Seminars in Oncology 49 (2022) 152–159

Contents lists available at ScienceDirect

Seminars in Oncology

journal homepage: www.elsevier.com/locate/seminoncol

Radiation recall dermatitis: A review of the literature

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ARTICLE INFO

Article history:
Received 20 May 2020
Revised 20 August 2021
Accepted 1 April 2022

Keywords:
Radiation recall
Radiation dermatitis
Radiation recall dermatitis

ABSTRACT

Purpose/Objectives: Radiation recall dermatitis (RRD) is a skin reaction limited to an area of prior radiation triggered by the subsequent introduction of systemic therapy. To characterize RRD, we conducted a literature search, summarized RRD features, and compared the most common drug classes implicated in this phenomenon.

Materials/Methods: PubMed, Embase, Scopus, Web of Science, and Cochrane DBSR databases were queried through July 1, 2019 using key words: radiation recall, RRD, and radiodermatitis (limited to humans and English language). Studies included case reports in which patients treated with radiotherapy were initiated on a new line of systemic therapy and subsequently developed a skin reaction in the irradiated area. RRD cases were organized by whether RRD occurred after a single drug or multiple drug administration.

Results: One-hundred fifteen studies representing 129 RRD cases (96 single-drug RRD, 33 multi-drug) were included. Sixty-three drugs were associated with RRD. Docetaxel (22) and gemcitabine (18) were the two drugs most commonly associated with RRD. Breast cancer (69 cases) was the most commonly associated tumor type. For single-drug RRD, the median radiotherapy dose was 45.0 Gy (range, 30.0–63.2 Gy). The median time from radiotherapy to drug exposure, time from drug exposure to RRD and time to significant improvement was 8 weeks (range, 2–132 weeks), 5 days (range, 2–56 days), and 14 days (range, 7–49 days), respectively. Variables significantly associated with grade $\geq$2 toxicity were docetaxel ($P = 0.04$) and non-antifolate antimetabolite ($P = 0.05$). The only variable significantly associated with grade $\geq$3 toxicity was capecitabine ($P = 0.04$).

Conclusions: RRD is a complex toxicity that can occur after a wide range of radiotherapy doses and many different systemic agents. Most commonly, it presents in patients diagnosed with breast cancer and after administration of a taxane or antimetabolite medication. RRD treatment generally consists of corticosteroids with consideration of antibiotics if superinfection is suspected. Drug re-challenge may be considered after RRD if the initial reaction was of mild intensity.

Introduction

Radiation recall dermatitis (RRD) is a recognized but incompletely understood clinical phenomenon. RRD occurs when a patient develops an acute skin reaction, most commonly erythema, confined to a previously irradiated area after the subsequent administration of systemic therapy. The true incidence of RRD is unknown and may depend on several factors including radiotherapy dose, type of systemic therapy, time from end of radiotherapy to systemic therapy administration, and systemic therapy dose. A subset analysis of breast cancer patients who received chemotherapy after radiation in the MammoSite RTS registry trial reported that approximately 10% of patients experienced radiation recall dermatitis when chemotherapy was administered at least 1 week after the end of radiation [1]. Although chemotherapy was not standardized, the majority of patients received a doxorubicin-based regimen.

Since one of the earliest RRD case reports in 1959, numerous instances of RRD have been reported in the literature [2]. While these have raised awareness of RRD as a potential adverse effect, the wide variation in description and lack of data from large patient cohorts make it challenging to define RRD. Furthermore, skin radiosensitization by a systemic agent must be distinguished from RRD. Camidge et al suggested that RRD should only be diagnosed

Abbreviations: Multi-Drug RRD, Radiation Recall Dermatitis occurring after the administration of a multi-drug regimen; RRD, Radiation Recall Dermatitis; Single-Drug RRD, Radiation Recall Dermatitis occurring after the administration of a single drug; CTCAE, Common Terminology Criteria for Adverse Events.

