Cytogenetic, Clinical, and Morphologic Correlations in 78 Cases of Fibromatosis: A Report from the CHAMP Study Group

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Whether fibromatoses are neoplastic or reactive lesions has long been controversial and the relationship, if any, between the superficial and deep forms (desmoid tumors) are poorly understood. Clinical, pathologic, and cytogenetic data of 78 cases of fibromatosis were analyzed and correlated with each other. The results demonstrate that clonal chromosome aberrations are a common feature of this entity, being present in 46% of desmoid tumors, although less frequent in the superficial types (10%). In the deep-seated extra-abdominal fibromatoses, trisomies 8 and 20 and loss of 5q material were the only recurrent features. No correlation between +8 and local recurrence was found. Our findings provide additional evidence for the neoplastic nature of fibromatoses.

KEY WORDS: Cytogenetics, Desmoid, Fibromatoses, Soft tissue tumors.

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Fibromatoses are a broad group of locally aggressive but nonmetastasizing, highly differentiated fibrous proliferations characterized by infiltrative growth and a tendency to local recurrence after surgery. Although their microscopic appearance is similar, they have been divided into subtypes with different clinical characteristics (1). Superficial fibromatoses are slowly growing lesions arising from the fascia or aponeurosis in the hand (palmar fibromatosis, Dupuytren's disease), foot (plantar fibromatosis, Ledderhose's disease), or penis (Peyronie's disease). Deep fibromatoses (desmoid tumors) are usually more rapidly growing tumors that can attain a large size. They can occur in the abdominal wall in young women, within the abdominal cavity, and most often in extra-abdominal localizations, especially limbs and limb girdles (2). Most desmoid tumors represent single sporadic lesions, but occasionally multiple tumors in the same anatomic region do occur (3, 4). Some present in a familial context, as in Gardner's syndrome in association with colonic polyps (5). In children, several distinctive types of juvenile "fibromatoses" are recognized (6).

Clinical characteristics of the fibromatoses have been well-documented (2). The incidence of palmar fibromatosis in the general population is 1 to 2%; it is rare in persons younger than 30 years but affects almost 20% after 65 years. It is four times more frequent in men, and is rarely encountered in Blacks and Orientals. In about half of the cases, the lesion is bilateral. Plantar fibromatosis is more infrequent and occurs at a younger age, including children. It is about twice as common in men as in women and is bilateral in 10 to 25% of the cases. Penile fibromatosis is more common in patients with palmar (2 to 4%) and plantar (1 to 2%) fibromatosis than in the general population. The incidence of deep fibromatosis is estimated at three to four new cases per million per year. In patients with familial adenomatous polyposis (FAP), caused by

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mutations in the *APC* gene, clinically detected desmoid tumors occur in 5 to 19% (5).

A spontaneous evolution over time occurs in some fibromatoses, especially superficial lesions with an early cellular proliferative phase and a late less cellular phase, with more mature fibroblasts and more collagen. The central portion of desmoid tumors is often hypocellular, whereas there is generally greater cellularity at the periphery. By electron microscopy, these lesions are found to consist principally of fibroblasts and myofibroblasts in varying proportions. By immunohistochemistry, the cells are found to be positive for vimentin, α-smooth muscle actin, and (rarely) desmin (7).

Although the lesions of fibromatosis often invade surrounding tissues, they never give rise to metastases (8). Some tumors grow relentlessly, whereas others become stationary and may even regress. The clinical issues are a function of tumor location, mesenteric and neurovascular involvement being the most critical. Most desmoid tumors are resected early after clinical presentation. Recurrence rates range between 10% for abdominal lesions in non-Gardner patients (9), 30% for sporadic extra-abdominal desmoid tumors (10), and almost 100% for mesenteric desmoid tumors in Gardner patients (11). A positive resection margin correlates with a high recurrence rate, but postoperative radiation reduces the risk significantly (12, 13). A correlation between the presence of undifferentiated mesenchymal cells and number of blood vessels with recurrence has been observed (14, 15).

