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Correlation between tumor growth and hormonal therapy with MR signal characteristics of desmoid-type fibromatosis: A preliminary study

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KEYWORDS
Desmoid type fibromatosis;
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Abstract
Purpose: To evaluate the impact of hormonal therapy on MRI characteristics of desmoid-type fibromatosis on T1-weighted, T2-weighted fat-saturated and post-contrast sequences.
Materials and methods: Nineteen patients with histologically-proven desmoid-type fibromatosis were prospectively followed up on MR imaging. Eight patients underwent hormonal therapy and 11 were only surveyed. Change in tumor size during follow-up was analyzed according to RECIST. Signal intensity on T1-weighted, T2-weighted fat-saturated and T1-weighted fat-saturated post-contrast images was graded from 0 to 5 using adjacent normal muscle as reference. Findings were compared with tumor growth and treatment option.
Results: There were seven men and 12 women with a mean age of 42.2 ± 16.4 (SD) years (range: 18 - 64 years) yielding twenty-six follow-up periods: eight of tumor progression and 18 of tumor stability/regression (some tumors exhibited more than one behavior type). Hormonal therapy was associated with tumor stability or regression ($P = 0.0207$). There was a significant reduction in enhancement among treated patients with stable/regressing disease ($P = 0.049$). The mean variation in enhancement grade was -1.3 ± 1.2 in these patients. All successfully treated patients presented a reduction in enhancement. Lesions with marked low enhancement or very low signal on T2-weighted images were rare in progressing lesions (0% and 13%).

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Conclusion: Hormonal therapy has an impact on desmoid-type fibromatosis signal characteristics reducing lesion enhancement.

Desmoid-type fibromatosis (DF), also known as aggressive or deep fibromatosis, are benign soft tissue tumors that account for 0.03% of all tumors [1]. They have no metastatic potential but can be locally aggressive, with an average recurrence rate of 24% to 77% [2]. Wide local excision (when R0 resection is feasible) or surveillance is the standard care for aggressive fibromatosis [3]. When complete surgical lesion removal is feasible (R0 margins), the prognosis is excellent [4]. When R0 margin excision is not obtainable or when the lesion compromises major vascular and nervous structures surgical treatment is not recommended due to high recurrence rates, high risk surgery and potential functional disability. In such cases, a wait-and-see therapeutic strategy is advised [5,6]. There is no consensus on non-surgical treatment for DFs. Observation, non-steroidal anti-inflammatory drugs, radiotherapy, chemotherapy (both standard and targeted), percutaneous ablative procedures and hormonal therapy can be used alone or in combination [7]. Magnetic resonance-guided focused ultrasound has also been reported to reduce viable tumor volume and pain [8]. Hormonal therapy is less toxicity than cytotoxic agents and can be considered first-line therapy for DFs [9].

Magnetic resonance (MR) is the imaging modality of choice for the follow-up of patients with DFs, particularly those treated conservatively, for whom imaging has a direct impact on management [10]. Although signal intensity on T2-weighted images has been reported to correlate with tumor collagen content and cellularity, only size criteria are currently used for the follow-up of DF [11—13]. Previous studies have failed to correlate signal changes with clinical progression and currently there are no defined criteria for the evaluation of non-surgical treatment response [14,15]. However, Gounder et al. have reported signal changes on T2-weighted images in DF in a series of 13 patients treated with a kinase directed therapy [16]. Hormonal therapy may also affect the histologic characteristics of DF and hence the MR appearance of these tumors.

The aim of this study was to evaluate the impact of hormonal therapy on MRI characteristics of desmoid-type fibromatosis on T1-weighted, T2-weighted fat-saturated and post-contrast MR sequences.

Materials and methods

Patients

From October 2009 to April 2014, 28 patients with histologically-proven sporadic DF were prospectively included in a tumor characterization research protocol. Histopathologic analysis was performed by a panel of experts and registered in the French national sarcoma database (Réseau expert pour la pathologie sarcome — RePPS, France). This study was approved by the local ethics committee and all patients signed an informed consent. All patients were 18 years-old or over and were examined by a physician before imaging. Patients with confirmed or suspected pregnancy, MR imaging contraindications and severe renal insufficiency were not included. Six of the included patients had been treated surgically and presented with recurrent tumor. Of these patients, only 19 underwent at least two MR studies (baseline and follow-up) with similar protocols. All of these patients had at least a three-month imaging follow-up. The images from these patients were retrospectively evaluated. One patient had two concurrent tumors, in two different anatomic locations, which were considered independently. Hence, in total, 20 DF lesions were included in the study.