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https://doi.org/10.1053/j.seminoncol.2022.04.001
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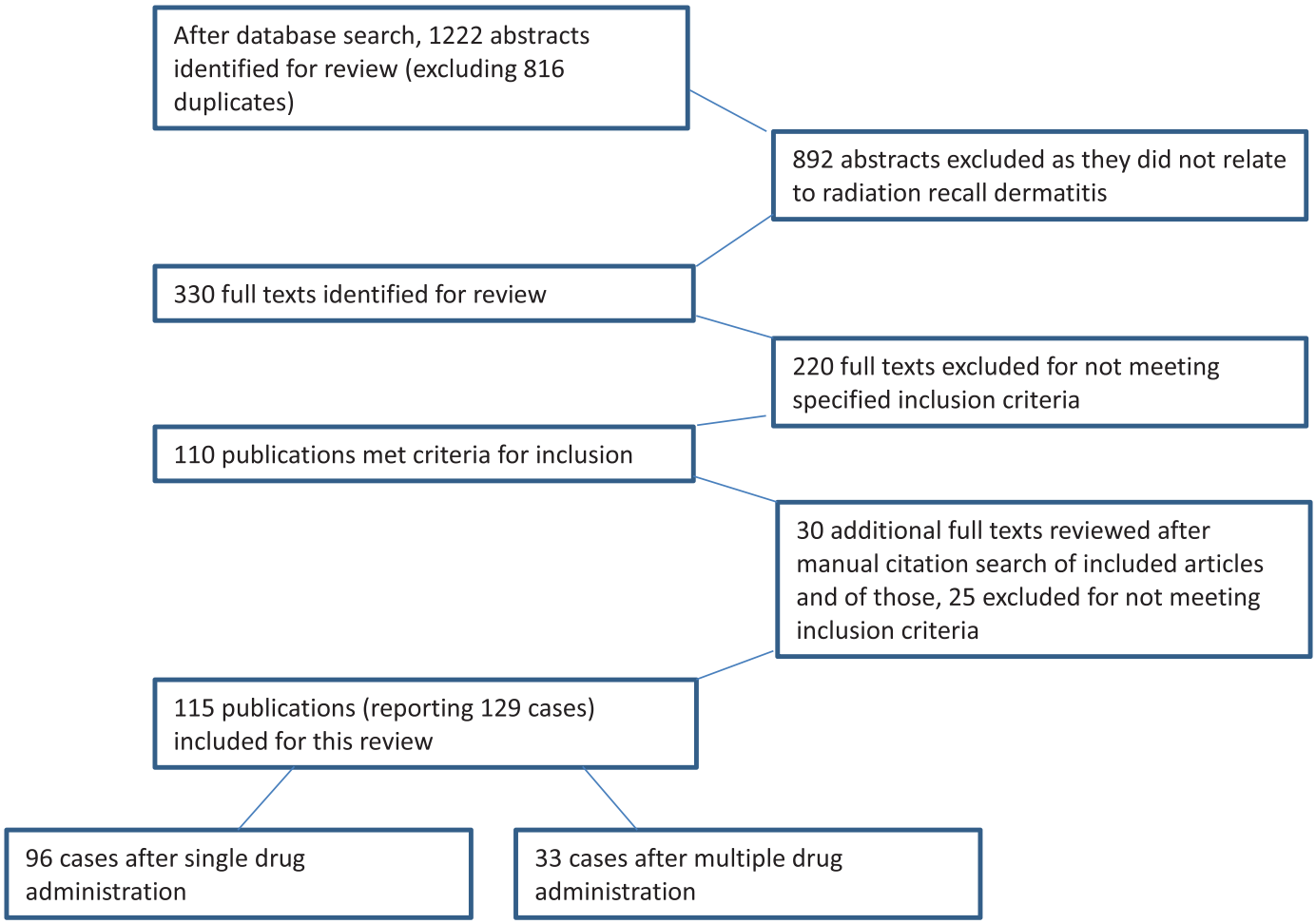

Figure 1. Search results.

when the systemic therapy is administered at least 7 days after the end of radiotherapy—a suggestion based on prior studies examining skin reactions within the context of radiotherapy and systemic therapy [3]. Radiosensitization, in contrast, happens when radiosensitizers are given before or during radiation and enhance the effect of radiation resulting in a more intense skin reaction than if radiation was administered alone. This distinction is complicated because many of the RRD-associated agents are also considered radiosensitizers.

The clinical importance of RRD is two-fold. First, the appropriate diagnosis of RRD must be made so that treatment—typically supportive care often with either topical or systemic corticosteroids—can be tailored to this etiology. Second, the therapeutic benefit of continuing the systemic therapy must be assessed with respect to the risk of exacerbating the reaction. Previously published reviews have described RRD and listed individual cases or separated them by individual systemic therapy agents [3–5]. Given the continued development of novel systemic anti-cancer therapies and the need for an updated understanding of RRD, we conducted a review of the literature.

