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Incidence of chronic radiodermatitis after fluoroscopically-guided interventions (FGI): a retrospective study

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radiodermatitis, interventional radiology, percutaneous coronary intervention, neuroradiology

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Introduction

Skin injury secondary to ionizing radiation has been documented since the early twentieth century. Radiation promotes the local production of free radicals, which are toxic for DNA and the molecular components of cells, and the resulting cell apoptosis and necrosis induce skin inflammation. Lesions appear in the areas exposed to radiation. Clinical presentation depends mainly on the dose delivered. Within a few hours or days, doses below 6 Gy result in transient erythema, which is often unnoticed by both patients and doctors. Higher doses also induce longstanding or permanent lesions of varying appearance: hair loss, erythema, desquamation, telangiectasia, dermal sclerosis or skin atrophy and chronic ulcerations that can lead to skin carcinomas. Radiodermatitis often evolves in two successive phases, acute radiodermatitis during the first three months after irradiation, usually followed by total skin recovery or the persistence of a discrete pigmentation or skin atrophy, and many months or years later, chronic radiodermatitis that does not heal. Medical radiotherapy commonly induces radiodermatitis, but fluoroscopically guided interventions (FGI), although delivering lower doses, can also cause skin damage. Since the first reports of radiodermatitis following cardiac catheterization were published in the 1990s, (1,2) more than 200 cases due to FGI have been documented (see Appendix). The cutaneous lesions are clinically similar to those induced by radiotherapy, they but have two features that help to distinguish them, a well-defined geometric shape and their location, which is determined by the FGI. In most cases, the lesions are close to the radiation emitting tube, on the back, on the scapulae or axillae areas and on the scalp. These features are helpful in differentiating FGI-induced dermatitis from other localized skin diseases, such as morphea or mycosis.

Lesions induced by FGI can have serious cutaneous consequences even several years after the procedure and, in this event, the radiologist can be held liable. It would be beneficial, therefore, to have a better understanding of their prevalence and risk factors. However, it is likely that most cases of FGI- induced

57 radiodermatitis are undiagnosed. Unlike radiotherapy, which commonly induces dermatitis and about
58 which patients are informed, FGI is not usually accompanied by warnings about possible skin side
59 effects. Patients are generally unaware that they have acute FGI-induced dermatitis because the radiation
60 burn is not painful, and the initial skin lesions are too discrete to be noticed in the context of the urgency
61 or severity of the disease that required the initial intervention. In our hospital, in accordance with
62 national recommendations, (3) when a procedure exceeds the risk threshold for radiodermatitis, an e-
63 mail is sent to the radiologist indicating that a skin injury could appear in the coming weeks and that a
64 clinical assessment of the exposed skin areas should be made for several weeks. This procedure allows
65 detection of acute radiodermatitis but not of the chronic form. As chronic radiodermatitis progresses
66 slowly for many months or years after FGI, only one third of patients spontaneously notice their lesions,
67 usually when ulceration has appeared, and, at this late stage, they are unable to connect the lesions with
68 the procedure (see Appendix).

69 Some studies have estimated the prevalence of FGI radiodermatitis, but the results are limited either
70 because the authors focused on one particular form of dermatitis (i.e. skin ulcerations) or one kind of
71 FGI (mainly coronary explorations and angioplasties) or because the data were collected retrospectively
72 without the expertise of a dermatologist to clearly identify the lesions. According to these studies, FGI
73 radiodermatitis was observed in 0 to 1.5% of patients. (4-7) Given this wide diversity in reported
74 prevalence, a skin examination by a senior dermatologist was proposed to patients who had undergone
75 FGI procedures of any kind in the previous months or years to assess the prevalence of chronic
76 radiodermatitis and identify the risk factors for lesion onset.

