 (100.0%) | 57 (95.0%) | 317 (81.3%) |
| Partial | 3 (6.5%) | 2 (18.2%) | 6 (15.8%) | 1 (33.3%) | 56 (26.4%) | 2 (20.0%) | 0 | 3 (5.0%) | 73 (18.7%) |
| Histological profile (n (%)) | | | | | | | | | |
| Carcinoma | 11 (23.9%) | 8 (72.7%) | 8 (21.1%) | 0 | 198 (93.4%) | 9 (90.0%) | 1 (10.0%) | 54 (90.0%) | 289 (74.1%) |
| Primary lymphoma | 28 (60.9%) | 1 (9.1%) | 24 (63.2%) | 0 | 4 (1.9%) | 0 | 6 (60.0%) | 4 (6.7%) | 67 (17.2%) |
| NET | 1 (2.2%) | 0 | 6 (15.8%) | 0 | 2 (0.9%) | 1 (10.0%) | 1 (10.0%) | 1 (1.7%) | 12 (3.1%) |
| Other | 6 (13.0%) | 2 (18.2%) | 0 | 3 (100.0%) | 8 (3.8%) | 0 | 2 (20.0%) | 1 (1.7%) | 22 (5.6%) |
| Period of regression (n (%)) | | | | | | | | | |
| <1 month | 4 (8.7%) | 0 | 3 (7.9%) | 0 | 6 (2.8%) | 0 | 0 | 0 | 13 (3.3%) |
| 1-1.5 months | 3 (6.5%) | 1 (9.1%) | 5 (13.2%) | 0 | 11 (5.2%) | 1 (10.0%) | 0 | 9 (15.0%) | 30 (7.7%) |
| 2-5 months | 2 (4.3%) | 2 (18.2%) | 6 (15.8%) | 1 (33.3%) | 27 (12.7%) | 0 | 3 (30.0%) | 13 (21.7%) | 54 (13.8%) |
| 6-11 months | 6 (13.0%) | 3 (27.3%) | 3 (7.9%) | 0 | 16 (7.5%) | 1 (10.0%) | 3 (30.0%) | 1 (1.7%) | 33 (8.5%) |
| 12-23 months | 6 (13.0%) | 2 (18.2%) | 5 (13.2%) | 0 | 41 (19.3%) | 2 (20.0%) | 0 | 9 (15.0%) | 65 (16.7%) |
| 24-35 months | 6 (13.0%) | 1 (9.1%) | 4 (10.5%) | 1 (33.3%) | 30 (14.2%) | 1 (10.0%) | 0 | 3 (5.0%) | 46 (11.8%) |
| 36-47 months | 5 (10.9%) | 0 | 2 (5.3%) | 0 | 14 (6.6%) | 1 (10.0%) | 1 (10.0%) | 5 (8.3%) | 28 (7.2%) |
| 48 months+ | 8 (17.4%) | 1 (9.1%) | 8 (21.1%) | 1 (33.3%) | 33 (15.6%) | 4 (40.0%) | 2 (20.0%) | 17 (28.3%) | 74 (19.0%) |
| Unspecified | 6 (13.0%) | 1 (9.1%) | 2 (5.3%) | 0 | 34 (16.0%) | 0 | 1 (10.0%) | 3 (5.0%) | 47 (12.1%) |
| Malignancy recurrence (n (%)) | | | | | | | | | |
| Reported | 5 (10.9%) | 0 | 1 (2.6%) | 0 | 14 (6.6%) | 1 (10.0%) | 2 (20.0%) | 1 (1.7%) | 24 (6.2%) |
| Not reported | 41 (89.1%) | 11 (100.0%) | 37 (97.4%) | 3 (100.0%) | 198 (93.4%) | 9 (90.0%) | 8 (80.0%) | 59 (98.3%) | 366 (93.8%) |

TABLE 1: Baseline characteristics of spontaneous regression within the study sample

Inclusion Criteria

Only publications that described the true SR of a histologically confirmed GI cancer were included following the inclusion criteria depicted in Figure 1. These criteria are based on the original criteria proposed by Cole, modified to emphasize histological diagnosis, and adapted to fit multiple clinical scenarios (2). These criteria are summarized as follows: (1) Partial or complete disappearance of the primary tumor or secondary metastasis was radiographically or pathologically demonstrated in the absence of systemic therapy; (2) localized therapy to the lesion prior to the observed shrinkage was excluded; and (3) the malignant neoplasm was histologically proven at some point during this course. Patients with primary neoplasms histologically determined to have originated from outside the GI pathway but demonstrating SR were excluded, even if they demonstrated SR of a secondary metastasis within the GI tract. Patients demonstrating regression of an extra-digestive lesion, histologically determined to have arisen in the GI tract, were included regardless of whether the primary GI lesion had also regressed.

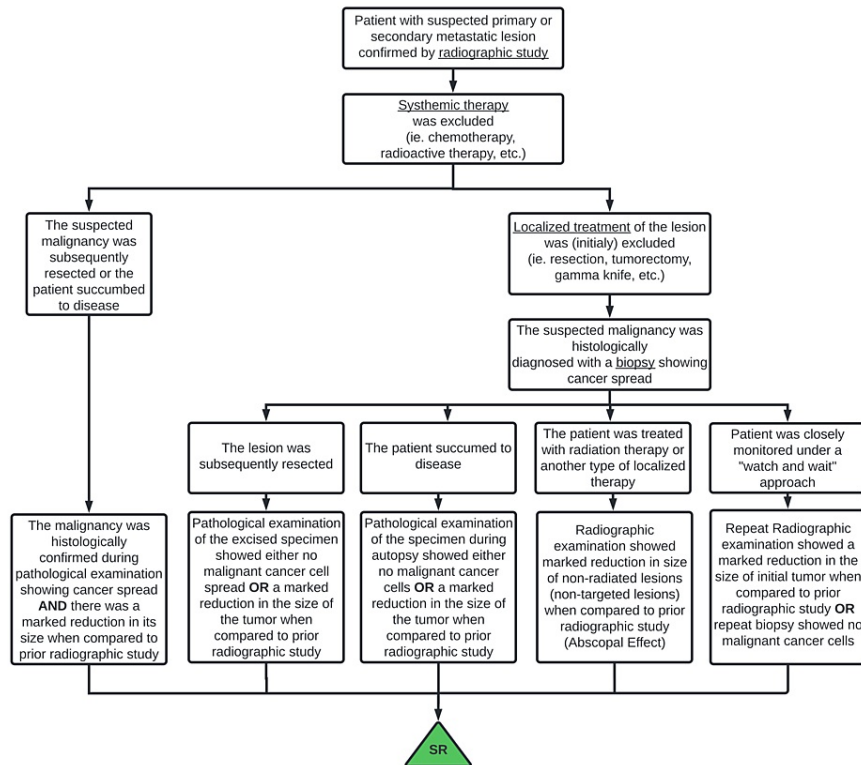


FIGURE 1: Clinician guidelines or criteria for reporting spontaneous regression

From a clinician's initial encounter with a patient with a suspected cancerous lesion to the demonstration of tumor shrinkage or disappearance, potential clinical manifestations of spontaneous regression are schematically investigated and shown. Original figure by the authors.

Data Extraction and Analysis

The following information was extracted and recorded from each article: patient age and sex, location and histological typing of the primary tumor, the site of regression, the period of regression or remission, and the etiological mechanism of regression proposed by the author. The demonstrated recurrence of cancer was also noted in some patients. Limited to each author's interpretation and the duration of follow-up included in each study, the period of remission was defined as one of the following, whichever was found to be the longest: (1) the total period of time during which the tumor demonstrated a shrinkage in size, beginning with the date when the tumor's size was found to be at its maximum to the date when the tumor's size was found to be at its minimum, (2) The total period of time between the partial or complete disappearance of cancer and the most recent follow-up date in which the patient continued to show no signs of metastatic spread or recurrence of the malignancy; (3) the total period of time during which the tumor demonstrated a shrinkage in size prior to its resection, from the proposed date when the tumor's size was found to be at its maximum size to the date of the resection. After the tumor was resected, the specimen was pathologically found to have shrunk or disappeared.

Results

Of the 390 cases of SR of GI malignancies reported meeting our criteria, a majority were noted in men (272 cases, 69.7%) compared to women (118 cases, 30.3%). The mean patient age was 63 years, with a majority of patients between 65 and 74 years of age (114 cases, 29.2%) or 55 and 64 years of age (95 cases, 24.4%). Overall, the literature search demonstrated a global incidence of SR, with cases spanning all six inhabited continents.

All reported cases detailing the SR of GI malignancies throughout the clinical literature are comprehensively reviewed in Appendix B, with pertinent findings summarized in Table 1. These reported cases of SR included various cases of carcinoma (289 cases, 74.1%), primary gastrointestinal and oral lymphomas (67 cases, 17.2%), and a few neuroendocrine tumors (12 cases, 3.1%), among other primary gastrointestinal cancers (22 cases, 5.6%). Hepatocellular carcinoma (HCC) represented almost half of all reported cases of SR in GI cancers (193 cases, 49.5%). Several rare forms of cancer, including extramedullary plasmacytoma (EMP), peritoneal alveolar soft-part sarcoma (ASPS), and gastric gastrinoma, were also observed to spontaneously regress. A complete list of reported histological manifestations of GI malignancies recorded to have undergone SR is in Figure 2.

| Proposed mechanism | n (%) |
|-----------------------------------|-------------|
| Immunological | 202 (51.8%) |
| Abscopal effect | 10 (2.5%) |
| Endocrine factors | 4 (1.0%) |
| Restored immunogenicity | 18 (4.6%) |
| Eradication of oncogenic virus | 7 (1.8%) |
| Fever/Infection | 35 (9.0%) |
| Inflammatory response | 14 (3.6%) |
| Transfusions | 1 (0.3%) |
| Treatment of primary/Metastases | 7 (1.8%) |
| Other/Not specified | 106 (27.2%) |
| Ischemic | 146 (37.4%) |
| Anti-angiogenic factors | 3 (0.8%) |
| Vascular ischemia/ thrombosis | 26 (6.7%) |
| Tumor ablation/Biopsy/Angiography | 44 (11.3%) |
| Tumor hypoxia/ Hypoperfusion | 30 (7.7%) |
| Tumor microenvironment disruption | 3 (0.8%) |
| Unpredictable/Rapid growth | 14 (3.6%) |
| Other/Not specified | 26 (6.7%) |
| Idiopathic | 92 (23.6%) |
| Apoptotic tumor cell death | 6 (1.5%) |
| Dislodged | 10 (2.6%) |
| Drugs | 14 (3.6%) |
| Genetic | 5 (1.3%) |
| Herbal medicines | 20 (5.1%) |
| Metabolic/Nutritional | 7 (1.8%) |
| Psychoneurological | 5 (1.3%) |
| Withdrawal of carcinogenic agent | 16 (4.1%) |
| Other | 9 (2.3%) |
| Not specified | 68 (17.4%) |

TABLE 2: Proposed mechanisms of spontaneous regression within the gastrointestinal pathway

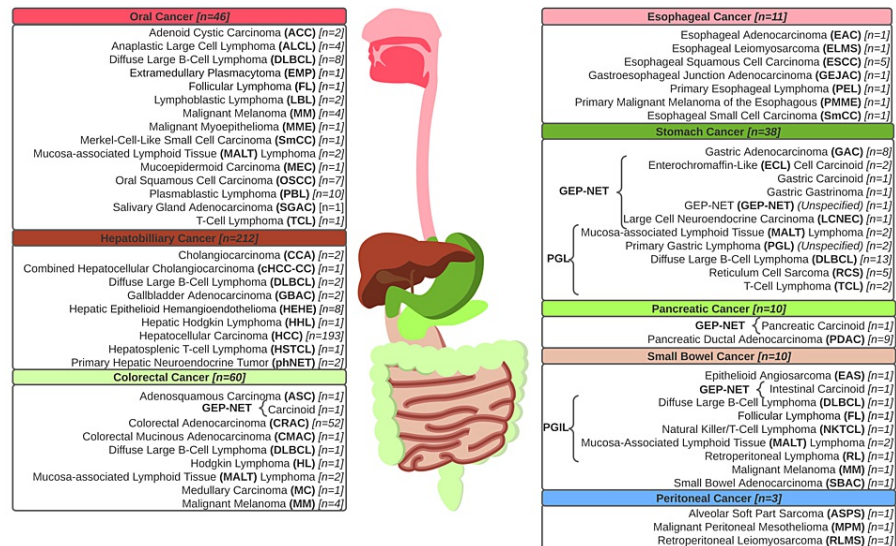


FIGURE 2: Histological manifestations of gastrointestinal malignancies are recorded to have undergone spontaneous regression throughout the clinical literature.