Eight patients were treated with 20 mg per os per day of tamoxifen (Nolvadex® AstraZeneca, London, UK) and 11 received no treatment during follow-up. One included patient was treated with both tamoxifen and radiotherapy. In accordance with the work of Kasper et al., in our institution, hormonal therapy is the first line of therapy for DF and is proposed, after patient information, for growing lesions during follow-up when no contraindications to hormonal therapy are present [3]. Treatment was considered successful if lesion size remained stable or regressed during follow-up. Individual patient treatment was decided by the national sarcoma multidisciplinary tumor board on a case-by-case basis. Tumor size was considered as the standard evaluation criteria for treatment response.

A hundred and thirty-five MR studies were performed on the patients enrolled. In accordance with National Comprehensive Cancer Network (NCCN) Guidelines for Soft Tissue Sarcomas, patients with stable or regressing DF were followed up on MR every 6 months for two years and then, if lesion size remained stable, they were followed-up yearly. Progressing DF was imaged every 3 months until progression stopped or surgical treatment was implemented [17].

The first and the last available MR studies of each period of tumor behavior (size variation) were evaluated. For each behavior period, patient records were analyzed to ascertain if the patient was or was not under hormonal treatment at that time.

Imaging protocol

MR imaging was performed at 1.5T (SignaHDxt®, GE Healthcare, Milwaukee, WI, USA) using dedicated coils. The

imaging protocol included at least one T1-weighted fast spin echo sequence, twoT2 fat-saturated FSE sequences in two different orthogonal planes and one contrast-enhanced T1-weighted FSE fat-saturated sequence obtained 5 minutes after the injection. A 0.2 mL/kg dose of a gadolinium-based contrast material (Multihance®, Bracco Diagnostics) was injected in a peripheral vein at a rate of 0.5 mL/s using an injection pump (Spectris Solaris EP, Medrad Inc, Indianola, PA, USA).

Acquisition parameters were adapted to the anatomy at the tumor location. FOV, slice thickness and gap varied from 200 to 440 mm, 3.5 to 5 mm and 0.5 to 3 mm, respectively. The matrix size varied from 224 × 256 to 416 × 352. The following parameters were used for T1-weighted sequences: repetition time (TR), 200—600 ms; echo time (TE) 2—17 ms; number of excitations (NEX) 1—4; bandwidth 13 to 50 kHz; Echo train length (ETL) 2 to 4. Acquisition parameters for T2-weighted sequences were: TR, 3500—10000 ms; TE, 48—77 ms; NEX, 1—4; bandwidth, 13 to 42 kHz; and ETL, 10 to 23.

Image analysis and data post-processing

Two radiologists with four and eight years of clinical experience in musculoskeletal MR imaging reviewed all images in consensus. The readers were blind to patient treatment. Conventional MR images were evaluated on a picture archiving and communication system (PACS) workstation (Impax V5, AGFA HealthCare, Ivry-sur-Seine, France).

Each lesion was assessed for variation in size over time using classic unidirectional response evaluation criteria in solid tumors (RECIST) [3,18,19]. The greatest diameter of each lesion was identified by analysis T2-weighed fat-saturated images in two orthogonal planes. The image depicting the largest tumor diameter was used for measurement. Peri tumoral edema was not considered as part of the tumor diameter. The largest tumor diameter was measured in millimeters at both the baseline and follow-up studies during the same readout session. Tumors that decreased in diameter by 30% or more were considered to be in regression; tumors that increased in diameter by 20% or more were considered to be progressing; otherwise, the lesion was considered stable. Tumors were then divided into two groups according to their growth (behavior):

- stable and regression (stable/regression group);
- progression (progression group).

If during follow-up patients exhibited different types of behavior (progression and stability/regression), the periods concerned were evaluated separately. Imaging findings were correlated with changes in tumor size.