The pathogenesis of fibromatosis remains obscure. Trauma, endocrine factors, radiation therapy, and scarring have been associated with its development. Reitamo and others (16, 17) found multiple minor bone malformations, such as cortical thickening, exostoses, areas of cystic translucency, compact islands, and sacralization of L5 in 80% of patients with desmoid tumors, and suggested abnormal regulation of connective tissue growth as an important predisposing factor. Recently, it was found that germline mutations in the *APC* gene also predispose to desmoid tumors (18) and that sporadic desmoid tumors also harbor mutations in *APC* or in an interacting *β-catenin* gene (19).

The first report on chromosomal aberrations in palmar fibromatosis appeared in 1975 (20), followed in 1986 by a report on Peyronie's disease (21) and in 1988 by the first karyotype of a desmoid tumor (22). To the best of our knowledge, abnormal karyotypes from 85 fibromatoses, fairly equally distributed among desmoids, palmar fibromatosis, and Peyronie's disease have been published. No

TABLE 1. Superficial Fibromatosis

Case No.	Age (yr)	Sex	Location	P/R	Size (cm)	Resection	Date	Status	Karyotype
Lu 374	44	F	Plantar	R1	1.5	Wide	25/04/85	NED	46,XX[14][a]
Lu 376	49	M	Plantar	P	5.5	Marginal	29/01/87	NED	46,XY[19]
Lu 382A	26	M	Plantar	P	1.5	Marginal	06/09/90		46,XY[22]
Lu 382B				R1	2	Marginal	08/04/91	LTF	46,XY[13]
Lu 383	62	F	Plantar	P	1	Marginal	06/05/91	LTF	47,XX,+8[2]/46,X,-X, +8[7]/46,XX[7]
Lu 387A	11	F	Plantar	P	4	Marginal	03/12/92		46,XX[21]
Lu 387B				R1	1	Marginal	13/03/95	LTF	46,XX[26]
Lu 390	23	F	Plantar	R1	1	Marginal	02/09/91	LTF	46,XX[10]
Lu 396	67	F	Plantar	P	1	Marginal	11/08/94	LTF	46,XX[30]
Lu 386	36	M	Palmar	P	1.5	Marginal	13/10/92	LTF	46,XY[12]
Leu 315	85	M	Finger	P	1	Marginal	10/12/91	NED	46XY[15]
Leu 316	61	M	Palmar	P	3	Marginal	14/01/92	NED	46XY[19]
Leu 317	58	F	Palmar	P	2	Marginal	05/02/92	NED	46XX[18]
Leu 318	66	M	Finger	P	1	Marginal	01/04/92	NED	47,XY,+8[3]
Leu 319	68	M	Palmar	R1	3	Marginal	19/05/92	R	46,XY[17]
Leu 320	61	F	Finger	P	1	Marginal	23/05/92	NED	46,XX[19]
Leu 321	72	M	Finger	P	1	Marginal	09/06/92	R	47,XY+8[15]
Leu 341	65	M	Palmar	P	2	Marginal	10/12/91	NED	46,XY[15]
Leu 342	44	M	Palmar	P	2	Marginal	25/02/92	NED	46,XY[15]
Leu 343	39	M	Palmar	P	3	Marginal	04/03/92	NED	46,XY[17]
Leu 342	44	M	Palmar	P	2	Marginal	25/02/92	NED	46,XY[15]
Leu 343	39	M	Palmar	P	2	Marginal	04/03/92	NED	46,XY[17]
Leu 344	51	M	Palmar	P	2	Marginal	10/03/92	NED	46,XY[18]
Leu 345	67	M	Palmar	P	2	Marginal	20/03/91	NED	46,XY[15]
Leu 346	48	M	Palmar	R1	2	Marginal	20/03/92	NED	46,XY[19]
Leu 347	31	M	Finger	R1	1	Marginal	25/03/92	NED	46,XY[16]
Leu 348	56	M	Palmar	P	2	Marginal	05/05/92	NED	46,XY[15]
Leu 349	69	M	Palmar	P	2	Marginal	17/07/92	NED	46,XY[18]
Leu 350	51	M	Finger	P	1	Marginal	09/09/92	NED	46,XY[15]
Leu 351	63	M	Palmar	R1	3	Marginal	11/09/92	R	46,XY[17]

P, primary tumor; R, local recurrence; NED, no evidence of disease; LTF, lost to follow-up.
[a] Figures in brackets indicate the number of metaphase cells.

case of plantar fibromatosis with chromosome aberrations has been reported up to 1999 (23).