Material and methods

Study identification

With the assistance of a research librarian, a systematic literature search was conducted to identify potentially relevant studies. PubMed, Embase, Scopus, Web of Science, and Cochrane DBSR databases were queried through July 1, 2019 using RRD-related key words: “radiation recall,” “RRD,” and “radiodermatitis” (limited to humans and English language but without limitation on year published). A manual check of references from all included studies was conducted to find potentially relevant studies not identified in the initial search. The study team reviewed all abstracts found through the above search strategy and identified relevant articles for full text review.

Inclusion criteria

Only case reports or case series were included for analysis. This decision was made because RRD events have primarily been published in case report format and because case reports provide more detail regarding the circumstances surrounding individual RRD events. Case reports were included if the patient had previously undergone radiotherapy for any cancer indication, began a new systemic therapy at least 7 days after the end of radiation and subsequently developed a skin reaction within a previously irradiated area. Patients of any age were included. Cases were included if the RRD occurred after administration of either single drugs (single-drug RRD) or after multiple drugs (multi-drug RRD). Systemic therapy may have been administered multiple times prior to the onset of RRD as long as the first administration was at least 7 days post-radiotherapy completion.

Data extraction

Relevant characteristics were collected from each included literature report in the following categories: patient, tumor, radio-

therapy, systemic therapy, symptom, treatment, outcome, and re-challenge (when applicable) characteristics. In addition to recording patient race, when color photographs were provided, patient skin types were graded according to the Fitzpatrick skin phototype scale—a subjective scale from 1 to 6 (in order of decreasing sensitivity, 3 is considered average sensitivity) used in evaluating skin sensitivity to sun exposure [6].

Each drug was organized into its pharmaceutical class. If there was only one case within a specified drug class, these systemic agents were grouped into an "other" category. Anti-folates were considered a separate drug class than antimetabolites for this review. For multi-drug RRD, a primary causative drug was noted when suggested in the case report itself. The radiation dermatitis section of the Common Terminology Criteria for Adverse Events (CTCAE) version 5 was used by the study team to grade skin toxicity based on the description provided in the case report.

Statistical analysis

Descriptive statistics were used to analyze single-drug RRD cases by drug class and multi-drug regimens for which multiple cases were recorded. Due to the small numbers within each drug class, Kruskal-Wallis and Fisher's exact test were used to compare the two most common drug classes which both had greater than 10 cases. Logistic regression was used for univariate analysis to determine the association of collected variables with grade $\geq$2 and $\geq$3 radiation dermatitis toxicity. Linear regression was used for univariate analysis to determine the association of collected variables with time to RRD improvement in days. Statistical analysis was conducted in R (R Core Team 2019 Version 3.6.2).

Results

Search results and study characteristics

The initial search of five databases yielded 1,222 abstracts for review after excluding duplicates (Fig. 1). Ultimately, 115 studies reporting 129 individual cases met inclusion criteria for this review (96 cases were after single-drug administration and 33 cases were after multi-drug administration) [5,7-120].

The majority of RRD case reports were published within the past two decades. One-hundred seven (83%) cases have been published since the year 2000 (52 [40%] cases from 2000 to 2009 and 55 [43%] cases from 2010 to 2019) compared to only 22 (17%) cases prior to 2000. For multi-drug RRD cases specifically, 21 (62%) cases were published from 2010 to 2019.

Systemic agents associated with RRD

Tables 1 and 2 display the individual systemic agents and drug classes associated with RRD. Forty drugs were associated with RRD after their administration as a single agent. Twenty-three additional drugs were associated with RRD but only in combination with other agents (multi-drug). The drugs most commonly reported with RRD were docetaxel (22 cases) and gemcitabine (18 cases). No other drugs were reported with RRD in more than 10 cases. Three drug classes were associated with more than 20 cases: taxanes (31), antimetabolites (30), and alkylating agents (25 cases). The remaining drug classes were associated with less than 10 cases each. The only immunotherapy agent associated with RRD was nivolumab (two cases). Given the variety of clinical scenarios in which systemic therapy was employed, wide variation of drug doses and regimens were noted but are not reported. However, 68 (53%) of cases did report that more than one dose of the medication was given prior to RRD onset.

Table 1
Systemic agents associated with RRD.