The signal intensity of each lesion on T1-weighted, T2-weighted fat-saturated and T1-weighted fat-saturated post-contrast images was graded using adjacent normal muscle as reference as follows [14]:

- complete signal hypointensity (grade 0);
- diffuse hypointensity with scant areas of signal hyperintensity (grade 1);
- heterogenous lesion more than 50% hypointense (grade 2);
- heterogenous lesion less than 50% hypointense (grade 3);
- almost complete signal hyperintensity (grade 4);
- complete signal hyperintensity (grade 5).

All slices containing tumor were considered in the evaluation.

Statistical analysis

Statistical analysis was performed using R software version 3.01 [20]. Continuous data were reported as mean ± standard deviation (SD). Cox regression analyses were performed for all imaging variables to determine their relationship with patient treatment and size change during follow-up. The imaging variables were the signal intensity variation on T1- and T2-weighted images as well as the enhancement variation between both examinations. The association of tumor behavior with patient treatment and imaging parameters (T1, T2 and enhancement variation) was assessed by using multivariate Cox proportional hazards model. The best model was chosen after a backward and forward stepwise algorithm application. The Akaike Information Criterion was used. Kaplan—Meyer estimates of survival curves were calculated and differences were tested using the log-rank test with G-rho family tests. A P value < 0.05 was considered significant.

Results

The study population comprised seven men and 12 women with a mean age of 42.2 ± [16.4] years (range: 18—64 years). The mean follow-up time was 28.2 ± [11.5] months (range: 4—99 months). Lesions were located in the upper extremity (n = 7), lower extremity (n = 8), abdomen/chest (n = 2), and neck (n = 3). The mean lesion size was 92 ± [42] mm (range: 27 to 198 mm). Among the 19 patients included 12 had b-catenin S54F mutation verified. Of these patients, 10 were positive for b-catenin mutation and two were negative. Of the seven patients in which b-catenin mutation research was not available, five were diagnosed before 2007. For the other two patients, immunohistochemistry analysis was consistent with DF but b-catenin mutation search was not performed. None of the patients included had known history of familial adenomatous polyposis or Gardner syndrome.

Of the 20 DF lesions evaluated, six exhibited two different types of behavior during follow-up, thus 26 behavior periods were available for evaluation: 16 of tumor stability and 2 of tumor regression with a mean size reduction of 10 ± [21]% (range: 19—61%); and 8 of tumor progression with a mean increase of 33.6 ± [15.5]% (range: 20—61%). The mean age in the stability/regression group was 43 ± 16.5 (SD) years and that in the progression group was 38.9 ± 16 (SD) years. Follow-up durations in the two groups were 19.9 ± 19.4 (SD) months and 10.6 ± 4.7 (SD) months, respectively. Spontaneous regression size was seen in one of the lesions studied. This lesion showed no changes in signal intensity or enhancement during the follow-up period. In patients with two behavior periods, five had progressing tumors that later became stable or started to regress and one had a stable tumor that started progressing. Table 1 demonstrates the general characteristics of the population studied.

Table 1 General characteristics of the DF behavioral periods studied. In patients with two behavior periods the latter are presented in chronologic order.

Patient number	β-Catenin mutation	Type	Age	Location	Behavior periods per patient[b]	Growth behavior	Treatment	Size variation (%)	Follow-up (months)
1[a]	NA	Recurrence	53	Upper limb	1	Regression	Surveillance	−51	20
1[a]	NA	Recurrence	53	Upper limb	1	Stable	Surveillance	−22	20
2	Negative	De novo	52	Lower limb	1	Stable	Surveillance	2	3
3	Negative	De novo	62	Lower limb	1	Stable	Surveillance	4	11
4	NA	De novo	19	Lower limb	1	Stable	Surveillance	−5	5
5	Positive	De novo	36	Upper limb	1	Stable	Surveillance	1	10
6	Positive	De novo	19	Lower limb	1	Stable	Surveillance	−6	5
7	NA	De novo	51	Neck	1	Stable	Surveillance	−2	7
8	Positive	De novo	60	Thorax	1	Stable	Surveillance	1	5
9	Positive	Recurrence	64	Upper limb	1	Stable	Surveillance	19	3
10	Positive	De novo	34	Lower limb	1	Stable	HT	11	61
11	Positive	De novo	27	Lower limb	1	Stable	HT	−3	18
12	Positive	De novo	28	Upper limb	1	Progression	Surveillance	22	7
13	Positive	Recurrence	29	Lower limb	1	Progression	HT	88	14
14	NA	Recurrence	18	Lower limb	2	Progression	Surveillance	21	20
						Stable	Surveillance	3	29
15	NA	Recurrence	32	Upper limb	2	Progression	Surveillance	20	11
						Stable	HT	−12	61
16	NA	Recurrence	43	Neck	2	Progression	Surveillance	61	12
						Regression	HT	−31	41
17	Positive	De novo	61	Upper limb	2	Progression	HT	38	7
						Stable	HT	−28	17
18	Positive	De novo	63	Neck	2	Stable	HT	1	6
						Progression	HT	49	8
19	NA	De novo	37	Abdomen	2	Progression	HT	20	6
						Regression	HT	−61	37