Biopsy-confirmed cancers of the gastrointestinal pathway, including various carcinomas, gastroenteropancreatic neuroendocrine tumors (GEP-NETs), and primary oral and gastrointestinal lymphomas, have been shown to demonstrate spontaneous regression. These cases have been observed throughout the entirety of the alimentary canal, spanning all of the organs of digestion. Cases of each distinct histological denomination were enumerated and systematically organized by the anatomical distribution of the primary lesion. Original figure by the authors.

The vast majority of cases of partial or complete regression occurred within the primary tumor (288 cases, 73.8%); nonetheless, multiple cases demonstrated regression of liver metastases (18 cases, 4.6%), lung metastases (37 cases, 9.5%), lymph metastases (15 cases, 3.8%), or other metastases (24 cases, 6.2%). The period of regression (as defined above) varied greatly in these cases, with some cases reporting just a few days of regression and others expressing several years of remission. Cancer recurrence was reported in 24 cases of SR, comprising 6.2% of total cases of SR in the GI pathway (noted with an asterisk "*" in Appendix B).

The authors proposed various putative mechanisms of SR, which are summarized in Table 2.

The majority of the reviewed authors provided at least one conjectural mechanism (322 cases, 82.6%), with most citing immunological (202 cases, 51.8%), ischemic (146 cases, 37.4%), or idiopathic (92 cases, 23.6%) processes.

Discussion

This systematic review includes the presentation of 390 cases reported in 346 scientific papers, journals, case studies, and published books. These publications were generated over the past 95 years. Interestingly, 325 cases were published in the modern era, defined as cases published in the past 30 years. Following the review of these articles, multiple common factors were revealed, including a tendency for SR to occur in patients over the age of 55 (297 patients, 76.2%), patients of the male sex (272 patients, 69.7%), and patients with primary liver tumors (209 patients, 53.6%) or secondary liver metastases (18 patients, 4.6%). The clinical features and proposed mechanisms surrounding these cases of SR within the GI pathway, along with the location and duration of remission, are documented in Appendix B. Patients within this cohort of reported cases displayed varied periods of stability, ranging from just a few days of observed partial tumor regression to several years of cancer remission, up to 20 years cancer-free.

Mechanisms of Spontaneous Regression

Historically, SR has been speculated to occur in the setting of a prolonged febrile illness due to viral or bacterial infection; nonetheless, only a fraction of cases of SR (35 patients, or 5.1%) have been attributed to a hyperthermic state and infection [8]. In cases of SR occurring during times of acute febrile infections, immune cell infiltrations and signaling cascades are postulated to lead to tumor cell death and cancer tissue necrosis via the release of interleukins, tumor necrosis factors, and interferons (specifically IL-2, IL-6, and IL-8) [9-17]. Viral infections notably induce the production of interferons, which are capable of their own

immunomodulatory effects involving macrophages, B-cells, and monocytes, alongside the induction of IL-2 receptors in some cancers [6-8]. Most recently, tumor regression has been reported after COVID-19 vaccination and infection with its wide-ranging pro-inflammatory effects on the host immune system [18-23].

Enhanced antitumoral immunogenicity is proposed to play a profound role in the involution of several GI cancers. In fact, examples displaying the correlation between SR and the elimination of immunodisruptive factors (e.g., medications, viral infection, checkpoint proteins) are perhaps the best evidence supporting the involvement of immunological mechanisms in the achieved SR of GI cancer [24,25]. Tumors have also occasionally been found to regress following systemic or localized treatment for some other disease process. For example, certain localized therapies have been observed to cause tumor regression of both the target lesion and any untreated tumors [26]. Described as the "abscopal effect," this phenomenon is purportedly mediated by a systemic anti-tumor response that follows after receiving radiation therapy for a metastatic lesion or an entirely separate neoplasm. Overall, more than half of the reported cases of SR within the GI tract have been attributed to immunological processes (202 cases, 51.8%), with authors also suggesting the involvement of endocrine factors (four cases, 1.0%) and inflammatory responses (14 cases, 3.6%).

Ischemic models of regression are also proposed to play a key role in the dynamic interplay of antitumoral mechanisms described in the SR of cancer. Tumor cells require an ample supply of blood, so limiting their blood supply and perfusion could intuitively starve the cells to death [27-29]. Consequently, systemic and tumoral hypoperfusion (30 cases, 7.7%), rapid and unpredictable growth (14 cases, 3.6%), anti-angiogenic factors (three cases, 0.8%), and vascular compromise (26 cases, 6.7%) are all theorized to lead to the SR of GI cancer [30-34]. For example, there are multiple cases of SR described as following profound systemic hypoperfusion associated with hemodialysis, surgical invasion, or GI hemorrhage [30-34]. Several reviewed cases of SR (44 patients, 11.3%) have been specifically attributed to diagnostic biopsy procedures alongside tumor ablation and angiographic techniques [3,7,35]. In addition to impairing the adequate delivery of essential nutrients and oxygenation to the remaining (AL3) malignant tissue, these procedures are known to set forth a landslide of tumor-derived antigens into circulation, thus acting as a therapeutic vaccine [4,36].

While endocrinologic mechanisms are largely considered to play a secondary role in the course of tumor regulation, notable hormonal changes are considered possible antecedents to SR [37]. In a case describing a presumed appendiceal neuroendocrine tumor (NET) during pregnancy, Sewpaul et al. observed rapid regression following the patient's completion of her pregnancy, suggesting that the pregnancy did not worsen the course of the disease but instead may have contributed to tumor regression [38]. Additional influences on the endocrine system by psychological events, such as trauma and stress, suggest that a patient's psychological status might also influence the course of tumor development. In a case study detailing the SR of one patient's recurrent oral squamous cell carcinoma (OSCC), Oya et al. describe how the 73-year-old patient was unable to understand the state of his recurrent cancer following cerebral infarction and dementia and postulate how this "unconsciousness" functioned as a preferable psychological condition for tumoral regression [39].

Spontaneous Regression in Cancers of Specific Pathohistology

Hepatocellular carcinoma: While testicular germ cell tumors, neuroblastomas, and renal carcinomas are conventionally the most frequent types of histologically diagnosed tumors presenting this phenomenon, several recent studies report an increasing incidence of SR within the GI pathway, particularly in primary hepatic lesions [6,40]. Correspondingly, we found that HCC was by far the most frequently observed type of cancer within the GI pathway to have undergone SR, with 199 total cases reported in the literature from 1982 to 2021. The reviewed cases proposed several mechanisms surrounding the involution of HCC, primarily citing ischemic and immunological antitumoral models of regression.

To prevent a barrage of immune responses to innocuous materials while still enabling immunity to pathogens, the complex cellular, functional, and molecular modeling of the liver allows for a dynamic, multifaceted approach to immune surveillance that incorporates the tolerogenic organ's inherently immunosuppressive microenvironment and its distinct hepatic regulatory pathways [41]. It is possible that any manipulation of this multipronged system, such as through the abatement of the tolerogenic characteristics of hepatic APCs or the enhancement of effector lymphocyte function, could potentially have the desired effect of increased anti-tumor activity and tumor regression [42].

Interestingly, several of the changes associated with the SR of the poorly prognosed tumor can also be observed following transarterial chemoembolization (TACE) treatment, thus suggesting that the SR of HCC should, to some degree, involve ischemic processes [43]. Regression of HCC has also been linked to rapid tumor infiltration, in which the notably hypervascular tumor grows more rapidly than its blood supply, leading to local or centralized ischemia, intratumoral bleeding, and hemorrhagic necrosis of the lesion [44]. These distinct immunologic and vascular attributes of the liver combine to form a tumoral environment wherein an intrahepatic malignancy is uniquely positioned to respond to immune and ischemic changes compared to tumors of other organs of the GI tract. Otherwise, abstinence from alcohol, persistent fever, withdrawal from androgens, blood transfusions, and the use of herbal medicines have also been described as

leading to the SR of primary hepatic lesions.

Primary oral and gastrointestinal lymphoma: Cases detailing the SR of primary oral and GI lymphomas were observed to span the entirety of the alimentary canal, from several primary extranodal lymphomas of the oral cavity to four cases of rectal lymphoma that regressed spontaneously. Regarding the spontaneous regression of aggressive NHLs of the digestive tract, several cases have been reported demonstrating the spontaneous involution of lymphoma following improved immunological status, particularly in HIV-infected patients receiving antiretroviral therapy [27-32].

While SR is an exceptionally rare occurrence in aggressive lymphomas, such as DLBCL and ALCL, it can occur relatively frequently in low-grade lymphomas such as follicular lymphomas (FLs) and mucosa-associated lymphoid tissue (MALT) lymphomas. Generally, well- or moderately-differentiated forms of cancer are considered low immunogenic tumors due to their limited mutational load and concomitant limited neoantigen expression. In a retrospective analysis of 209 cases of NHL from 1965 until 1978, Gattiker et al. reported the occurrence of SR in 18 out of 140 (12.9%) cases of nodular type malignant lymphoma and 2 out of 69 (2.9%) cases of diffuse type malignant lymphoma [45]. The relationship between gastric mucosa-associated lymphoid tissue (MALT) lymphoma and *H. pylori* is very well established, and low-grade gastric MALT lymphomas are known to regress following the bacteria's eradication [46]. This reversible reactivity of low-grade MALT lymphomas to *H. pylori* infection is a clearly documented phenomenon; hence, cases detailing the regression of low-grade MALT lymphomas involving *H. pylori* eradication through the use of eradication therapy were excluded from the scope of this careful review.

