

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. Secondarily, 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.

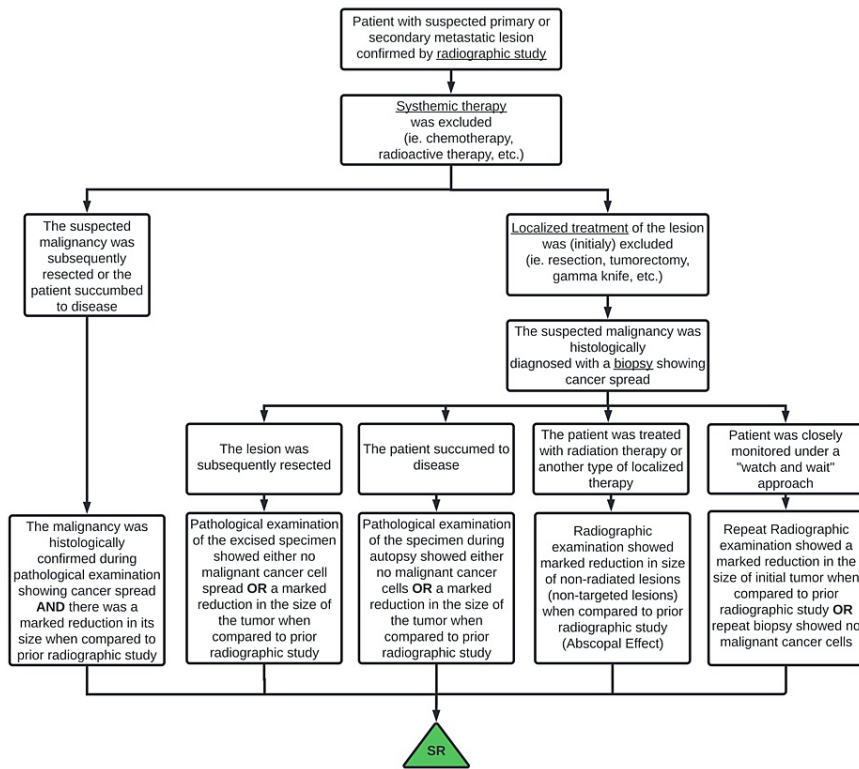


FIGURE 1: Clinician guidelines or criteria for reporting spontaneous regression

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

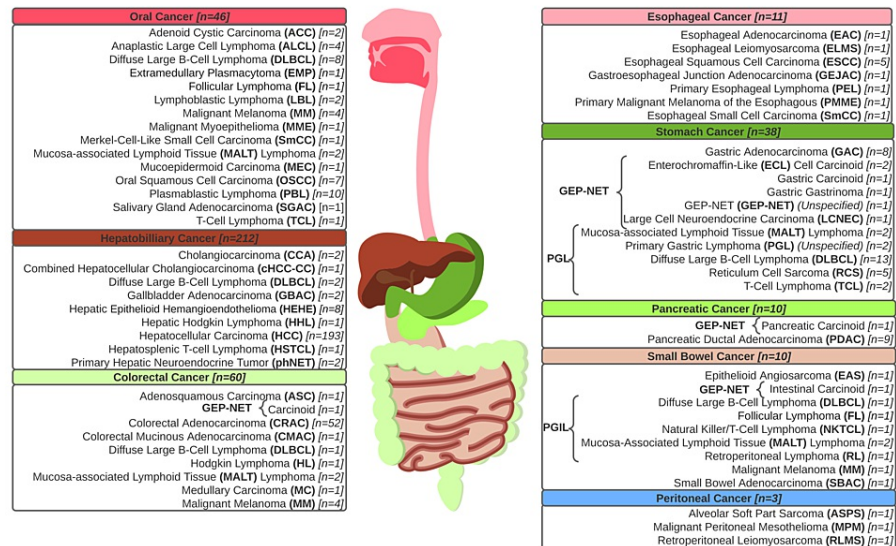


FIGURE 2: Histological manifestations of gastrointestinal malignancies are recorded to have undergone spontaneous regression throughout the clinical literature.

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	Extramedullary 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
	Grillet et al						

	2	(1984)	26/M	Parotid gland	ACC	Lung metastases	7 years	Diet
	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	Savarrio 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	Rujiroindakul 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
Oral cancer	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 (2014)	62/M	Mouth floor	OSCC	Primary tumor	3 months	Biopsy; Immunological
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)
47	Rees et al (1983)	49/M	Lower esophagus	EAC	Lung metastases	1 year	Abscopal effect
	Oshwada et		Thoracic		Primary tumor (partial)		Change in T-cell subsets;

Esophageal cancer	48	al (1990)	78/M	esophagus	ESCC	& pulmonary metastases	7 months	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)
	52	Takemura et al (1999)	63/F	Thoracic 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	

Stomach cancer	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
	77	Ogawa et al (1998)	63/F	Gastric corpus	PGL (DLBCL)	Primary tumor	13 months	H Pylori eradication
	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	

Primary peritoneal cancer	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
	102	McCaughan et 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-Royeula (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	76/M	Unspecified liver	HCC	Primary tumor	1 year	Not reported

	(1996)						
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	Magalotii et al (1998)	66/M	Unspecified liver	HCC	Primary tumor	4 years*	Not reported
128	Megalotii 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
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	Iiai 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
	Kato et al				Primary tumor & lung		

166	(2004)	77/M	Right lobe	HCC	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	Sunitinib (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 (2006)	70/M	Right lobe	HCC	Primary tumor (partial)	5 years	Bleeding (esophageal varices); Immunological
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

Hepatobiliary Cancer	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 (2011)	54/M	Right lobe	HCC	Primary tumor	2 years	Immunological; Necrosis
	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	Komatzu 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
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	Retroperitoneal lymph metastases	2 months	Immunological
236	Parks et al	67/M	Left lobe	HCC	Primary tumor	5 months	Immunological

237	(2015) 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 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 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)
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 hepatis	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 (Elythrocin 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
	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)	?/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	Not Reported
	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
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
	Takenaka et			MALT			

Colorectal cancer	353	al (2000)	76/F	Rectum	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)
	362	Sakuma et al (2011)	64/M	Sigmoid colon	CRAC	Primary tumor	3 months*	Not reported
	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
388	Yokota et al (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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