HT: Hormonal therapy.
[a] Patient with two distinct lesions.
[b] In patients with multiple behavior periods, the latter are presented in chronologic order.

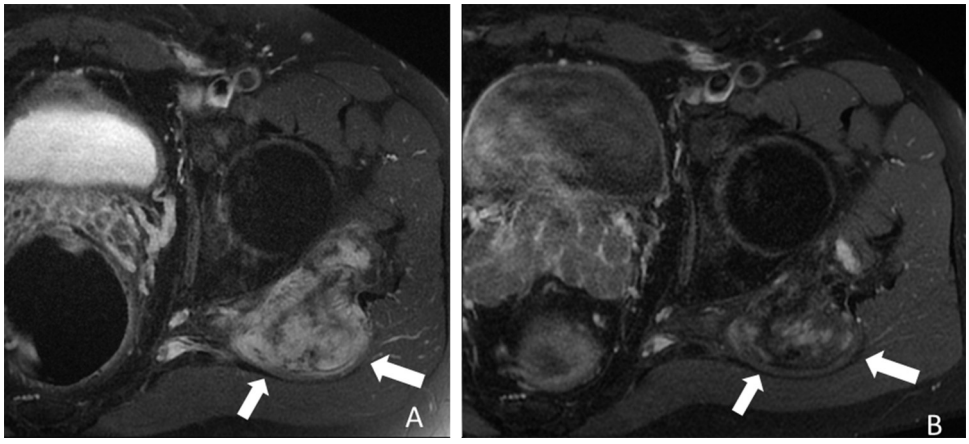

Figure 1.. A 34-year-old man with hip desmoid-type fibromatosis (DF) undergoing hormonal therapy. A. Post-contrast T1-weighted fat-saturated MR image in the transverse plane demonstrates a DF of the left gluteal region centered on the gluteus medius muscle (arrows). The lesion is less than 50% hypointense relative to adjacent muscle (grade 3). B. Post-contrast T1-weighted fatsaturated MR image in the transverse plane of the same patient after 22 months on the same. Anatomic region. Note the marked reduction in enhancement of the lesion (arrows) that shows diffuse hypointensity with scant enhancing areas (grade 1).

Impact of treatment on tumor growth

Tamoxifen was used in 11/26 periods evaluated. In the progression group, 4/8 lesions (50%) were treated and 4/8 (50%) were not, whereas in the stability/regression group 7/18 (39%) were treated and 11/18 were not (61%). Lesions under treatment were more likely to be stable or to decrease than lesions under surveillance ($P = 0.0207$). Among the 8 patients treated with tamoxifen, therapy was successful in 6 (75% success rate). The hazard ratio of hormonal therapy compared to surveillance was 0.9 for tumor progression.

Impact of treatment on tumor enhancement variation

The best model to explain tumor behavior was obtained with treatment and enhancement variation. T1 and T2 parameters were not retained during stepwise regression procedure. All tumors considered, the mean enhancement grades in post-contrast images of the baseline and follow-up studies were 3.6 ± 0.6 (SD) and 3.1 ± 1.1 (SD).