77

78 **Materials and methods**

79 Institutional Review Board approval was obtained for this retrospective study. The Radiology Unit
80 database, which contains all the collected data of all the FGI procedures, was used to identify patients
81 who had had an FGI between January 2010 and December 2016 and for whom irradiation procedures
82 were considered at risk for radiodermatitis owing to the presence of at least one of the following criteria:
83 exposure of the skin to more than 3 Gy, air kerma greater than 5 Gy, DAP exceeding 500 Gray.cm²,
84 and/or fluoroscopy exposition time longer than 60 minutes. (3, 8) During this period, 55,782 FGIs were

85 conducted, 26,455 by cardiologists in the Cardiology Unit and 29,327 by interventional radiologists in
86 the Radiology Unit. Three hundred and ninety procedures (359 patients) were at risk for radiodermatitis.
87 Dosimetric information was provided by the device itself and periodically controlled by external audits.
88 When available, the peak skin dose was calculated either with radiologic films or with the dedicated
89 software em.doses. (9) A letter was sent to patients explaining the aim and design of the study and
90 proposing a skin examination by a dermatologist. Patients who did not answer within three months were
91 contacted by telephone. Those who accepted the offer to be examined gave their informed written
92 consent. The following data were recorded: age of the patient at the time of the procedure, sex, BMI,
93 comorbidities (diabetes, autoimmune disease, dysthyroidism), medical treatments taken at the time of
94 irradiation and skin phototype. Procedure characteristics were also recorded: date and aim of the
95 procedure, maximum skin dose, air kerma, DAP, fluoroscopy time, number of lifetime FGIs received
96 and, if applicable, therapeutic irradiations. All skin examinations were performed by the same senior
97 dermatologist with more than ten years' experience. Total skin examination was done with special
98 emphasis on the areas that were exposed to radiation to identify chronic radiodermatitis. If a lesion was
99 identified, pictures were taken, and the dermatology chart was revised by another senior dermatologist,
100 one who had graduated twenty-eight years ago. For each patient, a thorough medical history was taken
101 by the dermatologist, who tried to identify if there had been an occurrence of an acute dermatitis in the
102 days or months after the FGI. As a primary outcome, the incidence of radiodermatitis was calculated.
103 Risk factors for onset were comparatively assessed in populations with radiodermatitis and those without
104 (for statistical analysis: see Appendix).

105

106 **Results**

107 Among the 359 patients at risk for radiodermatitis, after exclusion of those who had died, who declined
108 to participate or who were lost to follow-up, 91 patients (96 procedures) were included in the study. The
109 skin examinations were performed in 2017 (Table 1). Median time from the procedure to clinical
110 examination was 31.8 months (95%_{CI}: [14.2; 50.7]). Eight patients had radiodermatitis (Table 2). The
111 incidence was 8.8% (95%_{CI}: [3.9; 16.6]) per patient and 8.3% (95%_{CI}: [3.7; 15.8]) per procedure. In
112 univariate analysis, BMI was the only factor that was statistically significant for a risk of dermatitis

113 (33.1 ± 4.1 kg/m² in patients with radiodermatitis and 28.68 ± 6 in patients without, p = 0.03). However,
114 this was not confirmed by multivariate analysis adjusted for DAP (OR = 1.29 [0.94; 1.77], p = 0.11).
115 None of the other patient characteristics were associated with a risk. The DAP value was a risk factor
116 for a skin lesion (median DAP value 1421 Gy.cm² [892; 1488] in patients with radiodermatitis *versus*
117 572 Gy.cm² [450; 794] in patients without (p < 0.001) as was air kerma (median air kerma = 4.3 Gy
118 [3.8; 5.3] *versus* 3.5 Gy [2.5; 4.9]; p = 0.03). Fluoroscopy duration was not a risk factor (37 [31; 59] vs.
119 45 [26; 68], p = 0.47). Exposition to a photosensitizing drug at the moment of irradiation did not appear
120 as a risk factor for chronic radiodermatitis (p = 0.95). None of the other parameters tested (age, sex, skin
121 phototype, diabetes, number of lifetime procedures) were significant. Of the 91 patients included, 19
122 reported transient erythema or alopecia in the days following the procedure; both manifestations were
123 considered as acute radiodermatitis. The incidence of acute dermatitis was 20.9% (95%_{CI}: [13.1; 30.7])
124 per patient and 21.6% (95%_{CI}: [13.9; 31.2]) per procedure. Only one of the eight patients with chronic
125 radiodermatitis experienced acute radiodermatitis.
126 Chronic lesions were a non-infiltrated and non-scleral patch a few centimetres in diameter that was
127 hypo- or, hyperpigmented (Figure a) or erythematous (Figure b) (in 2, 2, and 1 patients respectively), a
128 large sclerotic plaque without telangiectasia (1 patient), an atrophic plaque with telangiectasia (1 patient,
129 Figure e) and a sclerotic plaque with a large disabling and deep ulcer in another (Figures c and d). None
130 of these patients had a history of autoimmune disease, dysthyroidism or previous skin cancers. Only
131 three of them noticed the lesions and consulted a doctor: in two cases, the diagnosis of radiodermatitis
132 was made, but in one case, the lesions were mistaken for a fungal infection.