Pancreatic ductal adenocarcinoma: With only a few cases reported in the literature, pancreatic tumors are seldom known to undergo SR, leaving clinicians skeptical of this lethal tumor's ability to truly demonstrate involution when left untreated. Despite numerous molecular and immunological approaches, pancreatic cancer is typically poorly responsive to existing chemotherapeutic and immunological antineoplastic agents. This lack of response to immunotherapies is largely due to cancer's low mutational burden and tendency to favor an immunosuppressive microenvironment characterized by self-isolating dense desmoplastic tissue and an exceptionally low number of infiltrating T cells [47,48]. In a recently published article investigating the possibility of misdiagnosis leading to a presumptive finding of SR in pancreatic cancer, Herreros-Villaneuva et al. emphasized how different types of pancreatic carcinomas must be cautiously distinguished from otherwise benign tumors, insulinomas, and immunoglobulin G4 (IgG4)-associated autoimmune pancreatitis during the process of recording SR [49]. Regardless, four additional cases of pancreatic ductal adenocarcinoma (PDAC) have since been published, citing various multifactorial models of SR, including acute pancreatitis and bacterial or fungal infection in the vicinity of the pancreatic tumor, leading to improved immunogenic tumor presentation [48,50-52].

Colorectal cancer: Like pancreatic cancer, colorectal cancer has long been considered poorly immunogenic, largely based on indirect data from epidemiological studies on the lack of SR in colorectal cancer [53,54]. This lack of immunogenicity in this cancer can be attributed to the failure of tumor-infiltrating lymphocytes to demonstrate substantial lytic activity against cancer cells, as demonstrated in in vitro models [55,56]. While colorectal cancer constitutes more than 15% of all malignancies, it represents less than 2% of all tumors to demonstrate SR [57]. Still, several other rare forms of GI cancer, including Merkel cell-like small cell carcinoma of the parotid gland and multiple gastroenteropancreatic neuroendocrine tumors (GEP-NETs) of the stomach, bile duct, and pancreas, were observed to spontaneously regress.

Strengths and Limitations

While prior retrospective analyses have investigated the incidence of SR for specific cancers and its occurrence within the individual organs of digestion, an observational study of this scope, broadly examining all prior cases of SR throughout the entire GI pathway, has never been published to date. This first-of-its-kind study systematically and thoroughly extracts and organizes information from an array of 390 individual cases of SR within the GI pathway. Although the majority of reports were restricted to the English literature, cases in other languages, including Spanish, Chinese, German, and Japanese, are included in this broad review in order to better demonstrate the global incidence of the otherwise rare phenomenon. Putative mechanisms for SR, including immunological, ischemic, and idiopathic modalities, are also explored and discussed in a detailed manner with the hopes of aiding in an understanding of SR as a dynamic interplay of complex and interconnected antitumoral responses.

In general, SR remains a poorly understood and somewhat vaguely defined phenomenon. Our review has multiple limitations. Recognizing true SR as a host response to specific tumors may continue to be obscured by bias in how the regression is reported. In addition to the possible underreporting of cases of SR by certain physicians, there is also a significant amount of variability in how SR is defined. Distinguishing SR from abscopal effects and tumor regression instigated by eradication therapy remains highly subjective and may result in the misreporting of true spontaneous antitumoral host responses to specific cancers. Overall, the literature is quite heterogeneous, and not every case study reported the duration of follow-up or the duration of remission in a similar manner as would be expected in a retrospective review of this kind.

Conclusions

SR is an extremely rare occurrence. Nonetheless, certain recurrent patterns in cases of SR, as demonstrated in this review, deserve ample consideration. To better study SR in the future, there must be an emphasis on standardizing how SR is reported. A well-defined registry would also be helpful. Ultimately, this broadly encompassing yet focused assessment is meant to bring attention to the phenomenon of SR and perhaps aid in the investigative efforts in the burgeoning field of immunotherapies.

Appendices

Appendix A

| Cohort P | Cohort 1 | Cohort 2a | Cohort 2b |
|------------------------|-----------------------------------|-----------------------------------|-------------------------------|
| Abscopal effect | Alveolar soft-part sarcoma | Anus (anal) | Adenocarcinoma |
| Spontaneous necrosis | Cholangiocarcinoma | Bile duct (biliary/hepatobiliary) | Anaplastic lymphoma |
| Spontaneous regression | Epithelioid hemangioendothelioma | Cecum (Ileocecal) | Cancer |
| Spontaneous remission | Extramедullary plasmacytoma | Colon (colorectal) | Carcinoid |
| | Gastrinoma | Duodenum (duodenal) | Carcinoma |
| | Insulinoma | Esophagus (esophageal) | Diffuse large B-cell lymphoma |
| | Islet cell cancer | Gallbladder | Follicular lymphoma |
| | Malignant myoepithelioma | Gastroesophageal junction | Hodgkin lymphoma |
| | Malignant peritoneal mesothelioma | Gastrointestinal | Leiomyosarcoma |
| | Mucoepidermoid carcinoma | Ileum (ileal) | Lymphoblastic lymphoma |
| | Pseudomyxoma peritonei | Jejunum (jejunal) | Lymphoma |
| | | Liver (hepatic/hepatocellular) | Malignant melanoma |
| | | Mesentery (mesenteric) | MALT lymphoma |
| | | Omentum (omental) | Natural killer lymphoma |
| | | Oral cavity (oral) | Neuroendocrine tumor |
| | | Pancreas (pancreatic) | Plasmablastic lymphoma |
| | | Peritoneum (peritoneal) | T-cell lymphoma |
| | | Rectum (rectal) | |
| | | Salivary gland (adenoid) | |
| | Small bowel/intestine (enteric) | | |
| | | Tongue | |

TABLE 3: Keywords included in search criteria

Each word from cohort P was cross-searched with a word or phrase from cohorts 1 or 2a and 2b. Phrases from cohort 1 were searched individually after being paired with a word from cohort P. Phrases searched with words from Cohort 2a were matched with a single word from cohort 2b (if applicable), and this pairing was used across the five databases.

Appendix B

| | Ref | Age/ Sex | Location | Pathologic Histology | Site of regression | Period of Remission | Proposed mechanism |
|---|--------------------|-------------|---------------|-------------------------|--------------------|------------------------|--------------------|
| 1 | Roxburgh (1935) | 60s/F | Tongue | OSCC | Primary tumor | 7 years | Partial Resection |
| 2 | Grillet et al | 26/M | Parotid gland | ACC | Lung metastases | 7 years | Diet |

| | | | | | | | |
|----|------------------------------|------|-------------------------|-----------------------|--------------------------------------|--------------|---|
| | (1984) | | | | | | |
| 3 | Grillet et al (1984) | 53/M | Submandibular gland | ACC | Lung & nasolabial metastases | 3 years | Not reported |
| 4 | Grem et al (1986) | 54/F | Vallecula | DLBCL | Primary tumor | 4 years | Immunological; viral/bacterial infection; Biopsy |
| 5 | Poppema et al (1988) | 12/M | Oropharynx | LBL | Primary tumor & cervical lymph node | 3 years | Immunological (cytotoxic) |
| 6 | Savarrío et al (1999) | 77/M | Soft palate | ALCL | Primary tumor | 1 year* | Immunological; Biopsy |
| 7 | King et al (2001) | 52/M | Parotid gland | MM | Primary tumor & regional lymph nodes | 6 weeks | Immunological |
| 8 | Notani et al (2002) | 77/M | Tongue | ALCL (TCL) | Primary & multiple oral recurrences | multiple | Not reported |
| 9 | Koga et al (2003) | 78/F | Maxillary gingiva | DLBCL | Primary tumor | 3 years | Biopsy |
| 10 | Yamamoto et al (2003) | 80/F | Maxilla | DLBCL | Primary tumor | 1.5 years | Biopsy |
| 11 | Yokoyama et al (2003) | 46/F | Hard palate | MALT Lymphoma | Primary tumor | 2.5 years | Biopsy |
| 12 | Heibel H et al (2004) | 70/M | Mandible | DLBCL | Primary tumor | 1.5 years | Biopsy |
| 13 | Lester et al (2004) | 50/M | Palate | PBL | Primary tumor | 4 months* | Restoration of immune function (HAART) |
| 14 | Sakuma et al (2006) | 70/F | Palatine salivary gland | MALT Lymphoma | Primary tumor | 3 years | Biopsy; Localized infection |
| 15 | Armstrong et al (2007) | 35/M | Maxilla | PBL | Primary tumor (partial) | 2 weeks | Restoration of immune function (antiretroviral) |
| 16 | Kurita et al (2007) | 67/M | Tongue | OSCC | Cervical lymph node metastases | 10 months | Enhanced Apoptosis |
| 17 | Oya andikemura K (2007) | 73/M | Tongue | OSCC | Primary tumor | 3.5 years | Psychoneurological ("unconsciousness to cancer" S/P cerebral infarct & dementia); Immunological |
| 18 | Rujirojindakul et al (2007) | 26/M | Submandibular gland | LBL | Primary tumor | multiple* | Not reported |
| 19 | Daly et al (2008) | 56/M | Maxillary gingiva | TCL | Primary tumor | 4 years | Biopsy |
| 20 | Mulder et al (2009) | 78/M | Parotid gland | Merkel cell-like SmCC | Primary tumor | 5 months* | T-cell mediated response triggered by trauma; Apoptosis |
| 21 | Brachet et al (2011) | 58/F | Hard palate | DLBCL | Primary tumor | 15 days* | Biopsy |
| 22 | Corti et al (2011) | 55/F | Oral | PBL | Primary tumor | 10 months | Restoration of immune function (antiretroviral cART) |
| 23 | Tamás et al (2011) | 66/F | Vallecula/tongue | DLBCL | Primary tumor | 7 years | Not reported |
| 24 | Fitzpatrick et al (2012) | 88/F | Labial mucosa | ALCL (TCL) | Primary tumor | 2 weeks | Biopsy |
| 25 | García-Noblejas et al (2013) | 78/F | Buccal mucosa | PBL | Buccal & cervical lymph node lesions | 2 years | Restoration of immune function (removal of methotrexate) |
| 26 | Choi et al (2014) | 52/F | Buccal mucosa | OSCC | Cervical lymph node metastases | Not reported | Tumor Microenvironment modification |
| 27 | Sousa et al | 62/M | Mouth floor | OSCC | Primary tumor | 3 months | Biopsy; Immunological |