In stability/regression group, mean enhancement grades in the baseline and follow-up studies were 3.4 ± 0.7 (SD) (range: 2—4) and $2.1 \pm [1.6]$ (range: 0—5) respectively. In this group, patients under treatment exhibited greater variation in contrast enhancement than untreated patients (Fig. 1). The mean variations in enhancement grade were $-0.2 \pm [0.6]$ (range: -2 to 0) in untreated patients and -1.3 ± 1.2 (SD) (range: -3 to 1) in those under treatment. In this group, 1/10 patients (10%) without treatment demonstrated decreased enhancement, compared with 6/7 patients (86%) under treatment. These differences were statistically significant ($P = 0.049$).

Enhancement variation was minimal between treated and untreated patients in the progression group. Mean enhancement grades in the baseline and follow-up studies were $3.7 \pm [0.4]$ (range: three to four) and $3.3 \pm [0.5]$ (range: 3—4) in this group. The mean variations in contrast enhancement grade were $-0.5 \pm [0.5]$ (range: -1 to 0) in patients without treatment and -0.25 ± 0.9 (SD) (range: -1 to 0) in patients under treatment. Among progressing tumors treated with tamoxifen, only one in four demonstrated a reduction in enhancement. This lesion became stable after 8 months of therapy. All patients treated successfully presented a reduction in enhancement at some point of follow-up.

A high grade of enhancement (grade 3 or higher) on the baseline study was noted in 8/8 (100%) lesions in the progression group and 16/18 lesions (89%) of the stability/regression group; 33% of lesions (six out of 18) in the stability/regression group presented low enhancement graded from 0 to 2 during the follow-up, compared with none of the progression group (Fig. 2). One lesion in the stability/regression group showed an increase in enhancement of one grade in the follow up-study. Fig. 3 shows the distribution of enhancement variation with respect to tumor behavior and treatment option.

Impact of treatment on tumor signal variation

The means and standard deviations of signal on T1- and T2-weighted images as well as enhancement in the periods studied are presented in Table 2. The variation of lesion signal on T1- and T2-weighted images showed a similar tendency to that of enhancement variation. Compared to the latter, there was a higher decrease in signal grade from the baseline to follow-up study is patients undergoing hormonal therapy in the stability/regression groups (Table 2). As with enhancement, little change in signal on T1 or T2-weighted images was seen in the progression group regardless of treatment. Nine out of 18 lesions (50%) in the stability/regression group presented a markedly low signal on T2-weighted images (grades 0 to 2) during the follow-up, compared to 1/8 lesion (13%) in the progression group (Fig. 4). The

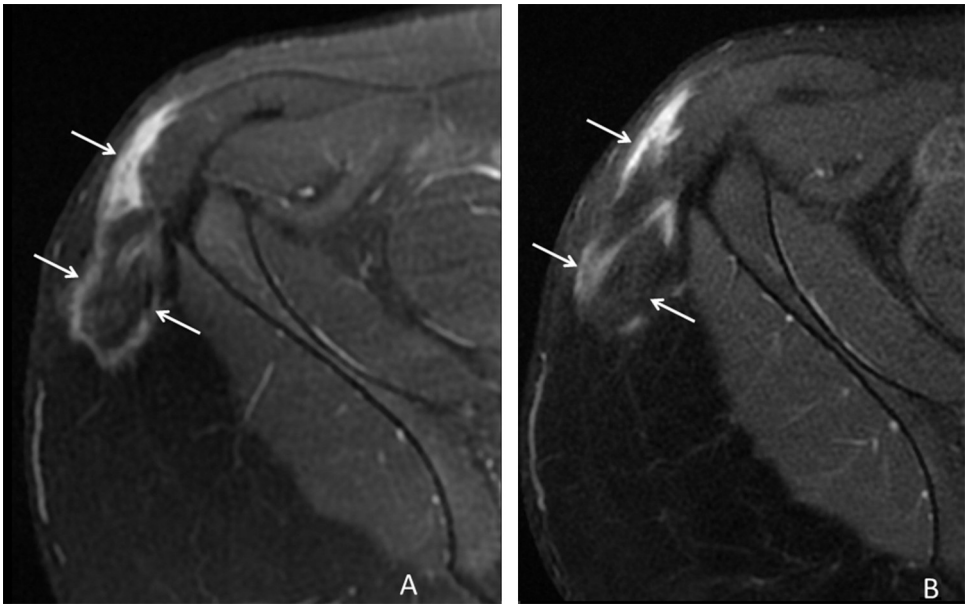

Figure 2. A. 19-year-old woman with desmoid-type fibromatosis (DF) of the right hip undergoing surveillance. A. Post-contrast T1-weighted fat-saturated MR image in the transverse plane demonstrates a heterogeneously enhancing DF adjacent to the antero-inferior iliac spine (arrows). Compared to the adjacent muscle the lesion is more than 50% hypointense (grade 2). B. Post-contrast T1-weighted fat-saturated MR image in the transverse plane of the same patient 18 months later on the same anatomic region demonstrating the same enhancement pattern seen on the baseline study despite of the slight decrease in enhancement.