133

134 **Discussion**

135 The estimated prevalence of chronic radiodermatitis after FGI varies greatly from one study to another.
136 Vlietstra et al. (4) estimated a frequency of 0.01% by extrapolating from 76 previously reported
137 independent cases to a million coronary angiographies performed each year in the United States. The
138 study of Wei et al. (5) reported an incidence of 0.34%, but they only considered radiation-induced ulcers,
139 a clinical form that is infrequent, and included only patients who had had a coronary exploration. In
140 addition, none of the patients were examined by a dermatologist, and clinical data were collected from

141 clinical charts only. In a series of 61 patients who had undergone a complex procedure, Kirkwood et al.
142 (6) reported no instance of radiodermatitis, but patient follow-up was short, less than one year. In a study
143 involving 400 patients who had had a percutaneous coronary intervention for chronic total occlusion, a
144 procedure that exposes patient skin to high radiation doses, Kato et al. (7) observed six (1.5%) cases of
145 radiodermatitis. In the present study, the whole skin of 91 patients who were considered at risk of
146 radiodermatitis following an FGI performed between 14.2 and 57 months previously was examined by
147 a dermatologist to estimate the prevalence of skin reactions as the primary aim and to identify risk
148 factors.

149 Radiodermatitis was identified in 8.8% of the 91 patients. This unexpectedly high proportion is mainly
150 due to the patient population studied. As it was impossible to examine all the patients who had a
151 procedure during the recruitment period, only patients who were at risk for skin injury according to
152 international recommendations were selected. (3, 8) This inclusion criteria was consistent with that used
153 in previous publications. (6) In this series, the main risk factor for chronic radiodermatitis was related
154 to FGI characteristics and, therefore, it is likely that the frequency of chronic radiodermatitis would be
155 lower in the total population of patients who had had an FGI. This series is the first in which all of the
156 patients were fully examined by an experienced dermatologist. This could be a further explanation of
157 the high frequency observed since a skin specialist was able to detect discrete lesions or hidden lesions,
158 such as on the scalp or in the pelvic area. Finally, the median time from the FGI to the skin examination
159 (31.77 months (95%CI [14.9–50.73])) was longer than in most previous studies, which enabled us to
160 identify late-onset lesions. BMI was shown to be a risk factor ($p = 0.03$) but only on univariate analysis.
161 The risk exists because higher radiation doses are required to penetrate the body in overweight patients.
162 (10) Diabetes and fair skin, like skin-debilitating situations, were not estimated to be risk factors. Owing
163 to memory bias, it was not possible to correlate medication intake with the risk of developing
164 radiodermatitis. As reported elsewhere, fluoroscopy duration was not a risk factor unlike the air kerma
165 level ($p = 0.03$) and the DAP value ($p < 0.001$). The best way to assess skin exposure during FGI is to
166 calculate the peak skin dose, but this measurement is often difficult to obtain even with modern
167 equipment. and it is generally calculated. These data support the fact that, in particular for the overweight
168 patients, procedure safety could be improved by using lower doses, keeping the X-ray tube as far away

169 from the skin as possible, bringing the detector as close to the skin as possible, avoiding radiation field
170 overlaps and using the scopic rather than the graphic mode for procedure control.