Oral cancer

| | | | | | | | | |
|------------|-----------------------------|----------------------|-------------------------|--------------------|-------------------------|--|--|---|
| | (2014) | | | | | | | |
| 28 | Cuenca-Jimenez et al (2015) | 65/M | Parotid gland | OSCC | Primary tumor | Not reported | Not reported | |
| 29 | Igawa et al (2015) | 80/M | Maxillary gingiva | PBL | Primary tumor | 8 months | Genetic (immunosenescence) | |
| 30 | Kaibuchi et al (2015) | 87/M | Gingiva | DLBCL | Primary tumor | 2.5 years | Biopsy | |
| 31 | Gonzalez-Perez et al (2016) | 75/M | Mandibular gingiva | EMP | Primary tumor | 1.5 years | Immunological (cytokines. Growth factors); Biopsy | |
| 32 | Wagner et al (2016) | 33/F | Mandibular gingiva | PBL | Primary tumor | 1.5 months | Restoration of immune function (HAART) | |
| 33 | Daroit et al (2017) | 66/F | Maxilla | PBL | Primary tumor | 1 year | Restoration of immune function (HAART) | |
| 34 | Kitamura et al (2017) | 61/M | Maxillary gingiva | PBL | Primary tumor | 2 years | Restoration of immune function (antiretroviral) | |
| 35 | Rajan & Samant et al (2017) | 49/F | Hard palate | MM | Primary tumor | Not reported | Immunological | |
| 36 | Yao et al (2017) | 80/M | Maxillary gingiva | PBL | Primary tumor | Not reported | Traumatic factors (Biopsy) | |
| 37 | Miyagawa et al (2018) | 46/M | Upper lip | ALCL | Primary tumor | 1 year | Biopsy | |
| 38 | Gamarra et al (2018) | 50/F | Maxilla | MEC | Primary tumor (partial) | 10 years | Not reported | |
| 39 | Ono et al (2019) | 69/M | Mandibular gingiva | PBL | Primary tumor | 2 years | Not reported | |
| 40 | Curioni et al (2020) | 51/F | Submandibular gland | SGAC | Primary tumor | 7 years | Metabolic derangement (hypoglycemia); Immunological | |
| 41 | Oliveira et al (2020) | 52/F | Hard palate | MM | Primary tumor | 6 years | Inflammatory; Immunological | |
| 42 | Peeters et al (2020) | 59/M | Buccal/Masticator space | FL | Primary tumor | 6 months | Biopsy | |
| 43 | Aoki et al (2021) | 84/M | Maxillary gingiva | DLBCL | Primary tumor | 2 years | Biopsy | |
| 44 | Lau et al (2021) | 66/F | Oropharyngeal tonsil | OSCC | Primary tumor | 7 months | Biopsy (Anti-tumoral T-cell response); Hyperthermal state (COVID-19 vaccine) | |
| 45 | Ueno et al (2021) | 83/F | Mandibular gingiva | MM | Primary tumor (partial) | 26 days | Not reported | |
| 46 | Sousa et al (2022) | 61/F | Parotid gland | MME | Primary tumor | 9 months | Inflammatory response (COVID-19 vaccine) | |
| Esophageal | 47 | Rees et al (1983) | 49/M | Lower esophagus | EAC | Lung metastases | 1 year | Abscopal effect |
| | 48 | Oshwada et al (1990) | 78/M | Thoracic esophagus | ESCC | Primary tumor (partial) & pulmonary metastases | 7 months | Change in T-cell subsets; surgical trauma |
| | 49 | Vergeau et al (1991) | 36/M | Mid esophagus | ESCC | Primary tumor (partial) | 1 year | Leukocyte infiltration; Inflammation |
| | 50 | Hahm et al (1993) | 30/M | Thoracic esophagus | PEL | Primary tumor | Not reported | H2-blocker (Cimetidine) |
| | 51 | Saruki et al (1994) | 82/F | Thoracic esophagus | ESCC | Primary tumor | 2 years | Infection (Pneumonia) |
| | | Takemura et al | | Thoracic | | | | |

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|----------------|-------------------------|-----------------------|-------------------------|------------------------------|---------------------------------------|--|---|-------------------------------------|
| cancer | 52 | (1999) | 63/F | esophagus | ELMS | Pleural & splenic metastases | 10 months | Removal of Primary |
| | 53 | Chang (2000) | 57/M | Lower esophagus | ESCC | Primary tumor | 9 years | Infection (Pneumonia); Inflammation |
| | 54 | Kubota et al (2003) | 73/M | Thoracic esophagus | SCEC | Primary tumor | 1 month | Infection (Hepatitis C) |
| | 55 | Hornby et al (2015) | 57/M | GEJ | MM | Primary tumor | 2 months | Immunological; Occult Primary |
| | 56 | Mitchell et al (2021) | 58/F | GEJ | GEJAC | Local & supraclavicular lymph metastases | 6 months | Not Reported |
| | 57 | Kahn et al (2021) | 66/M | Lower esophagus | ESCC | Primary tumor | 3 months | Immunological (T-cell response) |
| | 58 | Takeuchi et al (1971) | 39/M | Corpus-antrum | PGL (RCS) | Primary tumor (partial) | 2 months | Not reported |
| 59 | Nakano et al (1972) | 55/F | Corpus | PGL (RCS) | Primary tumor (partial) | 3 weeks | "malignant cycle" of early gastric cancer | |
| 60 | Rosenberg et al (1972) | 51/M | Lesser curvature | GAC | Liver metastases | 12 years | Fever | |
| 61 | Ohashi et al (1973) | 42/M | Corpus-antrum | PGL (RCS) | Primary tumor (partial) | 1 month | "malignant cycle" of early gastric cancer | |
| 62 | Tietjen et al (1974) | 60/F | Gastric antrum duodenum | PGL (RCS) | Primary tumor | 5 years | "malignant cycle" of early gastric cancer | |
| 63 | Yamazaki et al (1974) | 39/M | Pyloric antrum | PGL (RCS) | Primary tumor (partial) | 20 days | Not reported | |
| 64 | Kimura et al (1987) | 85/F | Residual stomach | GAC | Primary tumor | Not reported | Not reported | |
| 65 | Strauchen et al (1987) | 73/M | Pyloric antrum | PGL (DLBCL) | Primary tumor | 3 weeks | H2-receptor antagonist | |
| 66 | Strauchen et al (1987) | 84/M | Pyloric antrum | PGL (DLBCL) | Primary tumor | 1 month | H2-receptor antagonist | |
| 67 | Harvey et al (1988) | 78/F | Gastric body/fundus | GEP-NET (ECL-cell Carcinoid) | Multifocal gastric lesions (majority) | 10 years | Not reported | |
| 68 | Harvey et al (1988) | 55/M | Stomach | GEP-NET (ECL-cell Carcinoid) | Multifocal gastric lesions | 5 years | Not reported | |
| 69 | Sawant et al (1989) | 40/F | Unspecified stomach | GEP-NET (Carcinoid) | Primary tumor | 1 year | Biopsy | |
| 70 | Shigematsu et al (1989) | 40/F | Pyloric antrum | PGL (DLBCL) | Primary tumor (partial) | 2 months | Not reported | |
| 71 | Shigematsu et al (1989) | 73/M | Pyloric antrum | PGL (DLBCL) | Primary tumor | 2 months | Not reported | |
| 72 | Rebollo et al (1990) | 77/M | Gastric body/fundus | GAC | Primary tumor | 8 months | Infection (abdominal wall abscess) | |
| 73 | Yoshimine et al (1991) | 69/F | Gastric angulus | PGL | Primary tumor | 2.5 months | Dislodged (ulceration) | |
| 74 | Hayakawa et al (1992) | 62/F | Lesser curvature | PGL (DLBCL) | Primary tumor | 1 year | Necrosis & Detachment | |
| 75 | Takehara et al (1992) | 44/M | Gastric angulus/Antrum | PGL | Primary tumor (partial) | 1 month | H2-blocker | |
| 76 | Matsusaki et al (1996) | 64/F | Pyloric antrum | PGL (DLBCL) | Primary tumor | 1 month | Not reported | |
| Stomach cancer | 77 | Ogawa et al | 63/F | Gastric corpus | PGL (DLBCL) | Primary tumor | 13 months | H Pylori eradication |

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|-----|------------------------------|------|------------------------|----------------------|---|------------|---|
| | (1998) | | | | | | |
| 78 | Bariol et al (2001) | 24/M | Gastric antrum | PGL (TCL) | Primary tumor | 2 years | H Pylori eradication |
| 79 | Salam et al (2001) | 73/F | Greater curvature | PGL (DLBCL) | Primary tumor | 2.5 years | H Pylori eradication |
| 80 | Pentimone et al (2002) | 84/M | Residual stomach | PGL (MALT) | Recurrences | 15/5 years | Not reported |
| 81 | Chung et al (2003) | 48/M | Lesser curvature | GAC | Primary tumor | 4 years | Ischemia (angiography) |
| 82 | Watanabe et al (2003) | 22/M | Gastric body & Antrum | PGL (TCL) | Primary tumor | 1 month | Infection (Severe EBV viremia in CAEBV) |
| 83 | Watari et al (2005) | 60/F | Gastric angulus/Antrum | PGL (DLBCL) | Primary tumor | 1 year | H2-blocker; H. pylori eradication |
| 84 | Watari et al (2005) | 61/M | Gastric angulus | PGL (DLBCL) | Primary tumor | 6 months | H2-blocker; H. pylori eradication |
| 85 | Ohno et al (2006) | 14/M | Lower gastric corpus | PGL (MALT) | Primary tumor | 10 years | Immunological (Cessation of exposure to H pylori antigen) |
| 86 | Lee et al (2010) | 84/M | Cardia & Lower body | GAC | Primary tumor | 1 year | Not reported |
| 87 | Ip et al (2011) | 77/M | Gastric cardia | GEP-NET (LCNEC) | Primary tumor | 3 months | Infection (cytomegalovirus); Cross-autoimmune reaction against neuronal cells |
| 88 | Yang et al (2012) | 77/M | Gastric body | GAC | Primary tumor & recurrences | Multiple | Not reported |
| 89 | Rojas-Hernandez et al (2014) | 57/M | Greater curvature | PGL (DLBCL) | Primary tumor | 2 years | Immunological (B-cell stimulation by HCV) |
| 90 | Shibata et al (2016) | 75/M | Gastric antrum | GEP-NET | Peripancreatic lymph metastases | 6 months* | EUS-FNA; Bacterial infection |
| 91 | Sugiyama et al (2018) | 62/F | Gastric body | PGL (DLBCL) | Primary tumor | 10 years | Not reported |
| 92 | Bonilla et al (2019) | 78/F | Gastric antrum | GAC | Retroperitoneal adenopathies | 3 months | Abscopal effect |
| 93 | Hatsuse et al (2019) | 82/F | Unspecified stomach | PGL (DLBCL) | Primary tumor | 2 years | Not reported |
| 94 | Okamoto et al (2021) | 37/M | Gastric antrum | GEP-NET (Gastrinoma) | Primary tumor | 3 years | Biopsy; Resection of omental metastases |
| 95 | Zafar et al (2021) | 74/M | Lesser curvature | GAC | Primary tumor | 6 years | Not reported |
| 96 | Schwartz et al (1991) | 39/M | Peritoneum, omentum | MPM | Local regression | 8 years | Fever; Rheumatoid factor |
| 97 | BaniHani et al (2009) | 38/M | Mesentery | ASPS | Abdominal mass, heart & lung metastases | 5 months | Immunological; Herbal medicine |
| 98 | Jagodic et al (2018) | 66/F | Retroperitoneal space | RLMS | Liver metastases | 2 years | Not reported (possible delayed response to ChT) |
| 99 | Gottfried et al (1982) | 65/M | Diffuse hepatic | HCC | Primary tumor | 4 years | Abstinence from alcohol; A-P shunt; Portal vein thrombosis |
| 100 | Lam et al (1982) | 50/M | Unspecified liver | HCC | Primary tumor & lung metastases | 13 years | Chinese herbal medicine; Bronchopneumonia |
| 101 | McCaughan et al (1985) | 28/M | Right lobe | HCC | Primary tumor | 6.5 years | Androgen withdrawal |
| | McCaughan et | | | | | | |