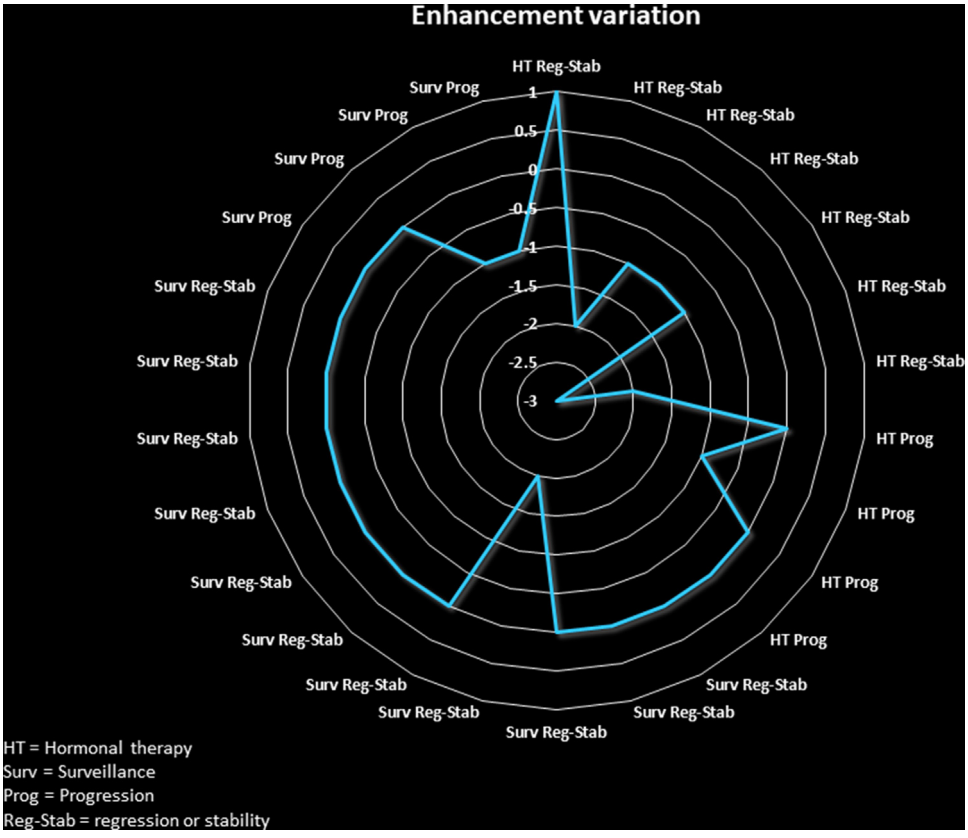

Figure 3. Spider plot demonstrating the enhancement variation in the behavior periods evaluated with respect to tumor behavior and treatment option.

Table 2 Variation of signal intensity grades of desmoid type lesion with respect to tumor behavior and treatment.

	Stability/regression group		Progression group	
	Tamoxifen	Surveillance	Tamoxifen	Surveillance
T1				
Base	2.2 ± 0.9 [1–3]	2.1 ± 0.6 [1–3]	3.0 ± 0.0 [3–3]	2.5 ± 0.5 [2–3]
Follow-up	1.2 ± 1.2 [0–3]	2 ± 0.6 [1–3]	3.0 ± 0.0 [3–3]	2.5 ± 0.5 [2–3]
Variation (%)	45.5	4.8	0.0	0.0
T2				
Base	2.7 ± 1.2 [0–4]	3.3 ± 0.6 [2–4]	3.5 ± 1.0 [3–4]	3.2 ± 0.5 [3–4]
Follow-up	1.1 ± 1.4 [0–4]	3.1 ± 0.8 [2–4]	3.2 ± 1.3 [2–4]	3.0 ± 0.0 [3–3]
Variation (%)	59.3	6.1	8.6	6.3
GD				
Base	3.4 ± 0.7 [2–4]	3.6 ± 0.6 [2–4]	3.7 ± 1.0 [3–4]	3.7 ± 0.5 [3–4]
Follow-up	2.1 ± 1.6 [0–5]	3.4 ± 0.8 [2–4]	3.5 ± 1.2 [3–4]	3.2 ± 0.5 [3–4]
Variation (%)	38.2	5.6	5.4	13.5