Drug name	Single-drug cases	Multi-drug cases	Total
Acetaminophen	0	1	1
Amlodipine	0	1	1
Anastrozole	0	1	1
Arsenic Trioxide	1	0	1
Azithromycin	1	0	1
Bleomycin	1	0	1
Capecitabine	4	1	5
Carboplatin	0	5	5
Carmustine	0	1	1
Cefotetan	1	0	1
Chlorambucil	1	0	1
Chlorpheniramine	0	1	1
Cisplatin	0	8	8
Codeine	1	0	1
Cyclophosphamide	1	6	7
Cytarabine	0	2	2
Dabrafenib	0	2	2
Dacarbazine	1	0	1
Docetaxel	15	7	22
Doxorubicin	3	3	6
Edatrexate	1	0	1
Epirubicin	0	1	1
Erlotinib	1	0	1
Etoposide	1	1	2
Everolimus	1	1	2
Exemestane	1	1	2
Fluorouracil	0	2	2
Gaitfloxacin	1	0	1
Ganitumab	1	0	1
Gemcitabine	11	7	18
Goserelin	0	2	2
Hydroxyurea	3	0	3
Hypericin	1	0	1
Idarubicin	0	1	1
Iodinated Contrast	1	0	1
Ixabepilone	2	1	3
Lanreotide	1	0	1
Letrozole	1	0	1
Leucovorin	0	1	1
Levetiracetam	0	1	1
Levofloxacin	2	0	2
Melphalan	0	1	1
Methotrexate	3	0	3
Nimesulide	0	1	1
Nitrofurantoin	1	0	1
Nivolumab	2	0	2
Oxaliplatin	0	1	1
Paclitaxel	5	4	9
Pazopanib	0	1	1
Pemetrexed	4	0	4
Phentermine	1	0	1
Rituxumab	0	1	1
Rosuvastatin	0	1	1
Simvastin	1	0	1
Sorafenib	6	0	6
Sunitinib	1	0	1
Tamoxifen	3	2	5
Trametinib	0	1	1
Trastuzumab	3	2	5
Trimethoprim-sulfamethoxazole	1	0	1
Vemurafenib	4	0	4
Vinblastine	2	0	2
Vincristine	0	2	2

RRD characteristics

Disease sites involved with RRD were the following: breast (n = 69, 53%), gastrointestinal (n = 18, 14%), lung (n = 9, 7%) hematologic (n = 8, 6%), genitourinary/head and neck/skin (n = 7, 5% each) and sarcoma (n = 4, 3%). Similarly, body locations of RRD were breast/chest wall (n = 60, 47%), abdomen/pelvis (n = 23, 18%), back (n = 19, 15%), head and neck (n = 16, 12%), and extremity (n = 11, 9%) (Fig. 2).