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|-----|---------------------------|------|--------------------------|----------------|---------------------------------|--------------|--|
| 102 | al (1985) | 40/M | Right lobe | HCC | Primary tumor | 9 years | Androgen withdrawal |
| 103 | Sato et al (1985) | 78/M | Right lobe | HCC | Primary tumor & bone metastases | 5 years | Ischemia (GI Bleeding) |
| 104 | Takayasu et al (1986) | 38/M | Unspecified liver | HCC | Primary tumor (partial) | 2 months | Subintimal injury (angiography) |
| 105 | Takayasu et al (1986) | 58/F | Unspecified liver | HCC | Primary tumor | 2.5 years | Subintimal injury (angiography) |
| 106 | Andreola et al (1987) | 75/M | S6/7 | HCC | Primary tumor | 18 days | Venous thrombosis |
| 107 | Saez-Royuela (1989) | 66/M | Unspecified liver | HCC | Primary tumor | 2.5 years | Not reported |
| 108 | Suzuki et al (1989) | 65/M | Posterior right lobe | HCC | Primary tumor | 6 years | Rapid growth |
| 109 | Ayres et al (1990) | 63/F | Diffuse hepatic | HCC | Primary tumor (partial) | 1 year | Not reported |
| 110 | Gaffey & Joyce (1990) | 63/M | Right lobe | HCC | Primary tumor (partial) | 1.5 years | Ischemia (GI Bleeding); Macrobiotic diet |
| 111 | Tocci, G et al (1990) | 79/M | Hepatic hilum | HCC | Primary tumor | 3 months | Ischemia (GI Bleeding) |
| 112 | Mochizuki et al (1991) | 61/M | Unspecified liver | HCC | Primary tumor (partial) | 1.5 years | Abscopal Effect |
| 113 | Yamamoto et al (1991) | 58/M | Unspecified liver | HCC | Primary tumor | Not reported | Ischemia (hemorrhage) |
| 114 | Yamamoto et al (1991) | 68/F | Unspecified liver | HCC | Primary tumor | Not reported | Not reported |
| 115 | Chien et al (1992) | 65/M | Right lobe | HCC | Primary tumor | 2.5 years | Herbal Remedies |
| 116 | Imaoka et al (1994) | 65/M | Left lateral lobe | HCC | Primary tumor | Not reported | Arterial thrombosis |
| 117 | McDermott & Khetry (1994) | 23/F | Left lobe | Clear cell HCC | Primary tumor (partial) | 5 years | Not reported |
| 118 | Grossmann et al (1995) | 52/M | Diffuse hepatic | HCC | Primary tumor (partial) | 1 year | Abstinence from alcohol |
| 119 | Herrera et al (1996) | 76/M | Unspecified liver | HCC | Primary tumor | 1 year | Not reported |
| 120 | Ozeki et al (1996) | 69/F | S3 | HCC | Primary tumor | 1 year | Herbal Remedies |
| 121 | Markovic et al (1996) | 62/M | S5/6 | HCC | Primary tumor | 8 years | Fever; Ischemia (hemorrhage S/P biopsy); Biological effects by cytokines |
| 122 | Yoshimitsu et al (1996) | 34/M | Intrahepatic (left lobe) | CCA | Primary tumor | 4 months* | Fibrous component |
| 123 | Iwasaki et al (1997) | 72/F | Posterior/lateral | HCC | Primary tumor (partial) | 1.5 years | Tumor's rapid growth |
| 124 | Van Halteren et al (1997) | 72/F | Right lobe | HCC | Primary tumor | 2 years | Ischemia & infarction due to Cirrhosis |
| 125 | Kaczynski et al (1998) | 73/M | Central part/Hilum | HCC | Primary tumor | 3 years | Not reported |
| 126 | Ohba et al (1998) | 76/M | S5 | HCC | Primary tumor (partial) | 2 years | Abscopal Effect |
| 127 | Magalotti et al | 66/M | Unspecified liver | HCC | Primary tumor | 4 years* | Not reported |

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|-----|------------------------|------|--------------------|------|--|--------------|---|
| | (1998) | | | | | | |
| 128 | Megalotti et al (1998) | 75/F | Unspecified liver | HCC | Primary tumor (partial) | 3 years* | Not reported |
| 129 | Sanz et al (1998) | 66/M | Right lobe | HCC | Primary tumor | 1 year | Immunological |
| 130 | Stoelben et al (1998) | 56/M | S6 | HCC | Primary tumor | 2 years | Immunological (Resection of tumor); Infection (abscess) |
| 131 | Stoelben et al (1998) | 74/M | S6 | HCC | Primary tumor | 3.5 years | Immunological (resection of tumor); Infection (abscess) |
| 132 | Takeuchi et al (1998) | 53/M | S8 | HCC | Primary tumor | Not reported | Ischemia (thrombus) |
| 133 | Itoh et al (1999) | 58/M | S5 | HCC | Primary tumor | 13 days | Tumor Hypoxia (Thick capsule) |
| 134 | Toyoda et al (1999) | 82/M | Right lobe | HCC | Primary regression (Primary & Lung metastases) | 1.5 years | Transition from necrosis to fibrosis |
| 135 | Izuishi et al (2000) | 50/M | S2/3 | HCC | Primary tumor | 5 years | Ischemia; Immunological; Angiography |
| 136 | Jang et al (2000) | 54/F | Right lobe | HCC | Primary tumor | 4 years | Not reported |
| 137 | Lee et al (2000) | 44/M | S4/8 | HCC | Partial regression (Primary) | 1 year* | Infection; Abstinence from alcohol |
| 138 | Lee et al (2000) | 63/M | Right lobe | HCC | Partial regression (Primary) | 3 years | Infection; Arterial thrombosis/intimal injury (angiography) |
| 139 | Takeda et al (2000) | 68/M | S4/5/6/7/8 | HCC | Primary tumor | 1 year | Herbal Remedies |
| 140 | Terasaki et al (2000) | 72/F | S5 | HCC | Primary tumor, peritoneal & splenic metastases | 2 years | Apoptosis |
| 141 | Uenishi et al (2000) | 65/M | Right lobe | HCC | Primary tumor (partial) | 1 year | Abstinence from alcohol; A-P shunt; Portal vein thrombosis |
| 142 | Ikeda et al (2001) | 75/M | S7 | HCC | Primary tumor (partial) | 6 years | Not reported |
| 143 | Jung et al (2001) | 58/M | Right lobe | HCC | Primary tumor (partial) & lung metastases | 1.5 years* | Herbal Remedies; cessation of smoking; |
| 144 | Kawai et al (2001) | 58/M | S6 | HCC | Primary tumor | 1 month | Ischemia; Immunological; Angiography |
| 145 | Matsuo et al (2001) | 72/M | S5 | HCC | Primary tumor (partial) | 1 year | Immunological; Hypoxia; Inflammatory cell infiltration |
| 146 | Nakai et al (2001) | 76/M | Residual liver | HCC | Primary tumor | 2 years | Immunological (NK cell response) |
| 147 | Sakurai et al (2001) | 65/M | Gallbladder fundus | GBAC | Primary tumor | Not reported | Ischemia; Inflammation; Pancreaticobiliary maljunction |
| 148 | Serrano et al (2001) | 71/M | Left lobe | HCC | Primary tumor | 3 years | Growth factors; Ischemia (hepatic artery) |
| 149 | Abiru et al (2002) | 70/M | Unspecified liver | HCC | Primary tumor, lung & bone metastases | 2 years | Immunological (IL-18) |
| 150 | Abiru et al (2002) | 65/F | Unspecified liver | HCC | Primary tumor, lung & lymph metastases | 4 months | Immunological (IL-18) |
| 151 | Abiru et al (2002) | 65/M | Unspecified liver | HCC | Primary tumor, lung & bone metastases | 1 year | Immunological (IL-18) |
| 152 | Lee et al (2002) | 70/M | S2/3 | HCC | Primary tumor | 24 days | Occlusion of feeding artery |
| 153 | Misawa et al (2002) | 62/M | Anterior segment | HCC | Primary tumor | 1 year | Biological effects by A-P shunt |

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|-----|-------------------------|------|-------------------|--------------------|---|--------------|--|
| 154 | Morimoto et al (2002) | 73/M | S2/3 | HCC | Primary tumor | 1 year | Arterial thrombosis |
| 155 | Zimmermann et al (2002) | 56/M | S6 | Medullary-like HCC | Primary tumor | 2 years | Immunological (Cytotoxic pathway); Apoptosis |
| 156 | liai et al (2003) | 69/M | S6/7 | HCC | Primary tumor | 4 years | Portal vein thrombosis; cessation of smoking |
| 157 | Jozuka et al (2003) | 52/M | Hepatic surface | HCC | Primary tumor | 2.5 years | Psychoneurological; Antidepressants; Immunological |
| 158 | Li et al (2003) | 53/M | S6 | HCC | Primary tumor | Not reported | Biological effects by cytokines |
| 159 | Ohta et al (2003) | 74/M | S2/3 | HCC | Primary tumor | 1 year | Immunological; hypoxia (arterial sclerosis) |
| 160 | Blondon et al (2004) | 64/M | Diffuse hepatic | HCC | Local regression | 9 months | Infection (peritonitis); Ischemia (Intraperitoneal bleed) |
| 161 | Blondon et al (2004) | 70/F | Diffuse hepatic | HCC | Local regression | 3 years | Infection (peritonitis); Ischemia (Intraperitoneal bleed); tamoxifen |
| 162 | Cheng et al (2004) | 74/M | Medial left lobe | HCC | Primary tumor | 6 years | Herbal remedies |
| 163 | Erturk et al (2004) | 69/M | Left lobe | HCC | Primary tumor | 3 years | Blood transfusion |
| 164 | Feo et al (2004) | 71/F | S3/5 | HCC | Primary tumor (partial) | 1.5 years | Ischemia |
| 165 | Kato et al (2004) | 72/M | Right lobe | HCC | Primary tumor (partial) | 2 years | Not reported |
| 166 | Kato et al (2004) | 77/M | Right lobe | HCC | Primary tumor & lung metastases | 1 year | Abstinence from smoking |
| 167 | Lin et al (2004) | 42/M | S8 | HCC | Primary tumor (partial) | 2 years | Herbal remedies |
| 168 | Nakajima et al (2004) | 80/M | S4/6 | HCC | Partial regression (Primary) | 6 months | Ischemia; Intratumoral bleeding/hemorrhagic necrosis |
| 169 | Jeon et al (2005) | 72/M | Right lobe | Clear Cell HCC | Primary tumor (partial) & chest wall metastases | 9 months | Metabolic derangement (hypoglycemia & HLD) |
| 170 | Moon et al (2005) | 72/M | S6 | HCC | Primary tumor | 2 years | Alcohol cessation |
| 171 | Nam et al (2005) | 65/M | Right lobe | HCC | Liver & bone metastases (partial) | 1 year | Abscopal Effect |
| 172 | Nouso et al (2005) | 85/M | S5/6/7/8 | HCC | Primary tumor (partial) | 2 years | Ischemia; Vitamin K administration |
| 173 | Ohtani et al (2005) | 69/M | S4 | HCC | Primary tumor (partial) | 3 years | Tumor Hypoxia (Thick capsule) |
| 174 | Randolph et al (2005) | 56/M | Left lateral lobe | HCC | Primary tumor | 1.5 years | Alcohol cessation, ischemia (obstruction of portal vein thrombosis); Infection (Pneumonia) |
| 175 | Rizell et al (2005) | 58/M | Central liver | HCC | Primary tumor (partial) | 1.5 years | Sirolimus (Immunosuppressive) |
| 176 | Yano et al (2005) | 71/F | S8 | HCC | Primary tumor (partial) | 2 years | Hypoxia (artery rupture) |
| 177 | Otrock et al (2006) | 75/F | Diffuse hepatic | HEHE | Primary tumor | 3.5 years | Not reported |
| 178 | Kojima et al (2006) | 79/M | S8 | HCC | Lung metastases | 6 months | Steroids, Hormones, or Herbal Remedies |
| 179 | Kondo et al (2006) | 67/M | S4 | HCC | Primary tumor (partial) | 2 months | Immunological |
| 180 | Kondo et al (2006) | 67/M | S5/3 | HCC | Primary tumor & lung metastases | 4 years | CAM's; Immunological |
| 181 | Kondo et al | 70/M | Right lobe | HCC | Primary tumor (partial) | 5 years | Bleeding (esophageal varices); Immunological |