The international CHAMP collaborative group (CHromosomes And Morphology), comprising cytogeneticists, pathologists, and surgeons, has carried out systematic studies of a variety of soft tissue tumors. As part of these studies, the clinical, pathologic, and cytogenetic data of 78 fibromatoses were analyzed and correlated with each other.

MATERIALS AND METHODS

Fibromatosis specimens that had been karyotyped successfully in Leuven or Lund between 1984 and 1997 were selected for the study. For cytogenetic analysis, fresh tumor specimens were disaggregated with collagenase, cultured for less than 10 days, harvested, and analyzed by G-band staining. Chromosomal aberrations were recorded according to the International System for Human Cytogenetic Nomenclature (24). The histologic features were reexamined separately by three group members (CDMF, JR, GT) without knowledge of the clinical or karyotypic data or of the original histopathological diagnosis.

A total of 78 tumor specimens obtained from 61 patients were karyotyped. Forty-one specimens from 27 patients were deeply located extra-abdominal fibromatoses: 28 specimens from the primary tumor, eight from a first recurrence, three from a second recurrence, one from a third and one from a fourth recurrence. Fibromatosis of the abdominal wall represented seven specimens (six primaries, one first recurrence) from six patients. There were 28 specimens (20 primaries, eight first recurrences) of superficial fibromatosis obtained from 26 patients. Finally there were two cases of mesenteric fibromatosis. Gardner's syndrome was present in two patients (Leu 296, Leu 311).

RESULTS

The karyotype was normal in 52 specimens and abnormal in 26. Among the 28 specimens of superficial fibromatosis, only three showed aberrations: trisomy 8 and additional loss of the X chromosome in one example of plantar fibromatosis (Table 1). No aberrations were found in the two tumors from Gardner patients, nor in the mesenteric tumor from a non-Gardner patient. In fibromatosis of the anterior abdominal wall, two of seven specimens were abnormal: one had trisomy 8 and the other had a 4;19 translocation (Table 2). Of the deep extra-abdominal fibromatosis cases, 21 out of 41 specimens (51%) had an abnormal karyotype (Table 3). No correlation was found between clinical features such as age, gender, location of the tumor, primary or recurrent tumor, and an abnormal karyotype. The most frequent and consistent abnormalities were numerical, as observed in 11 specimens: gains (usually trisomy) of chromosome 8 (five cases), chromosome 20 (four cases), and of both 8 and 20 in two cases (Fig. 1). Structural changes were noted in seven cases, without any obvious pattern. Loss of 5q material, either through monosomy 5 or interstitial deletion, was seen in two cases. Complex karyotypes were found in two patients, one of whom (Leu 306) had been irradiated for a right-sided breast carcinoma, and developed a desmoid tumor on the thoracic wall 10 years later.

DISCUSSION

The present study of fibromatosis, which constitutes the largest reported series of cytogenetically analyzed cases to date, demonstrates that clonal chromosome aberrations are a consistent feature of the disease process. Although some of the subtypes (*i.e.*, mesenteric fibromatosis and fibromatosis associated with Gardner's syndrome) were too infrequent to allow definite conclusions, it seems, however, as if the

TABLE 2. Abdominal Fibromatosis

Case No.	Age (y)	Sex	Location	P/R	Size (cm)	Resection	Date	RT	Status	Karyotype
Leu 311[a]	24	F	Mesentery	P	12	Marginal	19/10/94	−	SD	46,XX[15]
Leu 310	57	M	Mesentery	P	3	Wide	17/09/97	−	NED	46,XY[20]
Lu 380	21	F	Wall	P	5	Wide	30/11/89	−	NED	46,XX[25]
Lu 392	29	F	Wall	P	6	Marginal	06/09/93	−	NED	46,XX[24]
Leu 297A[a]	35	F	Wall	P	3	Biopsy	12/02/91			46,XX,t(4;19)(q21;q13)[2]/46,XX[45]
Leu 297B				P		Wide	22/02/91	−	NED	46,XX[9]
Leu 308	28	F	Wall	P	10	Marginal	24/02/97	+	NED	47,XX,+8[2]/46,XX[17]
Leu 312	26	F	Wall	P	10	Marginal	10/03/95	−	NED	46,XX[20]
Leu 314	47	F	Wall	R1	6	Marginal	08/01/97	+	NED	46,XX[20]