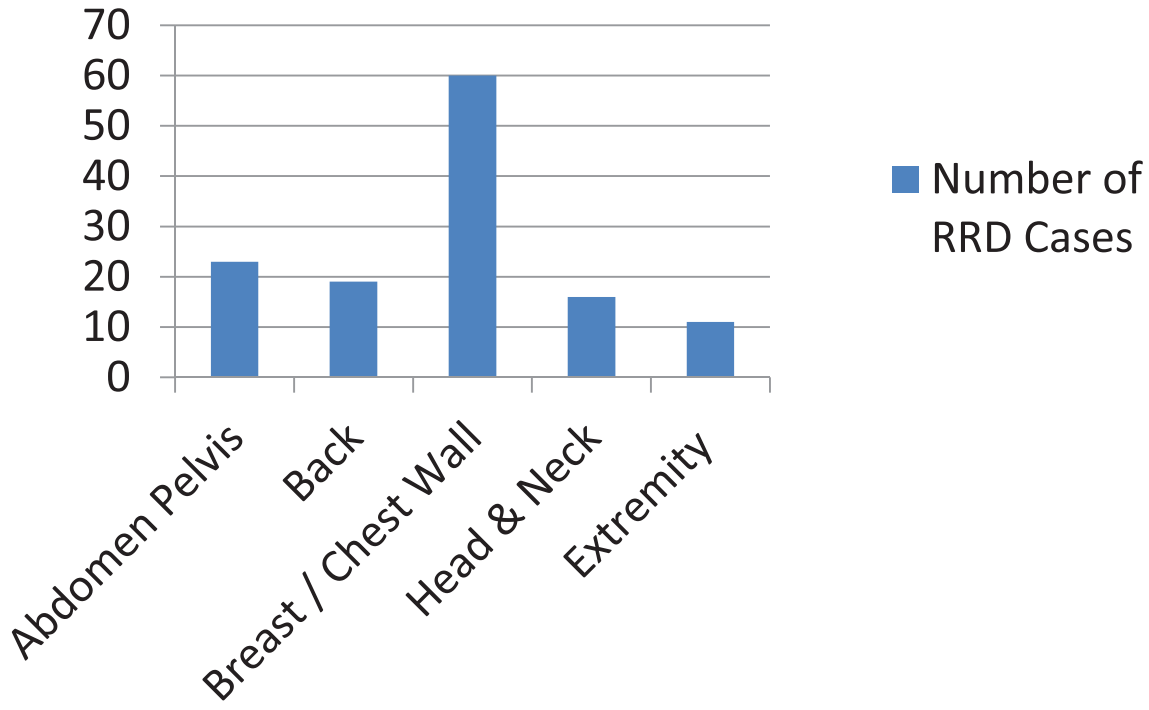

Figure 2. RRD cases by body site.

Table 2
Drug classes associated with RRD.

Drug class name	Single-drug cases	Multi-drug cases	Total
Alkylating agent	3	22	25
Anthracycline	3	5	8
Antifolate	8	0	8
Anti-HER2	3	2	5
Antimetabolite (non-antifolate)	18	12	30
Antimicrotubule	2	1	3
Aromatase inhibitor	2	2	4
BRAF inhibitor	4	2	6
Fluoroquinones	3	0	3
GnRH agonist	0	2	2
mTOR inhibitor	1	1	2
Other	13	8	21
PD-1 inhibitor	2	0	2
SERM	3	2	5
Statin	1	1	2
Taxane	20	11	31
TKI	7	1	8
Topoisomerase II inhibitor	1	1	2
Vinca alkaloid	2	2	4

Of the 24 cases reporting a patient's race, 15 (63%) patients were Caucasian. Seventy publications provided a color photograph of the patient and of these, 57 (81%) were Fitzpatrick skin phototypes 1–2 (indicating increased sun sensitivity compared to average). On presentation, patients experienced pain in 46 (37%) cases and pruritus in 21 (16%) cases. The most common terms to describe the patient's skin reaction were erythema 103 (80%), edema 26 (20%), desquamation 20 (16%), vesicular 10 (8%), and ulceration 7 (5%). A second site of RRD due to additional prior radiation was reported in 12 (9%) of cases. Furthermore, concurrent non-dermatitis radiation recall reactions occurred in 17 (13%) patients—including mucositis, myositis, and stomatitis.

Radiation dose, fractionation, time from end of radiotherapy to drug administration in weeks, time from systemic therapy administration to RRD in days, time to resolution, and CTCAE grade are reported by drug classes for single-drug RRD and by systemic regimen for multi-drug RRD (Tables 3 and 4). For single-drug RRD, the median radiotherapy dose was 45.0 Gy (range, 30.0–63.2 Gy). The median time from radiotherapy to drug exposure, time from drug exposure to RRD and time to significant improvement was 8 weeks (range, 2–132 weeks), 5 days (range, 2–56 days), and 14 days (range, 7–49 days), respectively. The two most common drug classes administered in single-drug RRD were antimetabolites (non-antifolate) and taxanes. Comparing these two groups, similar RT doses (median 44.6 *v* 47.5 Gy, P= 0.79), time to drug exposure after RT (median 8 *v* 5 weeks, P= 0.48), time to RRD diagnosis from most recent drug dose (median 10 *v* 5 days, P= 0.22) and CTCAE grade 3+ (11 *v* 20%, P= 0.453) were observed. However, median time to RRD improvement differed significantly with taxane-induced RRD resolving more quickly than antimetabolite-induced RRD (median 21 *v* 7 days, P= 0.03).

CTCAE radiation dermatitis grade was grade 1 in 71 cases (55%), grade 2 in 44 cases (34%), grade 3 in 10 cases (8%), and grade 4 in 4 cases (3%).

Skin biopsy was performed in 28 (22%) cases. From the provided pathologic descriptions, inflammation/infiltration was noted to be lymphocytic in 14 (50%) cases, perivascular in 12 (43%) cases, radiation-related 6 (21%) cases, and drug-related 1 (4%) case (none of these descriptions were mutually exclusive).