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|-----|-------------------------|------|-------------------|-----------------|---------------------------------|--------------|---|
| | (2006) | | | | | | |
| 182 | Kondo et al (2006) | 75/M | S7 | HCC | Lung metastases (partial) | 2 years | Immunological |
| 183 | Shibuya et al (2006) | 71/M | S5 | HCC | Primary tumor | 2 months | Ischemia; Immunological; Angiography |
| 184 | Heianna et al (2007) | 70/F | Unspecified liver | HCC | Lung metastases | 5 years | Immunological (Host cytokines); Systemic inflammatory (TACE of primary) |
| 185 | Matsunaga et al (2007) | 71/F | Left lateral lobe | Sarcomatoid HCC | Peritoneal metastases | 4 months* | Ischemia (rapid growth) |
| 186 | Meza-Junco et al (2007) | 56/F | S5 | HCC | Primary tumor | 2 years | Hypoxia (Thick capsule) |
| 187 | Peddu et al (2007) | 57/M | S4 | HCC | Primary tumor | 2 months | Auto-infarction |
| 188 | Vardhana et al (2007) | ?/M | S2/3/4 | HCC | Primary tumor | 8 months | Immunological |
| 189 | Arakawa et al (2008) | 78/F | S2/3 | HCC | Primary tumor | 2.5 years | Immunological; Portal vein thrombosis |
| 190 | Hori et al (2008) | 71/M | Gallbladder | GBAC | Primary tumor | Not reported | Increased intraluminal pressure (PBM); Pancreatic enzymes |
| 191 | Sibartie et al (2008) | 76/M | S5 | HCC | Primary tumor (partial) | 2 years | Ischemia (disturbance of blood flow) |
| 192 | Del Poggio et al (2009) | 77/F | S6 | HCC | Primary tumor (partial) | 1.5 years | Immunological (tumor antigens) |
| 193 | Hsu et al (2009) | 66/M | S7/8 | HCC | Primary tumor (partial) | 1.5 years | Hypoxia; Immunological; Silymarin; Portal vein thrombosis |
| 194 | Kanzaki et al (2009) | 52/M | S8 | HCC | Primary tumor | 8 months | Tumor Hypoxia (thick capsule) |
| 195 | Nishijima et al (2009) | 86/F | S7 | HCC | Primary tumor (partial) | 4 months | Tumor infarction |
| 196 | Oquiñena et al (2009) | 54/M | S6 | HCC | Primary tumor | 2 years | Vascular ischemia; Immunological |
| 197 | Oquiñena et al (2009) | 61/M | S1 | HCC | Primary tumor | 1.5 years | Vascular ischemia; Immunological |
| 198 | Oquiñena et al (2009) | 60/M | Right lobe | HCC | Primary tumor | 3 years | Vascular ischemia; Immunological |
| 199 | Park et al (2009) | 57/M | S5/6/7/8 | HCC | Primary tumor (partial) | 5 years | Infiltrating lymphocytes |
| 200 | Harada et al (2010) | 70/M | S7 | HCC | Primary tumor | 2 years | Ischemia; Herbal remedies |
| 201 | Hong et al (2010) | 67/M | Resection margin | HCC | Primary tumor & lung metastases | 1 year | TACE of Primary |
| 202 | Kai et al (2010) | 58/F | S6/7 | HCC | Primary tumor | 1 month | intimal injury (angiography) |
| 203 | Kai et al (2010) | 49/M | S6 | HCC | Primary tumor | 3 weeks | intimal injury (angiography) |
| 204 | Satou et al (2010) | 83/M | Right lobe | HCC | Primary tumor | Not reported | NSAIDS (Ketoprofen) |
| 205 | Storey et al (2010) | 52/M | S5/6 | HCC | Primary tumor | 3 years | Cessation of alcohol |
| 206 | Alqutub et al (2011) | 65/M | Right lobe | HCC | Primary tumor | 2 years | Ischemia (Rapid growth; Intratumoral hemorrhage) |
| 207 | Arora et al | 54/M | Right lobe | HCC | Primary tumor | 2 years | Immunological; Necrosis |

Hepatobiliary
Cancer

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| | (2011) | | | | | | |
| 208 | Fukushima et al (2011) | 69/M | Right lobe | HCC | Lung metastases | 7 years | Immunological (TACE of Primary) |
| 209 | Maejima et al (2011) | 68/M | S3/5 | HCC | Primary tumor | 3 months | Ischemia; Immunological |
| 210 | Okano et al (2011) | 68/M | Right lobe | HCC | Tumor recurrence | 2 years | PVT; Ischemia (rapid growth) |
| 211 | Okuma et al (2011) | 63/M | Right lobe | HCC | Lung metastases | 3 years | Abscopal Effect |
| 212 | Bastawrous et al (2012) | 63/M | Right lobe | HCC | Primary tumor (partial) | Not reported | Ischemia |
| 213 | Harimoto et al (2012) | 73/M | S6/7 | HCC | Primary tumor & lung metastases | 1 year | Ischemia (hypotension during dialysis) |
| 214 | Komatsu et al (2012) | 65/M | Right lobe | HCC | Primary tumor & recurrences | 6 months (x3) | Not reported |
| 215 | Nakayama et al (2012) | 92/F | Right lobe | HCC | Primary tumor | Not reported | Ischemia (rapid growth); Immunological |
| 216 | Takeura et al (2012) | 69/F | Unspecified liver | HCC | Primary tumor & bone metastases | 10 months | Inflammatory (trauma) |
| 217 | Takeura et al (2012) | 84/F | Unspecified liver | HCC | Primary tumor & peritoneal carcinomatosis | 1.5 years | Inflammatory (trauma) |
| 218 | Yamamoto et al (2012) | 60/M | S4-8 | HCC | Primary tumor | 3 weeks | Immunological; Diabetes Control |
| 219 | Yokoyama et al (2012) | 80/M | S4 | HCC | Primary tumor | 1 month | Immune; Ischemia (thrombus) |
| 220 | Katayama et al (2013) | 74/M | S5/6 | HCC | Primary tumor | 1 month | Tumor Hypoxia (thick capsule) |
| 221 | Okano et al (2013) | 77/M | S4/6/7/8 | HCC | Primary tumor (partial) | 1 year | Ischemia (Disruption of feeding artery); Abstinence from alcohol |
| 222 | Sasaki et al (2013) | 79/M | S2 | HCC | Primary tumor | 2 months | Not reported |
| 223 | Tomishige et al (2013) | 76/F | S6 | HCC | Primary tumor | Not reported | Not reported |
| 224 | Ueda et al (2013) | 63/F | S7 | HCC | Primary tumor | Not reported | Ischemia (Hypotension during dialysis) |
| 225 | Bhardwaj et al (2014) | 74/M | Left lobe | HCC | Primary tumor | Not reported | Not reported |
| 226 | Chiesara et al (2014) | 65/M | S6 | HCC | Primary tumor (partial) | 2 years | Herbal remedies; Ischemia; Inflammatory Processes |
| 227 | Inoue et al (2014) | 57/M | S5 | HCC | Primary tumor | Not reported | Drugs (Inhibition of angiogenesis by rifampicin & minocycline) |
| 228 | Lim et al (2014) | 64/M | Right lobe | HCC | Primary tumor | 6 months | Immunological; Herbal medicine |
| 229 | Miyake et al (2014) | 79/M | S6/8 | HCC | Primary tumor | 1 month | Tumor Hypoxia (thick capsule) |
| 230 | Saito et al (2014) | 74/M | S8 | HCC | Primary tumor (partial) | 2 months | Immunological; Cessation of drinking & smoking |
| 231 | Tomino et al (2014) | 77/M | S1 | HCC | Primary tumor | 1 month | Hypoxia; Fever; Biopsy |
| 232 | Tsai et al (2014) | 74/M | Left lobe | HCC | Primary tumor | 4 years | Not reported |

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|-----|---------------------------|------|-----------------|---------|---|--------------|---|
| 233 | Zhao et al (2014) | 22/F | Diffuse hepatic | HEHE | Primary tumor (partial) | 3 years | Not reported |
| 234 | Parks et al (2015) | 69/M | S8 | HCC | Recurrent Hepatic Lesions | 6 months* | Immunological (Vitiligo autoimmunity) |
| 235 | Parks et al (2015) | 63/M | S7 | cHCC-CC | Retropertoneal lymph metastases | 2 months | Immunological |
| 236 | Parks et al (2015) | 67/M | Left lobe | HCC | Primary tumor | 5 months | Immunological |
| 237 | Kim et al (2015) | 57/M | S6 | HCC | Primary tumor | Not reported | Immunological; Ischemia |
| 238 | Kohda et al (2015) | 80/M | S1 | HCC | Primary tumor | Not reported | Ischemia (rapid growth) |
| 239 | Kuwano et al (2015) | 84/M | S4 | HCC | Primary tumor | Not reported | Ischemia |
| 240 | Matsuoka et al (2015) | 67/M | S6 | HCC | Primary tumor | 3 years | Hypoxia; Hepatic arterial & portal vein thromboses |
| 241 | Okano et al (2015) | 73/M | S8 | HCC | Primary tumor | 6 months | Ischemia; Angiography |
| 242 | Sugamoto et al (2015) | 77/F | S3 | HCC | Primary tumor | 9 months | Immunological (weight loss) |
| 243 | Takeda et al (2015) | 68/M | S4 | HCC | Primary tumor (partial) | 1 year | Hypoxia; Vessel thrombosis |
| 244 | Tazawa et al (2015) | 77/M | S7 | HCC | Primary tumor | 3 months | Ischemia (postoperative hypotension) |
| 245 | Verla-Tebit et al (2015) | 53/M | Right lobe | HCC | Primary tumor & lung metastases (partial) | 1.5 years | Anti-hepatic viral medication for Hepatitis C (sorafenib & ribavirin) |
| 246 | Wang et al (2015) | 50/M | S7/8 | HCC | Primary tumor | Not reported | Immunological |
| 247 | Yang et al (2015) | 59/M | S6 | HCC | Primary tumor | 6 months | Seroconversion of HBV |
| 248 | Yoo et al (2015) | 62/M | S5 | HCC | Primary tumor (partial) | 2 years | Immunological; Hypoxia/Ischemia |
| 249 | Gunasekaran et al (2016) | 49/M | Left lobe | HCC | Pulmonary metastases | 5 months | Consumption of Guaynabo fruit extract |
| 250 | Heron et al (2016) | 61/F | S7 | HCC | Primary tumor | 1 year | Withdrawal of azathioprine in Crohn's Disease; Biopsy |
| 251 | Jianxin et al (2016) | 64/M | S6 | HCC | Tumor recurrence and omental metastases | 2.5 years | Immunological; Herbal medicine |
| 252 | Kumar et al (2016) | 40/M | S2/3/5 | HCC | Primary tumor | 7 years | Cessation of immunosuppressive therapy |
| 253 | Kumar et al (2016) | 74/M | Right | HCC | Primary tumor (partial) | 8 months | Cessation of immunosuppressive therapy |
| 254 | Luo et al (2016) | 61/F | S4 | HCC | Primary tumor | 2.5 years | Cirrhosis related hypoxia |
| 255 | Mahmood et al (2016) | 59/M | S4/8 | HCC | Primary tumor | 4 months | Anti-hepatic viral medication for Hepatitis C (sorafenib & ribavirin) |
| 256 | Ooka et al (2016) | 63/M | S7/8 | HCC | Primary tumor | 6 months | Ischemia (PVT) |
| 257 | Pectasides et al (2016) | 53/M | S4 | HCC | Primary (partial) & lung metastases (partial) | 2 months | Portal vein thrombosis; Immunological reaction |
| 258 | Sawatsubashi et al (2016) | 59/M | S5-8 | HCC | Primary tumor | 1 month | Tumor Hypoxia (thick capsule) |