Leu 311: Gardner's syndrome; SD, stable disease; NED, no evidence of disease; RT, radiotherapy

[a] Cytogenetic findings previously described (29, 30).

TABLE 3. Deep Extra-Abdominal Fibromatosis

Case No.	Age (Yr)	Sex	Location	P/R	Size (cm)	Resection	Date	RT	Status	Karyotype
Leu 296	23	M	Back	R4	20	Marginal	14/09/90	−	SD	46,XY[2]
Leu 298[a]	42	F	Back	R2	3	Marginal	25/10/91	+	NED	46,XXdel(4)(p?)[25]
Leu 299A[a]	14	F	Axilla	P	10	Biopsy	06/03/92	−		46,XX[15]
Leu 299B				P		Marginal	27/03/92	−		46,XX[35]
Leu 299C				R1	5	Marginal	03/08/94	+	NED	46,XX[20]
Leu 300[a]	38	F	Back	P	4	Marginal	06/03/92	−	NED	46,XX,del(1)(p33),t(6;13)(q25;q14)[3]/46,idem,add(11)(p14)[4]
Leu 301[a]	42	F	Neck	R1	9	Marginal	18/07/92	+	NED	46,XX[19]
Leu 302A[a]	64	M	Thoracic wall	P	15	Biopsy	11/12/92	−		47,XY,+8[15]/46,XY[25]
Leu 302B				P		Marginal	06/01/93	+	NED	46,XY[10]
Leu 303[a]	14	M	Foot, ankle	P	8	Biopsy	04/02/93	+	NED	46,XY[40]
Leu 304A[a]	10	F	axilla	P	13	Marginal	11/05/93	−		46,XX[20]
Leu 304B				R1	6	Marginal	12/02/95	−	NED	46,XX[15]
Leu305A	42	M	Thoracic wall	P	10	Biopsy	23/10/96	−		46,XY,inv(4)(p14p16),del(5)(q13q22)[15]/46,idem,t(5;11)(q31;q24)[5]
Leu 305B				P		Resection	12/11/97	+	NED	46,XY,inv(4),del(5)[10]/46,XY[10]
Leu306A	82	F	Thoracic wall	P	3	Biopsy	05/10/93	−		46,XX,−4,−5,add(9)(p23),t(10;11;13)(q24;q21;p11),del(12)(q12), der(14)t(4;14)(q21;q21),−16,+5,+mar[10]
Leu306B				P		Wide	03/11/93	−	NED	Same as above
Leu 307A	55	F	Thoracic wall	P	8	Biopsy	11/05/94	−		47,XX,+20[15]/46,XX[5]
Leu 307B				P		Marginal	08/06/94	+	NED	47,XX,+20[10]
Leu 309A	15	M	Thigh	P	12	Biopsy	14/03/97	−		46,XY[19]
Lea 309B				P		Marginal	03/04/97	+	NED	47-48,XY,+8,+20
Leu 313	45	M	Back	P	19	Marginal	26/07/95	−	NED	46,XY[12]
Lu 375	34	F	Shoulder	P	3	Marginal	11/86	+	NED	43-45,XX,der(1)add(1)(p36)add(1)(q21),der(1)del(1)(p11p31)add(1)(q32),−3,del(3)(p12),−8,−9,−9,add(10)(p13),−12,−13, −13,−15,−19,add(21)(p13),add(22)(q13),+4−6mar[9]/46,XX[25]
Lu 378	76	M	Thoracic wall	P	7	Biopsy	10/12/97	−	SD	46,Y,inv(X)(p22q13)[2]/46,XY,t(13;14)(q12;q31)[2]/46,XY[9]
Lu 379	37	M	Thigh	R3	5	Marginal	27/04/89	−	NED	46,XY[24]
Lu381	50	F	Buttock	P	9	Marginal	29/03/90	−	NED	46,XX[39]
Lu 384	65	M	Thigh	P	2	Wide	29/04/91	−	LTF	45,X,−Y[3]/46,XY[8]
Lu 385A	11	F	Thigh	P	16	Biopsy	05/10/92	−		46,XX[25]
Lu 385B				P		Intralesional	09/10/92	−	SD	47,XX,+8[4]/45,XX,−6[12]/48,XX,+2,del(8)(q13q22), +der(20)t(1;20)(q21;p11)[10]/46,XX[17]
Lu 388	31	M	Thigh	P	8	Marginal	03/12/92	−	NED	47,XY,+8[5]/48,XY,+7,+8[4]/46,XY[8]
Lu389	11	M	Neck	R2	3	Marginal	17/12/92	+	NED	46,XY,del(3)(q24)[3]/46,XY[19]
Lu 391A	24	F	Thigh	P	7	Wide	20/07/92	−		46,XX[23]
Lu 391B				R1	5	Wide	05/08/93	−	NED	46,XX[25]
Lu 397[a]	73	F	Lower leg	P	5	Marginal	05/01/95	−	DOC	47,XX,+20[2]/46,XX[19]
Lu 400A	50	F	Thoracic wall	P	8	Wide	27/10/92	−		46,XX[24]
Lu 400B[a]				R1	7	Marginal	27/01/95	+	NED	48,XX,+8,+20[4]/48,XX,+20,+mar[4]/46,XX[15]
Lu 401A	16	M	Thigh	P	15	Marginal	17/08/95	−		46,XY[43]
Lu 401B			Thigh	R1	5	Marginal	07/03/96	−	NED	47,XY,+8[6]/46,XY[21]
Lu 402A	17	M	Lower arm	P	15	Wide	19/10/95	−		46,XY[21]
Lu 402B				R1	5	Marginal	15/10/96	+	NED	47,XY,+8[3]/46,XY[29]
Lu 403A	22	M	Shoulder	R1	3	Marginal	11/07/96	−		47,XY,+20[3]/46,XY[18]
Lu 403B				R2	1	Wide	20/12/96	+	NED	46,XY[25]