Numbers in brackets are ranges.

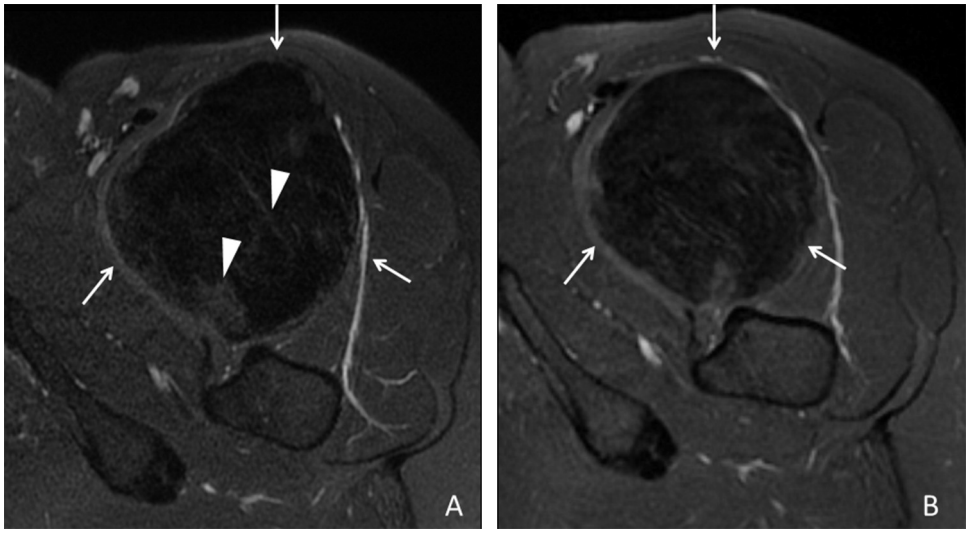

Figure 4. A. 27-year-old man with desmoid-type fibromatosis (DF) of the proximal left thigh undergoing surveillance. A. T2-weighted fat-saturated MR image in the transverse plane demonstrates a diffusely hypointense lesion with well-defined contours located in the anterior thigh compartment (arrows). Scant areas of high signal intensity are seen in the lesion (arrowheads), which was graded 1. B. T2-weighted fat-saturated MR image in the transverse plane of the same patient 4 years later at the same anatomic region. The lesion (arrows) has slightly decreased in size (3% reduction in the greatest diameter) but kept the same signal characteristics.

differences were not significant ($P = 1$ and 0.860 for signal variation on T1- and T2-weighted images, respectively).

Discussion

Signal on T1-, T2-weighted images and degrees of enhancement varied in a similar manner in the population studied and a reduction in lesion enhancement over time was seen significantly more often in treated patients with stable or regressing tumors ($P = 0.049$). In stable or regressing lesions, a mean reduction of 38.2% in tumor enhancement grade could be found in treated lesions compared to a 5.6% reduction in untreated ones. Secondarily, hormonal therapy was associated with tumor stability or regression in 75% of cases, all of which showed a reduction in lesion enhancement during follow-up. Among progressing tumors, there was no significant variation in enhancement between treated and untreated patients. These results indicate that a reduction in enhancement may be indirectly associated a positive response to hormonal therapy but further studies are needed to confirm this assumption.