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|-----|-------------------------|------|------------------|-------|-------------------------------------|--------------|---|
| 259 | Sugiura et al (2016) | 90/F | S6 | HCC | Primary tumor | Not reported | Not reported |
| 260 | Alam et al (2017) | 65/M | S5/6 | HCC | Primary tumor (partial) | 3 months | Immunological |
| 261 | Iwatani et al (2017) | 59/M | S8 | HCC | Primary tumor | Not reported | Ischemia (Duodenal Ulcer) |
| 262 | Murata et al (2017) | 67/M | S1/8 | HCC | Primary tumor | 2 months | Ischemia |
| 263 | Noij et al (2017) | 74/M | Diffuse hepatic | HCC | Primary (partial) & lung metastases | 6 months | Not reported |
| 264 | Oyama et al (2017) | 79/M | Diffuse hepatic | HCC | Primary tumor | Not reported | Hypoxia |
| 265 | Oyama et al (2017) | 78/F | Left lobe | HCC | Primary tumor | Not reported | Inflammatory; Immunological; Infection (Bacterial) |
| 266 | Sakamaki et al (2017) | 78/M | S8 | HCC | Primary tumor & lymph metastases | 3 months | Immunological; hemodialysis |
| 267 | Sano et al (2017) | 30/F | Common bile duct | NET | Primary tumor | Not Reported | Biopsy; Central necrosis |
| 268 | Yamaguchi et al (2017) | 63/M | Right lobe | HCC | Primary tumor | Not reported | Not reported |
| 269 | Yamaguchi et al (2017) | 67/M | S5 | HCC | Primary tumor | Not reported | Not reported |
| 270 | Yamaguchi et al (2017) | 84/F | S7 | HCC | Primary tumor | Not reported | Not reported |
| 271 | Yamaguchi et al (2017) | 60/M | S8/S1 | HCC | Primary tumor | Not reported | Not reported |
| 272 | El-Badrawy et al (2018) | 45/F | Porta hepatitis | DLBCL | Primary tumor | 18 days | Biopsy (Aspiration); Regional immune reaction |
| 273 | Goto et al (2018) | 64/M | S6/7 | HCC | Primary tumor | 1 month | Portal vein thrombosis; Immunological |
| 274 | Koya et al (2018) | 83/M | S2/3/4 | HCC | Primary tumor | 1 year | PVT |
| 275 | Lee et al (2018) | 67/M | Diffuse hepatic | HCC | Primary tumor | 1 year | Infection (diabetic foot); Ischemia (obstruction of portal vein thrombosis) |
| 276 | Taniai et al (2018) | 74/M | S7 | HCC | Primary tumor | 2 years | Not Reported |
| 277 | Taniguchi et al (2018) | 70/M | S3 | HCC | Primary tumor | Not reported | Ischemia (dialysis); Drugs (Elythrocine Steroids) |
| 278 | Alhatem et al (2019) | 60/M | Right lobe | DLBCL | Primary tumor | 4 years | Immunological (HIV, Hep C); Genetic |
| 279 | Chohan et al (2019) | 79/F | S6 | HCC | Primary (partial) & lung metastases | 1.5 years* | Ischemia; Immunological |
| 280 | Fujikawa et al (2019) | 78/M | S2/7 | HCC | Primary tumor | Not reported | Anemia (fracture) |
| 281 | Hirota et al (2019) | 67/M | S7 | HCC | Primary tumor | Not reported | Ischemia; Cessation of alcohol and smoking |
| 282 | Kim et al (2019) | 70/M | Bile duct | CCA | Liver Metastasis | 3 months | Abscopal effect; Post-radiotherapy antitumoral immunity |
| 283 | Kawaguchi et al (2019) | 68/M | S3 | HCC | Primary tumor | 2.5 months | Antiangiogenesis (SGLT2i) |
| 284 | Lee et al (2019) | 78/F | S5-8 | HCC | Primary tumor | 1 month | Immunological; Ischemia (rapid tumor growth or |

| | | | | | | | disruption of feeding artery) |
|-----|---------------------------|------|-------------------|----------------|--|--------------|---|
| 285 | Yoshida et al (2019) | 71/F | Right lobe | Clear cell HCC | Primary tumor | 1 year | Not reported |
| 286 | Arjunan et al (2020) | 53/M | Liver | HCC | Pulmonary, Omental, retroperitoneal metastases | 5 years | Immunological |
| 287 | Arjunan et al (2020) | 48/M | Left Lobe | HCC | Pulmonary metastases | 13 years | Immunological |
| 288 | Arjunan et al (2020) | 62/M | Liver | HCC | Systemic metastases | 11 years | Immunological |
| 289 | Arjunan et al (2020) | 73/M | Liver | HCC | Primary tumor | 6 years | Immunological |
| 290 | Costa-Santos et al (2020) | 68/M | S4 | HCC | Hepatic lesions | 5 years | Megestrol; Herbal remedies |
| 291 | Hokkoku et al (2020) | 77/M | S6 | HCC | Primary tumor | Not reported | Not reported |
| 292 | Muroya et al (2020) | 78/M | Right lobe | HCC | Lung metastases | 1 year | Hypoxia; Immunological; Dialysis |
| 293 | Nakamoto et al (2020) | 74/M | Right lobe | HSTCL | Hepatic, splenic, & osseous lesions | 1.5 months | Biopsy |
| 294 | Ohmatsu et al (2020) | 77/M | Right lobe | HCC | Lung metastases | 1 month | Abscopal Effect |
| 295 | Onishi et al (2020) | 28/M | Left lobe | HEHE | Primary lesion (partial) | 6 years* | Unpredictable growth (new lesions) |
| 296 | Onishi et al (2020) | 44/M | Unspecified liver | HEHE | Primary lesion (partial) | 4 years* | Unpredictable growth (new lesions) |
| 297 | Onishi et al (2020) | 47/M | Unspecified liver | HEHE | Primary lesion (partial) | 12 years | Calcification |
| 298 | Onishi et al (2020) | 51/F | S6 | HEHE | Primary lesion (partial) | 11.5 years* | Unpredictable growth (new lesions) |
| 299 | Onishi et al (2020) | 61/F | Unspecified liver | HEHE | Primary lesion (partial) | 5.5 years* | Unpredictable growth (new lesions) |
| 300 | Onishi et al (2020) | 63/M | Unspecified liver | HEHE | Primary lesion (partial) | 6 years* | Unpredictable growth (new lesions) |
| 301 | Raufi et al (2020) | 63/M | Porta hepatis | PHNEC | Pulmonary metastases | 2 months | Immunological |
| 302 | Sakamoto et al (2020) | 62/M | S3 | HCC | Primary tumor | Not reported | Ischemia |
| 303 | Sakamoto et al (2020) | 75/F | S4 | HCC | Primary tumor | Not reported | Tumor hypoxia (bleeding from rectal varicose veins) |
| 304 | Sonbare et al (2020) | 74/M | S8 | HCC | Primary tumor | 1 year | Immunological; Ischemia |
| 305 | Franses et al (2021) | 64/M | S4 | HCC | Primary tumor | 2 months | Immunological |
| 306 | Kakuta et al (2021) | 71/M | Not reported | HCC | Lung metastases | 3 months* | TACE of Primary |
| 307 | Kimura et al (2021) | 84/F | S8 | HCC | Primary tumor (partial) | Not reported | Immunological |
| 308 | Liu et al (2021) | 67/M | Diffuse hepatic | HCC | Pulmonary metastases | 5 months | Immunological; Chinese herbal remedies |
| 309 | Obu et al (2021) | 83/M | S2 | HCC | Primary tumor | 1 year | Ischemia (rapid growth); Capsule formation; PVT |