171
172 One salient point to emerge from this study is the difficulty in identifying radiodermatitis. Often, it
173 appears clinically as visible erythema or as a pigmented non-palpable plaque. Even when the lesions are
174 typical, with sclerosis and telangiectasias caused by radio dystrophia, (11) they may be confused with
175 other sclerotic lesions, such as morphea, by an unaware clinician. However, morphea usually presents
176 as multiple lesions versus a single lesion like in radiodermatitis. In addition, radiodermatitis occurs in
177 specific areas corresponding to the zones exposed to radiation. (12,13) Little is known about the outcome
178 of radiodermatitis lesions. The probability of skin sclerosis, which is, with ulceration, the most disabling
179 feature of radiodermatitis, is not known, and this study provides no new evidence. However, the only
180 two patients in this series with a deep sclerosing lesion were those whose DAP levels were the highest
181 (2530 and 2095 Gy/cm²), unlike their BMI, exposure time and air kerma level. As ulcerative and
182 sclerosing lesions seem to be rare, it can be assumed that, in most cases, erythema or pigmented
183 radiodermatitis do not progress over time. To date, no cases of skin carcinoma following FGI have been
184 reported (see Appendix). However, the possibility that many basal cell carcinomas removed by
185 dermatologists unfamiliar with radiodermatitis were due to the radiation procedure cannot be excluded.
186 Due to these difficulties in clinically identifying chronic radiodermatitis, patients should be clearly
187 informed of the occurrence of adverse outcomes and offered a bi-annual lifetime dermatology
188 consultation whenever the X-ray exposure threshold is exceeded.

189 Therapeutic management is not established. Excision of the lesion at the early stage of sclerosis, before
190 massive sclerosis and ulceration, should be recommended. Surgical reconstruction in sclerotic
191 conditions is difficult whereas fat transfer can help wound healing. (14,15)

192 The relationship between acute and chronic radiodermatitis is unclear. Acute radiodermatitis onset did
193 not seem to be a risk factor as only one of the eight patients concerned with chronic
194 radiodermatitis reported an inflammatory lesion just after the procedure, whereas its rate of incidence
195 in the total population examined was 21%. Acute dermatitis could have a protective role in the
196 occurrence of chronic dermatitis, but the limited size of the population makes statistical analysis

197 impossible. The biological mechanisms of acute and chronic radiodermatitis are different. Acute
198 lesions result from cell destruction directly induced by radiation, whereas chronic lesions are due to
199 the chronic production of TGF β , which stimulates fibroblasts and neovascularization. (16) As there is
200 no evidence that acute irradiation results in a durable production of TGF β , “acute and chronic
201 radiodermatitis” should be renamed as “early- and late-onset radiodermatitis”, respectively.
202 This study has some limitations. Only 25% of the patients at risk were examined either because they
203 were lost to follow-up, declined to participate or had died. However, patient characteristics (sex, age)
204 and irradiation parameters (total dose, air kerma level, DAP value, fluoroscopy time) in patients at risk
205 who were examined and those who were not were compared. No differences were found (data not
206 shown), which led us to assume that the 8.8% rate observed was the same in the unexamined at-risk
207 patients. On the basis of the inclusion criteria, patients who had undergone several FGIs all under the
208 risk threshold for skin injury were excluded from the study. This bias could have underestimated the
209 incidence of radiodermatitis. (17) Also, it cannot be excluded that patients with a discrete and, thus,
210 unknown chronic radiodermatitis, with no other dermatoses since their FGI did not feel concerned by
211 the study when receiving our information and did not participate. This bias, if it existed, may have
212 lowered the observed incidence of radiodermatitis. Owing to the retrospective nature of the study, many
213 patient risk factors, such as tobacco abuse, inflammatory or nutritional status and sun exposure level at
214 the time of FGI were unable to be taken into account and assessed. Only a prospective study could
215 eliminate these limitations. In contrast, the fact that the patients were examined by a dermatologist and
216 had their case reviewed by a second dermatologist lends strength to the findings of the study. First, a
217 thorough history-taking made by a skin specialist avoided the cohort from being enriched by patients
218 who had experienced dermatoses of any kind after FGI. Second, a dermatologist is able to detect even
219 discrete lesions. Last, although memory bias is undeniable in recording acute dermatitis, its effect is
220 reduced when a dermatologist who is aware of the different forms of radiodermatitis performs a detailed
221 history record. In any case, if such a recall bias existed, it did not impact the evaluation of late-onset
222 radiodermatitis frequency.