Leu 296, Gardner's syndrome; NED, no evidence of disease; SD, stable disease; DOC, death of other cause; LTF, lost to follow-up; RT, radiotherapy.
[a] Cytogenetic findings previously decribed (29–31).

frequency of cytogenetic abnormalities varied with the type of fibromatosis. Whereas approximately half of the samples from deep-seated sporadic extra-abdominal fibromatoses and fibromatoses of the abdominal wall (21/41 and 2/7, respectively) had clonal changes, only three out of 28 superficial cases had an abnormal karyotype. Whether quantitative cytogenetic differences between superficial and other forms of fibromatosis reflect separate pathogenetic pathways is unknown, but indirect support for this interpretation could be derived from the fact that the spectrum of clonal changes is also different among them. Although one of the three chromosomal changes characteristic for deep-seated fibromatosis (*i.e.*, trisomy 8) has also been detected as a recurrent change in superficial forms of fibromatosis, +20 and dele-

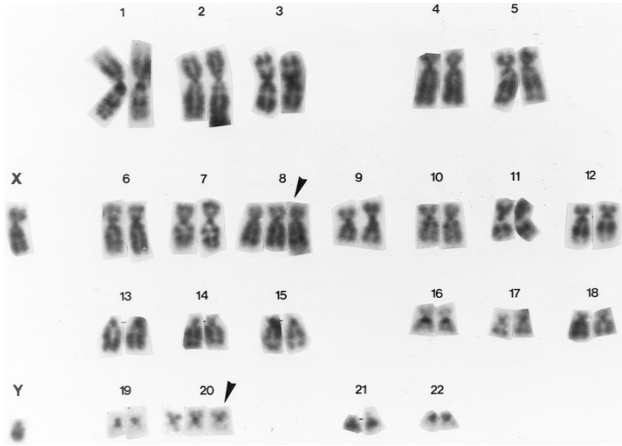

FIGURE 1. Karyotype of a desmoid tumor showing trisomy 8 and trisomy 20. The surnumerary chromosomes are indicated by ***arrows***.

tions of 5q have not (23). Conversely, loss of the Y chromosome and trisomy 7 seem to be more frequent among the superficial forms of fibromatosis (23).