Treatment and re-challenge

Corticosteroids and antibiotics were commonly used in the treatment of RRD. Fifty-two (40%) patients received a corticosteroid treatment. Of those, routes of administration were as follows: 29 (56%) topical only, 16 (31%, 5 patients also received topical) oral, 5 (10%) intravenous, and 2 (4%) were not specified. As infection was within the differential for these skin reactions, antibiotics were prescribed in 22 (17%) of cases with the following routes of administration: 4 (18%) topical, 13 (59%, 4 patients also received topical) oral, and 5 (23%) intravenous. Fifteen (68%) patients who received antibiotics also received some form of corticosteroid treatment. Only seven (5%) patients were hospitalized likely related to RRD.

Table 3
Single-drug RRD characteristics by drug class.

Drug class	Number of cases	RT Dose (Gy)[1]	Fractions[1]	TTDE RT (wk)[1]	TFDE RRD (d)[1]	CTCAE Grade[1]	TTSI (d)[1]
BRAF inhibitor	4	32.5 (27.5, 44.0)	22 (13, 30)	5 (3, 7)	11 (7, 14)	2 (1, 2)	35 (25, 47)
Other[2]	16	50.0 (43.7, 60.0)	25 (25, 33)	32 (6, 60)	3 (1, 6)	1.(1, 1)	13 (7, 21)
PD-1 inhibitor	2	37.5 (23.3, 51.8.)	3 (3, 3)	21 (11, 30)	4 (4, 5)	2 (1, 2)	14 (14, 14)
SERM	3	52.2 (51.3, 53.1)	NA	104 (104, 108)	56 (31, 70)	1 (1, 2)	49 (32, 85)
TKI	7	30.0(30.0, 33.0)	8 (6, 10)	2 (1, 5)	11 (7, 14)	1 (1, 2)	14 (14, 14)
Alkylating agent	3	36.0 (33.0, 43.0)	25 (25, 25)	24 (20, 28)	7 (4, 9)	1 (1, 2)	3 (2, 4)
Anthracycline	3	30.0 (25.0, 30.0)	NA	3 (3, 4)	12 (12, 15)	2 (2, 2)	23 (19, 28)
Anti-HER2	3	50.4 (47.7, 52.2)	28 (24, 29)	6 (4, 26)	21 (15, 95)	1 (1, 1)	11 (9, 12)
Antifolate	8	40.0 (34.5, 49.0)	11 (11, 12)	22 (6, 199)	3 (1, 6)	2 (1, 2)	14(7, 21)
Antimetabolite (non-antifolate)	18	44.6 (30.0, 50.3)	14 (10, 26)	8 (4, 36)	10 (3, 40)	1 (1, 2)	21 (15, 25)
Antimicrotubule	2	30.3 (30.3, 30.3)	NA	83 (42, 124)	5 (4, 6)	3 (2, 3)	16 (13, 18)
Aromatase Inhibitor	2	63.2 (61.8, 64.6)	NA	5 (3, 6.)	16 (8, 23)	2 (1, 2)	16 (10, 23)
Fluoroquinolone	3	59.4 (54.7, 60.1)	25 (25, 25)	132 (80, 138)	3 (3, 6)	1 (1, 2)	10(7, 12)
Taxane	20	47.5 (32.2, 50.3)	25 (19, 25)	5 (2, 40)	5 (4, 8)	2 (1, 2)	7 (7, 10)
Vinca alkaloid	2	41.7 (34.4, 49.1)	15 (15, 15)	24 (16, 32)	2 (1, 2)	2 (2, 2)	10 (7, 12)

RRD = radiation recall dermatitis; RT = radiotherapy; TTDE = time to drug exposure from RT; TFDE = time from drug exposure to RRD; CTCAE = Common Terminology Criteria for Adverse Events; TTSI = time to significant improvement
[1] All are median values (first, third quartiles). All values are rounded to nearest whole number, except for RT dose, which is rounded to the nearest tenth.
[2] Other category includes drug classes for which there was only one instance of RRD.

Table 4
Multi-drug RRD characteristics by drug regimen.