223

224 In conclusion, this study identified a late-onset radiodermatitis after FGI procedures in 8.8% of cases,
225 which is the highest rate reported to date mainly because patients were systematically examined by a
226 skin specialist. Irradiation parameters (air Kerma level, DAP value) and patients' BMI were the only
227 risk factors identified. Several questions remain unanswered, including other risk factors due to patient
228 characteristics, the long-term outcome of sclerotic lesions, their treatment and the risk of skin cancer.

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275

276

277 **Legends**

278

279 Table 1: Patients' characteristics.

280

281 **Phototypes:**

282 **1**: **Hair**: Red/blonde. **Eyes**: blue, grey, green. **Skin**: Very pale white, pale white. **Tanning ability**:
283 burns very easily, never tans.

284 **2**: **Hair**: Red/blonde/light brown. **Eyes**: Blue, grey, green, hazel. **Skin**: Pale white. **Tanning ability**:
285 burns easily, rarely tans.

286 **3**: **Hair**: Chestnut, dark blonde. **Eyes**: Brown, blue, grey, green, hazel. **Skin**: White, light brown.
287 **Tanning ability**: Sometimes burns, gradually tans.

288 **4**: **Hair**: Brown, medium brown, dark brown. **Eyes**: Hazel, brown. **Skin**: Medium brown, dark brown.
289 **Tanning ability**: Hardly ever burns, tans very easily.

290 **5**: **Hair**: Dark brown. **Eyes**: Brown. **Skin**: Dark brown. **Tanning ability**: Rarely burns and quickly
291 darkens.

292 **6**: **Hair**: Black. **Eyes**: Brown. **Skin**: Black. **Tanning ability**: Never burns, tans very dark.

293

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296

297 Table 2: Patients with chronic radiodermatitis.

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300

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304 Figure: Clinical aspects of cutaneous lesions

- 305 a. Hyperpigmented patch on the left side of the abdomen after hepatocarcinoma
306 chemoembolization. (Patient 4 table 2)
- 307 b. Erythematous plaque after cerebral aneurysm embolization. (Patient 6 Table 2)
- 308 c. Sclerous plaque on the abdomen after mesenteric artery angioplasty. (Patient 2 Table 2)
- 309 d. Ulcer with sclerous borders on the abdomen after mesenteric artery angioplasty. (Patient 2
310 Table 2)
- 311 e. Atrophic plaque with telangiectasias on the right axillary fold after pulmonary arteriovenous
312 malformation embolization. (Patient 7 Table 2)
- 313 f. HES x 100, skin biopsy of a chronic radiodermatitis: dermal fibrosis, atypical stellar
314 fibroblasts (Patient 7 Table 2)

APPENDIX 1: Statistical analyses

Risk factors for radiodermatitis onset were assessed in populations with radiodermatitis and those without by the chi2 test in univariate analysis and the log rank test in multivariate analysis. Patient characteristics and radiation exposure data were compared in patients with radiodermatitis and those without. Analyses were performed with Stata software (Version 13, StataCorp, College Station, TX) for a two-sided Type I error at 5%. Patient characteristics were expressed as mean \pm standard deviation (SD) or median [interquartile range] for continuous data (assumption of normality assessed by the Shapiro-Wilk test) and as numbers and associated percentages for categorical parameters. When appropriate, the results were expressed with a 95% confidence interval. The continuous variables were compared in two groups of patients (with and without radiodermatitis) by Student's t-test or the Mann-Whitney test if the assumptions of the t-test were not met ([i] normality and [ii] homoscedasticity as assessed by the Fisher-Snedecor test). Categorical data comparisons were performed by the chi-squared or Fischer's exact tests. For repeated data, analyses were carried out with random-effect models to take into account inter- and intra-patient variability (patient as random effect). Multivariable analyses were performed with adjustment for DAP. Results were expressed as odds-ratio (OR) and 95% confidence interval.