The largest clinical subgroup in the present series (*i.e.*, the deep-seated extra-abdominal fibromatoses) displayed a variety of clonal chromosomal changes, with +8, +20, and loss of 5q material being the only recurrent features. These findings are in agreement with previous reports on this tumor type (23) and are different from those seen in the spindle cell sarcomas which enter the differential diagnosis (25). Whereas nothing is known about the molecular alterations resulting from the two trisomies, the loss of 5q, either through deletions or monosomy, is thought to represent one step in the functional inactivation of the *APC* gene (26). It has previously been suggested that the presence of trisomy 8, as detected by chromosome banding analysis or interphase-FISH, could be associated with increased risk of local recurrence (27). In the present series, however, no clear support for this hypothesis could be found. Trisomy 8 was indeed slightly more common in samples from recurrences (3/13) than in primary lesions (4/28). However, no recurrence has occurred 2 to 6 years postoperatively in the four patients with trisomy 8 in their primary tumor. Further complicating interpretation of the clinical significance of +8, none of the primary lesions in the three patients (Lu 400, Lu 401, and Lu 402) from whom the recurrences demonstrated trisomy 8 had any clones with +8, and in the three cases (Leu 302, Leu 309, and Lu 385) in which two samples from the same primary tumor had been obtained, only one showed +8.

The frequent finding of chromosome aberrations in fibromatosis, particularly in deep-seated lesions, and the recurrent pattern of the chromosomal changes detected, provide further evidence for the neoplastic nature of these lesions, in line with molecular data showing that fibromatosis is a clonal disorder (28). Although major structural aberrations of 5q seem infrequent in the deep fibromatoses (whether sporadic or associated with Gardner's syndrome), mutations of the *APC* gene are more common (18, 19). Further identification of the downstream targets will likely enhance our understanding of the pathogenesis of these often persistent and enigmatic tumors.

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Book Review

Meyers WM, editor: Pathology of Infectious Diseases, Volume 1: Helminthiases, 530 pp, Washington, DC, Armed Forces Institute of Pathology, American Registry of Pathology, 2000 ($145).

There are few if any pathogens that are more photogenic than helminths. Likewise, there are few that have fancier names (I picked as my favorite Archiacanthocephala, known already to Leeuwenhoek, whose curiosity, I learned from the book, made him examine the intestines of the eel!!). Unquestionably a fascinating topic for a well-illustrated book. And here it is, appearing as Volume 1 of the long-expected update of two encyclopedic works that preceded it over the past 50 years.

In my position as a hospital pathologist, I will rarely encounter most of those worms that are described in this book, and I almost feel sorry that I will not have an excuse to open this book on a daily basis for consultation. Nevertheless, even if you do not need it for day-to-day diagnostic work, you would be well advised to have it handy. It is probably the best practical manual and guide to helminthic diseases. There are several other books on this and related themes, but the beauty of the pictures, assembled in this book for our viewing pleasure, would make me rank this book one notch above all others in this category. Most of the pictures deserve to be rated as excellent (except for a few old black and white photos), but the beauty of some illustrations cannot be adequately described, without waxing poetic or schmaltzy. For morphologists like me, these pictures are a real treat and a source of wonderment as well as delight. It is a book that I will keep handy, and I will open it to my students when trying to explain why I decided to become a pathologist.

The text that accompanies the pictures is succinct and annotated with current references. It is written clearly, without jargon, and it is remarkably well edited. It complements well the photographs.

This book heralds a new era of publishing at AFIP, and we should all applaud the team that stands behind it. It is a major new step in the right direction, and we all are to be the beneficiaries—of course if the other books that follow turn out to be as beautiful as this one.

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