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|-------------------|--------------------|-------------------------|----------------------|----------------------|-----------------------|---|------------------------------------|---|
| | 310 | Tanaka et al (2021) | 71/F | Diffuse hepatic | HHL | Hepatic lesions | 1 year | Cessation of immunosuppressive therapy |
| Pancreatic cancer | 311 | Shapiro (1967) | ?/F | Unspecified pancreas | PDAC | Primary tumor | 7.5 years | Not reported |
| | 312 | Lokich et al (1973) | 42/M | Pancreatic head | PDAC | Primary tumor | 2 years* | Not reported |
| | 313 | Eidemiller et al (1971) | 7/M | Pancreatic head | PDAC | Primary tumor | 6 years | Not reported |
| | 314 | Tchertkoff et al (1974) | 21/M | Pancreatic head | PDAC | Primary tumor | 12 years | Bacterial Infection |
| | 315 | Cann et al (2003) | 50/M | Pancreatic body | PDAC | Primary tumor | 6 months | Acute febrile response; alternative therapies; Chinese herbs; High-dose Vitamin C |
| | 316 | Chin et al (2017) | 77/M | Pancreatic head | PDAC | Primary tumor & liver metastases | 1 year | Leukocyte activation; Fever; Allergenic & hormonal influences |
| | 317 | Sreevathsa et al (2018) | 32/M | Pancreatic body | pNET (Carcinoid) | Primary tumor | 19 years | Apoptosis (cytokines); VEGF blockade |
| | 318 | Lemus et al (2019) | 56/F | Pancreatic body/tail | PDAC | Primary tumor & liver metastases | 3 years | Immunogenic; angiogenic effects on the tumor microenvironment. |
| | 319 | Ibrahimi et al (2019) | 59/F | Residual pancreas | PDAC | Primary tumor (partial) | 1 year | Acute pancreatitis; Bacterial/fungal infection (abscess) |
| | 320 | Kawaguchi et al (2021) | 66/F | Pancreatic tail | PDAC | Primary tumor (partial) | 1 month | Not reported |
| | Small bowel cancer | 321 | Sroujeh et al (1988) | 55/M | Occult (Ileal lesion) | MM | Intestinal lesion (occult primary) | 7 years |
| 322 | | Nagashima et al (1996) | 58/M | Duodenum | MALT Lymphoma | Primary tumor | 1 year | Eradication of H. Pylori |
| 323 | | Rayson et al (1996) | 45/F | Ileum | GEP-NET (Carcinoid) | Liver metastases | 5 months* | Valvular surgery for carcinoid heart disease |
| 324 | | Horiuhi et al (2003) | 74/F | Diffuse enteric | NKTCL | Upper Abdominal tumors (non-radiated) | 1 year | Abscopal Effect |
| 325 | | Makino et al (2010) | 38/M | Terminal Ileum | MALT Lymphoma | Primary tumor & ileocecal lymphadenopathy | 1 year | Resolution of infection; Inflammatory |
| 326 | | Hayashi et al (2013) | 64/F | Duodenum | FL | Primary tumor | 5.5 years | Eradication of H. Pylori |
| 327 | | Tanaka et al (2014) | 61/F | Duodenum | SBAC | Primary tumor & liver metastases | 4 months | Methotrexate |
| 328 | | Sasaki et al (2016) | 60/M | Ileum | RL | Primary tumor | Not reported* | Radiography Radiation |
| 329 | | Hori et al (2017) | 20/F | Small intestine | EAS | Lung & mediastinal lymph metastases | 2 months | Immunological; Biopsy (transbronchial); Inflammatory |
| 330 | | Tanaka et al (2019) | 35/M | Small intestine | DLBCL | Primary tumor & lymph metastases | 3 years | Immunological (PD-L1/PD-1 axis) |
| | 331 | Most (1927) | 57/M | Rectum | CRAC | Local recurrence | 9 years | Sepsis |
| | 332 | Henry (1944) | 60/M | Rectum | CRAC | Primary tumor & liver metastases | 11 years | Not reported |
| | 333 | Ferguson (1954) | 45/M | Descending colon | CRAC | Primary tumor with local extension | 10 years | Severe sepsis (abscess) |
| | 334 | Dunphy (1956) | 46/M | Rectum | CRAC | Primary tumor & liver metastases | 8 years | Severe debilitation; Fecal diversion |
| | 335 | Ellison (1956) | 59/M | Rectum | CRAC | Peritoneal carcinomatosis | 3 years | Persistent high fever (Pneumonia) |
| | 336 | Fallis (1959) | 42/M | Transverse colon | CRAC | Primary tumor with local extension | 18 years | Severe sepsis (abscess); Fecal diversion; Religious rituals |

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| | 337 | Brown (1961) | 54/F | Sigmoid colon | CRAC | Primary tumor & liver metastases | 3 years | Not reported |
| | 338 | Brunschwig et al (1963) | 68/F | Rectum | CRAC | Local recurrence | 14 years | Not reported |
| | 339 | Mayo et al (1963) | 63/F | Descending colon | CRAC | Liver metastases | 16 years | Not reported |
| | 340 | Fullerton & Hill (1963) | 58/F | Transverse colon | Anaplastic CRAC | Primary tumor | 16 years | Not reported |
| | 341 | Rankin et al (1965) | 44/M | Rectum | CRAC | Liver metastases | 9 years | Not reported |
| | 342 | Margolis & West (1967) | 71/M | Rectum | CRAC | Peritoneal carcinomatosis | 1 year | Fecal diversion |
| | 343 | Synder et al (1968) | 62/F | Sigmoid colon | CRAC | Primary tumor with local extension | 15 years | Persistent high fever (wound infection); Immunologic; Genetic |
| | 344 | Synder et al (1968) | 60/M | Cecum | CRAC | Peritoneal carcinomatosis | 9 years | Severe Sepsis (abscess w/ fecal fistula); Immunologic; Genetic |
| | 345 | Weinstock (1977) | 40/F | Sigmoid colon | CRAC | Primary tumor with local extension & liver metastases | 20 years | Psychological |
| | 346 | Meares (1979) | 64/M | Rectum | CRAC | Primary tumor | 1 year | Intensive meditation |
| | 347 | Glasser et al (1979) | 36/M | Ascending colon | CRAC | Primary tumor & peritoneal carcinomatosis | 28 years | Genetic factors |
| | 348 | Beechy et al (1986) | 23/F | Ascending colon | CRAC | Primary tumor with local extension, peritoneal, & liver metastases | 4 years | Not reported |
| | 349 | Tominaga et al (1999) | 44/F | Transverse colon | CRAC | Primary tumor | 3 years | Dislodged |
| | 350 | Wadsworth et al (1999) | 67/M | Rectum | CRAC | Pulmonary metastases | 3 years | Infection (Pneumonia) |
| | 351 | Okamura et al (2000) | 54/M | Rectum | MALT Lymphoma | Primary tumor | Not reported | Not reported |
| | 352 | Kamesui et al (2000) | 66/F | Ascending colon | CRAC | Primary tumor | 1 year | Dislodged |
| | 353 | Takenaka et al (2000) | 76/F | Rectum | MALT Lymphoma | Primary tumor | 1.5 years | Not reported |
| | 354 | Ikuta et al (2002) | 60/M | Rectum | ASC | Liver metastases | 2 years | Interruption of blood supply; growth factors |
| | 355 | Abdelrazeq et al (2005) | 51/M | Rectum | CRAC | Local recurrence & peritoneal carcinomatosis | 16 years | Immunologic; Metabolic; Endocrine; Diversion of carcinogen |
| | 356 | Itano et al (2006) | 63/M | Rectum | MM | Primary tumor | 2 years | Dislodged |
| | 357 | Tomiki et al (2007) | 80/F | Rectum | CRAC | Primary tumor | 5 years | Dislodged |
| | 358 | Kochi et al (2008) | 80/M | Transverse colon | CRAC | Primary tumor | 3 years | Dislodged |
| | 359 | Bir et al (2009) | 86/F | Ascending colon | CRAC | Peritoneal lymph node metastases | 2 years | Immunologic |
| | 360 | Sakamoto et al (2009) | 80/M | Rectum | CRAC | Primary tumor | 3 months | Immunological |
| | 361 | Shimizu et al (2010) | 80/M | Transverse colon | CRAC | Primary tumor | 5 years | Physical stimulation (peristaltic movement; dislodged) |
| Colorectal cancer | 362 | Sakuma et al (2011) | 64/M | Sigmoid colon | CRAC | Primary tumor | 3 months* | Not reported |

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| 363 | Nakashima et al (2012) | 76/F | Ileocecal junction | CRAC | Primary tumor | 2 months | Dislodged |
| 364 | Flynn et al (2013) | 36/M | Descending colon/rectum | DLBCL | Primary tumor | 6 months | Cessation of immunosuppressive therapy (infliximab & azathioprine) |
| 365 | Flynn et al (2013) | 52/M | Sigmoid colon | HL | Primary tumor | 1 year | Cessation of immunosuppressive therapy (infliximab & azathioprine) |
| 366 | Nakamura et al (2013) | 60/M | Rectum | CRAC | Primary tumor | 1.5 years | Biopsy; Immunological |
| 367 | Sekiguchi et al (2013) | 76/F | Cecum | CRAC | Primary tumor | 1.5 months | Hyperimmunity & perioperative stress (lung surgery) |
| 368 | Kihara et al (2014) | 64/M | Transverse colon | CRAC | Primary tumor | 1.5 months | Immunological |
| 369 | Mitchell et al (2014) | 75/F | Cecum | MC | Regional lymph metastases | 1 year | Immunological |
| 370 | Sewpaul et al (2014) | 35/F | Appendix (presumed) | GEP-NET (Carcinoid) | Primary tumor | 1 year | Pregnancy |
| 371 | Kyoichi et al (2015) | 65/M | Transverse colon | CRAC | Primary tumor | 1.5 months | Drugs (Metformin); Immunological |
| 372 | Serizawa et al (2015) | 75/M | Transverse colon | CRAC | Primary tumor | 2.5 months | Immunological; Apoptosis |
| 373 | Ito et al (2016) | 73/M | Ascending colon | CRAC | Primary tumor | 3.5 months | Biopsy; mechanical stimulation (intestinal peristalsis) |
| 374 | Nemésio et al (2016) | 51/F | Splenic angle | CRAC | Liver metastases | Not reported | Removal of Primary; Tumor necrosis |
| 375 | Chida et al (2017) | 80/M | Transverse colon | CRAC | Primary tumor | 1 month | Immunological (CD4 +T-cells) |
| 376 | Matsuki et al (2016) | 72/F | Ascending colon | CRAC | Liver metastases | 2 months | Infection (Biliary S/P pancreaticoduodenectomy) |
| 377 | Chuang et al (2018) | 74/F | Occult | CRAC | Lung metastases | 2 months | Abscopal effect |
| 378 | Yoshida et al (2018) | 73/M | Transverse colon | CRAC | Primary tumor | 2.5 months | Not reported |
| 379 | Fukutomi et al (2019) | 87/F | Transverse colon | CRAC | Primary tumor | 1 month | Immunological (CD8+ T-cells) |
| 380 | Karakuchi et al (2019) | 70/M | Transverse colon | CRAC | Primary tumor | 2 months | Immunological; Biopsy |
| 381 | Kawakita et al (2019) | 62/M | Ascending colon | CRAC | Primary tumor | 1 month | Immunological (CD4 +T-cells) |
| 382 | Tanaka et al (2019) | 63/F | Sigmoid colon | CRAC | Primary tumor | 2.5 months | Injection for non-lifting sign; Ischemia (vasoconstriction S/P epinephrine) |
| 383 | Utsumi et al (2020) | 78/M | Ascending colon | CRAC | Primary tumor | 1 month | Immunological (dMMR) |
| 384 | Utsumi et al (2020) | 66/M | Ascending colon | CRAC | Primary tumor | 1.5 months | Immunological (dMMR) |
| 385 | Utsumi et al (2020) | 73/M | Ascending colon | CRAC | Primary tumor | 1.5 years | Immunological (dMMR) |
| 386 | Mehawej et al (2020) | 18/F | Occult | CRAC | Primary tumor | Unknown | Not reported |
| 387 | Nishiura et al (2020) | 67/F | Transverse colon | CRAC | Primary tumor | 3 months | Immunological |
| | Yokota et al | | | | | | |

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| | 388 | (2020) | 76/F | Transverse colon | CRAC | Primary tumor | 2 months | Immunological |
| | 389 | Yokota et al (2020) | 64/F | Cecum | CRAC | Primary tumor | 3 months | Immunological |
| | 390 | Yokota et al (2020) | 64/M | Transverse colon | CRAC | Primary tumor | 1 month | Immunological |
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TABLE 4: Clinical features of cases demonstrating the spontaneous regression of gastrointestinal cancers

Additional Information

Disclosures

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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Authors' contributions: Conceived and designed the analysis: CDM, PL, CC, VR. Collected the data: CDM, PL, CC. Contributed data or analysis tools: CDM, PL, CC, AP. Performed the analysis: CDM, PL, AP, GA, AL. Wrote the manuscript: CDM, PL, AP, CC, GA, AL, VR. Approved final manuscript version: CDM, PL, AP, CC, GA, AL, VR.

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