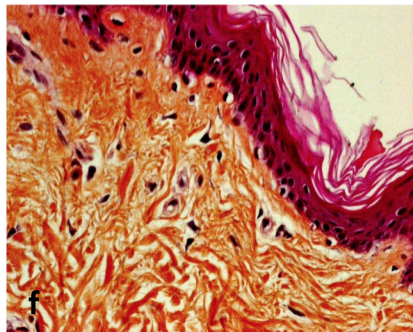
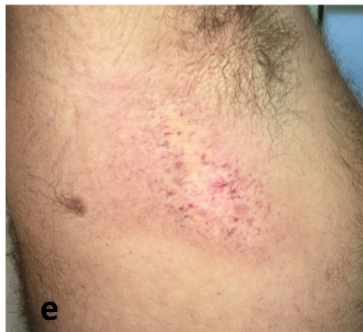


Table 1. Patient's characteristics

Gender	35 F / 56 M
Age (years), median [IQR]	63.4 [53.7 ; 72.9]
BMI (kg/m²), median [IQR]	28.2 [24.7 ; 32.8]
Phototype	
2	5
3	39
4	40
5	3
6	2
Diabetes	13/91
Cutaneous cancer history	5 / 91 (basal cell carcinoma, melanoma)
Previous exposition in the same skin area	18/91
Procedure type	
1. Endovascular cephalic interventional neuroradiology	25
2. Cardiac procedure (PTCA, interventional rythmology)	3
3. Vascular peripheral interventional radiology (supra-aortic trunks, thoraco-abdomino-pelvic and limbs vessels)	68
Renal artery	14
Chemoembolization	9
Aorta (EVAR and other procedures)	6
Mesenteric artery	5
Splenic artery	5
Pulmonary embolization	5
Post-partum hemorrhage	4
Digestive embolization	3
Hepatic artery	3
Iliac angioplasty	2
Hypogastric embolization	2
Duodenopancreatic aneurism	2
Pelvic embolization	2
Portal embolization	1
TIPS	1
Epistaxis	1
Gluteal artery revascularization	1
Carotid	1
Muscular hematoma embolization	1
Fluoroscopy Time (min), median [IQR]	45 [28 ; 67]
DAP (Gy.cm²), median [IQR]	603 [470 ; 878]
Air Kerma (Gy), median [IQR]	3.75 [2.64 ; 5.01]
Median follow-up (months), median [IQR]	31.8 [14.2 ; 50.7]

Table 2. Eight patients with chronic radiodermatitis.

PATIENT						PROCEDURE				SEMIOLOGY			
N°	Sex	Age	BMI	Phototype	Medical history	Type	F. time (min)	DAP (Gy.cm ²)	AK (Gy)	Acute RD	localization	semiology	time to onset
1	F	44	37.8	3		uterine arteries embolization (postpartum hemorrhage)	31	2 530	4.65		lumbar	Large sclerotic area	3 months
2	F	81	30.8	3	Diabetes, Photosensitizing treatment	Mesenteric artery angioplasty	61	2 095	11		left flank	Ulcer with sclerotic banks	5 months
3	M	80	27.8	4		hypogastric artery embolization + aortic aneurysm prosthesis	36	974	2.69		lumbar	Hypopigmented patch	Unknown
4	M	67	31.4	3		hepato carcinoma chemoembolization 1	8	319	0.68				
					Previous procedure	hepato carcinoma chemoembolization 2	13	740	2.39		upper right quadrant	Hyperpigmented lesion	Unknown
5	M	49	31.7	4	Photosensitizing treatment	interventional cardiology	40	1427	4.29		right sub-scapularis	Hypopigmented patch	Unknown
6	M	70	32.8	4		neuro-radiology : cerebral aneurysm embolization	73	386	3.94		occipital	Erythematous plaque	Unknown
7	M	45	39.2	3	Photosensitizing treatment	pulmonary arteriovenous malformation embolization 1					x right axillary fold	Atrophic plaque, numerous telangiectases	2 weeks (after embolization 1)
					Previous procedure for embolization 2	embolization 2	59	1487	10.24				
							37	1420	5.29				

8	M	71	38.3	4	Diabetes, Photosensitizing treatment	Abdominal muscular bleeding embolization	31	892	3.75	left abdominal	hyperpigmented lesion	Unknown
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