Drug regimen[1]	Number of cases	RT dose (Gy)	Number of fractions	TTDE RT (wk)	TFDE RRD (d)	CTCAE grade	TTSI (d)
Docetaxel, Cyclophosphamide[3]	3	38.5 (36.3, 44.3)	10 (10, 18)	3 (3, 4)	7 (7, 14)	2 (2, 2)	35 (19, 40)
Gemcitabine, Cisplatin[3]	2	32.0 (20.0, 44.0)	32 (32, 32)	11 (8, 15)	11 (9, 12)	2 (1, 2)	28 (NA)
Gemcitabine, Carboplatin[3]	4	45.0 (40.5, 56.5)	25 (19, 30)	32 (20, 61)	8 (8, 15)	1 (1, 1)	30 (25.5, 36)
Goserelin, Tamoxifen[3]	2	45.0 (45.0, 45.0)	23 (23, 23)	12 (12, 12)	2 (2, 2)	2 (2, 23)	3 (NA)
Paclitaxel, Cisplatin[3]	4	47.2 (44.0, 55.4)	16 (16, 22)	24 (12, 32)	2. (1, 4)	1 (1, 1)	3.5 (1, 8)
Capecitabine, Ixabepilone	1	57.8	40	2	3	2	NA
Cyclophosphamide, Doxorubicin, Vincristine[2]	1	54.8	25	5	3	1	NA
Dabrafenib, Pazopanib	1	30.0	NA	8	1	1	16
Dabrafenib, Trametini	1	56.0	NA	16	90	1	NA
Docetaxel, Carbopaltin, Trastuzumab	1	60.0	NA	2	3	2	14
Docetaxel, Cisplatin	1	70.0	33	64	5	1	8
Docetaxel, Epirubicin	1	NA	NA	312	5	2	14
Doxorubicin, Docetaxel, Cyclophosphamide	1	64.4	NA	2	4	3	14
Docetaxel, Vincristine	1	45.0	9	364	NA	4	NA
Everolimus, Exemestane	1	NA	NA	550	3	2	14
Fluorouracil, Cisplatin	1	50.4	NA	4	90	1	NA
Gemcitabine, Trastuzumab	1	30.0	10	22	14	1	NA
Idarubicin, Cytarabine	1	81.0	NA	156	14	2	21
Luecovorin, Fluorouracil, Oxaliplatin	1	20.0	5	2	14	1	NA
Levetiracetam, Anastrozole	1	60.0	NA	10	7	1	5
Nimesulide, Acetaminophen, Chlorpheniramine	1	66.0		130	1	1	4
Rituximab, Carmustine, Etoposide, Cytarabine[2]	1	30.0	NA	2	3	1	5
Rosuvastatin, Amlodipine	1	50.4	28	264	14	2	21

RRD = radiation recall dermatitis; RT = radiotherapy; TTDE = time to drug exposure from RT; TFDE = time from drug exposure to reaction; CTCAE = Common Terminology Criteria for Adverse Events; TTSI = time to significant improvement; NA = not available.
[1] The suspected primary causative agent (per the publication) is listed first except for those regimens designated[2].
[2] No primary causative agent identified (per the publication).
[3] Descriptive statistics utilized given more than one case with associated drug regimen. All are median values (first, third quartile). All values are rounded to nearest whole number, except for RT dose which is rounded to the nearest 10th.

Seventy-one (55%) patients underwent drug re-challenge in which they received a subsequent dose of the presumed RRD-inciting agent. For these patients, the majority (56, 79%) received the same drug dose as initially administered while the minority received a smaller (16, 23%) or an increased (9, 13%) dose. A skin reaction developed in 35 (49%) patients after drug re-challenge with 16 (23%) described as less intense compared to the initial RRD, 10 (14%) were the same intensity, and 9 (13%) were worse intensity.

Univariate analysis for CTCAE grade and time to resolution

The following variables were analyzed for their association with CTCAE grade ≥2 and grade ≥3 radiation dermatitis toxicity: drug, drug class, drug route of administration (oral *v* intravenous), Fitzpatrick skin type, primary tumor site, location of RRD, radiation dose and number of drug doses received prior to reaction. Variables significantly associated with grade ≥2 toxicity were docetaxel (P= 0.04) and non-antifolate antimetabolite (P= 0.05).The only variable significantly associated with grade ≥3 toxicity was capecitabine (P= 0.04).

In addition to the above variables, corticosteroid and antibiotic treatment variables were analyzed for their association with time to RRD improvement in days. Variables significantly associated with decreased time to RRD improvement were patients who received intravenous administration of the systemic agent (P< 0.01), oral antibiotic treatment (versus no treatment, P< 0.01) and fewer number of systemic agent doses received prior to RRD (P< 0.01).