Anti-hormonal agents such as tamoxifen alone or in association with non-steroidal anti-inflammatory drugs are considered as the first line of medical therapy for patients with DF [3]. The response rates of such treatment have varied in the different reports and might be influenced by the presence of B-catenin mutation or in the context of familial adenomatoid polyposis [3,19,21]. The presented findings differ from those reported by Castellazzi et al., who identified

no correlation between MR characteristics and the behavior of DF with or without treatment in 27 patients [14]. These authors included patients from 1985 to 2005, which certainly implies variable acquisition protocols and heterogeneous image quality. In the present study, all patients included were evaluated in the same MR unit with dedicated coils, which implies higher image quality compared to the earlier work. These results are also discordant with the unpublished report by Mitra et al. that have demonstrated a poor correlation between lesion size and signal on T2 weighted images [21]. These authors however did not consider contrast enhancement and it is not clear if they analyzed patients with and without hormonal therapy separately. Finally, the presented findings are in accordance with previous literature reports including the work of Gounder et al. and Sheth et al. that have reported a signal decrease on T2-weighted images in patients with a positive response to systemic therapy [15,16,22]. Further studies in larger patient populations, remain necessary as these differences could also be related to the small number of patients evaluated or spontaneous regression here as well as in previous literature reports.

High signal intensity on T2-weighted images and high enhancement grades were very frequent in the baseline studies of DF regardless of clinical behavior or therapy, making them of little use for patient follow-up. However, in the stability/regression group, lesions more than 50% hypointense on T2- and T1 post-contrast sequences with respect to normal muscle were moderately frequent (50% and 33.3% respectively). This finding was rare in progressing lesions (13 and 0% for signal on T2-weighted images and contrast enhancement). These results are in accord with literature data and can help optimize imaging follow-up intervals and plan therapy [14,23]. Size is the most commonly used criterion for oncologic imaging follow-up [24,25]. Size criteria are, however, not applicable or insufficient in a significant number of lesions that are too small or show little variation in dimension [26,27]. Contrast enhancement has been used as an additional criterion for follow-up of various tumor types [28,29]. As the variation in behavior of DF is not always reflected by changes in tumor size, the analysis of signal and enhancement could improve the performance of imaging in follow-up and treatment response assessment.

Myoblast proliferation, differentiation, migration, angiogenesis and extracellular matrix synthesis in fibromatous processes are regulated by various cytokines including transforming growth factor-β (TGF-β) and cyclooxygenase-2 COX2 [30]. These cytokines might be important for DF growth [31]. Sex-hormone receptors appear to modulate TGF-β and COX2 signaling [30]. Hormonal therapy may therefore lead to changes in DF histologic composition that could explain changes in MR signal characteristics. Contrast enhancement was the only significant variable related to tumor behavior found in this study. Perfusion techniques could offer a more thorough analysis of DF vascularity and could be interesting to evaluate in future studies on treatment response and growth behavior [32].

This study has several limitations that need to be acknowledged. The number of patients included was small. Progressing tumors were particularly scarce. Patients with newly diagnosed DF were analyzed together with post-surgical recurrences of DF, which may have a different behavior. DF is relatively rare and it is difficult to find lesions that fulfill the inclusion criteria for an image follow-up study. Multicenter studies could help overcome this problem. A comparison of the signal characteristics between regressing and stable tumors was not possible in this study. Although only enhancement analysis yielded significant results, signal changes on T1- and T2-weighted sequences showed similar variation tendencies. This is probably due to the relatively small number of patients and the statistical model required for data analysis, which need to take in account multiple variables (signal characteristics, clinical behavior, treatment and follow-up time). Although RECIST is frequently used for the follow-up of DF it is known that some lesions show asymmetric size reduction, which tends to be more important in the short axis than the long axis. Further studies are necessary to ascertain this matter. The grading system used was subjective. A simpler evaluation method was preferred to facilitate use in clinical setting, because quantitative assessment can be time consuming and technically challenging.

In conclusion, hormonal therapy has an impact on DF contrast enhancement and likely signal on T2-weighted images. A decrease in DF enhancement over patient follow-up is seen in patients undergoing hormonal therapy, which in the studied population was more effective in controlling lesion size than simple observation. Very low signal on T2-weighted images and/or enhancement grades (more than 50% hypointense) were more frequent in stable or regressing DF lesions. On the other hand, high signal on T2-weighted images and diffuse enhancement showed no clear association with lesion behavior. This information should be considered for image interpretation in the imaging follow-up of DF.

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Disclosure of interest

The authors declare that they have no competing interest.

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