Discussion

RRD is an uncommon clinical phenomenon but intriguing given the diverse circumstances under which it presents. Our review of individual cases provides an updated description of RRD compared to other historical reviews [3,5]. Moreover, this review provides a

quantitative description of some of the key characteristics related to this clinical entity and separates the analysis by single-drug and multi-drug RRD, hopefully mitigating some of the confusion regarding individual agents associated with RRD.

The most commonly implicated tumor and body site of RRD were breast cancer (53%) and breast/chest wall (47%), respectively. This could be explained by the prevalence of breast cancer and because many breast cancer patients undergo radiation and systemic therapy at some point during their treatment course. Likewise, the most common drug implicated was docetaxel (22 cases) and drug class was taxanes (31 cases), which are often used in breast cancer treatment. Across all tumor types, however, the time course of this clinical phenomenon is quite variable from end of radiotherapy to initiation of systemic therapy to RRD.

There is no standard approach to the treatment of RRD. Patients are often treated with topical steroids (the most common treatment in this study) but this was not shown to impact the time to RRD improvement. However, time to improvement was faster for patients treated with oral antibiotics. Therefore, there should be strong consideration for starting oral antibiotics in these patients when potential superimposed infection is suspected. Improvement was also faster in patients who received a fewer number of systemic agent doses prior to RRD onset, which may indicate that increased time to healing is needed after multiple doses of a RRD inciting drug. Interestingly, patients who received intravenous systemic therapy also experienced a faster time to improvement compared to those who received oral treatment. With regard to RRD severity, docetaxel and capecitabine were both associated with more severe reactions which should encourage earlier and more aggressive treatment of RRD for these patients. Taken together, the above factors should be considered to better understand the expected clinical course and recommended treatment for an individual patient.

The decision to re-challenge with the same medication is also important for medical oncologists as they weigh the therapeutic benefit of the drug against the risk of exacerbating the reaction. In our review, approximately half of all patients underwent drug re-challenge with approximately 50% of those patients developing another skin reaction. Only 13% had a worse skin reaction. Consequently, it may be reasonable to re-challenge an RRD patient with the inciting systemic agent at the same dose if the initial skin reaction was mild.

One central question left unanswered is related to the mechanism of RRD. Approximately one fourth of cases reported pathology from skin biopsies; about half of those noted inflammation/infiltration that was lymphocytic and/or perivascular. These descriptions do not provide much insight into the possible etiologies of RRD. Camidge et al discussed vascular damage from and stem cell inadequacy/sensitivity to radiotherapy as possible explanations. However, they ultimately suggested RRD is an idiosyncratic drug hypersensitivity reaction, inflammatory but not immune-mediated [3]. This explanation appears broad enough to encompass the wide breadth of RRD characteristics discussed in the current review.

Several limitations exist for this study. First, a widely accepted definition of RRD has not been firmly established given the ambiguous nature of this phenomenon. As discussed previously, we decided to build upon the definition provided by Camidge et al in one of the first comprehensive reviews on the topic [3]. Moreover, this review is limited by the quality of published evidence on RRD. While several prospective studies have reported the incidence of RRD, these have been reported without identifying the definition of RRD used or specific characteristics of the RRD [1,121,122]. Thus, this review focuses on RRD case reports given the number of these publications as well as the detail they provide. Another limitation is that skin toxicity was retrospectively graded based on the description and photos provided in the case report. However, one objective of this review was to attempt to provide a quantitative analysis of RRD given the lack of uniformity in the reported literature.

Conclusion

RRD is a complex process which manifests under heterogeneous conditions. Most commonly, it presents in patients diagnosed with breast cancer and after administration of a taxane or antimetabolite medication. RRD treatment generally consists of corticosteroids with consideration of antibiotics if superinfection is suspected. Drug re-challenge may be considered after RRD if the initial reaction was of mild intensity.

Conflicts of interest

None.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Authors' contributions

Bhangoo RS: conceptualization, data curation, formal analysis, methodology, validation, writing - original draft, writing - review and editing. Cheng TW: data curation, methodology, validation, writing - review and editing. Petersen MM and DeWees TA: formal analysis, methodology, and validation. Thorpe CS: methodology, validation, writing - review and editing. Anderson JD, Vargas CE, and Halyard MY: writing - review and editing. Patel SH: methodology, writing - review and editing. Schild SE: validation, writing - review and editing. Wong WW: conceptualization, formal analysis, methodology, writing - review and editing.

Acknowledgments

We would like to thank librarian Diana Almader-Douglas, MLS, AHIP for her assistance in developing the search strategy for this review.

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