

Cutaneous Inflammatory Pseudotumor

Lesions Resembling "Inflammatory Pseudotumors" or "Plasma Cell Granulomas" of Extracutaneous Sites

Mark A. Hurt, M.D., and Daniel J. Santa Cruz, M.D.

This report describes four cases of a previously undocumented circumscribed idiopathic inflammatory fibrosclerotic lesion of the skin. The lesions occurred in two black women and two white men; they had been apparent from months to a year. The nodules were solitary; two were located on the arm, one on the calf, and one on the posterior neck. The clinical diagnoses included nodules, keratinous cyst, pilar tumor, pilomatricoma, vascular leiomyoma, dermatofibroma, and metastatic carcinoma. The lesions were grossly white, homogeneous, and circumscribed; one was located in the superficial subcutis and three in the reticular dermis. Histologically, they were sharply circumscribed and surrounded by a dense peripheral rim of lymphocytes and plasma cells. Peripheral germinal centers were noted in two cases. All cases had variable amounts of fibrosis centrally, giving them the low-power appearance of lymph nodes; however, no subcapsular or medullary sinuses were found. In the center of the lesions, the mononuclear infiltrate was accompanied by eosinophils and neutrophils. Virtually imperceptible vasculature was noted in portions of three lesions; in the fourth lesion, high endothelial venules were seen in the lymphoid portions, and "targetoid" perivascular sclerosis was seen in the central portions. There appears to be a transition from the early, mostly inflammatory, lesions to the sclerotic ones. The process seems to be reactive in nature. No obvious local or systemic etiology was found in our series, and follow-up of 2 to 5 years has been non-contributory. These mixed-cell proliferations resemble the extracutaneous masses that have been described in numerous viscera—particularly the lung—as "inflammatory pseudotumors" or "plasma cell granulomas." In our opinion, these lesions also bore a superficial resemblance to cutaneous lymphoid hyperplasia secondary to insect bites. Other differential diagnostic considerations include dermatofibroma, nodular fasciitis, Kimura's disease, epithelioid (histiocytoid) hemangioma, and reactive lymph nodes.

Key Words: Inflammatory pseudotumor—Plasma cell granuloma—skin.

Am J Surg Pathol 14(8): 764-773, 1990.

From the Section of Cutaneous Pathology, Division of Surgical Pathology, Department of Pathology, University of Texas Health Science Center, San Antonio, Texas (M.A.H.); and the Division of Cutaneous Pathology, Department of Pathology, St. John's Mercy Medical Center, St. Louis, Missouri (D.J.S.C.).

Address correspondence and reprint requests to Mark A. Hurt, M.D., Surgical Pathology Division (Cutaneous), University of Texas Health Science Center, 7703 Floyd Curl Drive, San Antonio, TX 78284-7750, U.S.A.

Presented in part at the meeting of the American Society of Dermatopathology, December 2, 1987, San Antonio, Texas.

"Inflammatory pseudotumors" or "plasma cell granulomas" are uncommon idiopathic benign lesions that have been described in a variety of visceral organs and deep tissues, particularly the lung (10,58,60,77). In general, they are discrete, circumscribed, nonencapsulated masses composed of a mixed cellular infiltrate that includes plasma cells, lymphocytes, eosinophils, and neutrophils. Sclerosis is a finding in many lesions; high endothelial venules are also usually seen in the most prominent areas of lymphoid infiltration. Germinal centers are present in some lesions.

Over the last few years, we have had the opportunity to study four cutaneous nodules resembling the deep or visceral inflammatory pseudotumors described in the literature. Thus, we have termed these lesions "cutaneous inflammatory pseudotumors." This paper details the clinicopathological features of these cases.

MATERIALS AND METHODS

The cases were obtained from the consultation files of one us (D.J.S.C.) and from the files of Barnes Hospital and St. John's Mercy Medical Center divisions of surgical pathology. All tissues were stained with hematoxylin and eosin (H & E) by standard methods. Case 2 was supplied with slides stained with anti-IgM, IgG, IgA, κ , λ , and appropriate controls; information concerning the specific manufacturer was not supplied by the referring pathologist. In addition, histochemical and immunohistochemical stains were applied to one case (case 4) in which a paraffin block was available. The following histochemical stains were obtained: Leder, reticulin, trichrome, iron, congo red, GMS, PAS (fungal control), AFB, and B&B. Immunohistochemical stains included S-100 protein (polyclonal, 1:500 dilution, DAKO), vimentin (monoclonal, 1:10 dilution, DAKO), and Factor VIII-related antigen (polyclonal, 1:200 dilution, DAKO).

RESULTS

The clinical features are summarized in Table 1. There were two black women and two white men. The patients were 19, 39, 40, and 64 years old, respectively. The only patient with a known disease was case 4, who had, in the year prior to the development of the skin lesion, undergone segmental colectomy for Dukes' A colon adenocarcinoma. None of the patients gave any history of bites or injections, and the lesions were of relatively uniform size. A wide differential diagnosis was considered, including fibrous, adnexal, and metastatic tumors. The follow-up period ranged from 2 to 5 years. Postexcision, there were no recurrences at the site or development of new lesions.

The pathologic features are summarized in Table 2. The epidermis was normal in all cases; no hyperplasia was present. All of the nodules were well circumscribed but not encapsulated (Fig. 1a, 2, 3a). There were no sinuses between the surrounding tissue and the nodules; all the lesions showed a direct transition (Fig. 1b). Case 1, however, had a small artifact in the deep portion that mimicked the subcapsular sinus of a lymph node and was responsible for the initial diagnosis of a dermal lymph node (Fig. 1a). Cases 1-3 were located in the reticular dermis; the fourth nodule was located principally in the subcutis and connected by a small track of reticular dermis.

Low-power architecture was diffuse to slightly fascicular in cases 1 and 2 (Figs. 1a and 2). Cases 3 and 4 (Fig. 3c) displayed some storiform arrays; this is the most likely reason that case 3 was initially diagnosed elsewhere as a dermatofibroma. All of the lesions contained varying amounts of sclerosis, which were blue with the trichrome stain, but it was most prominent in case 4 (Fig. 3b, d). In fact, this particular case showed a slight multinodular pattern with thickened collagen fibers, but no keloidal collagen was found. Reticulin stains highlighted the complex supporting meshwork (Fig. 3d inset). Congo red stain was negative, and minimal iron was seen within the lesion.

The vascular pattern was virtually imperceptible

on H & E in cases 1 and 2; however, cases 3 and 4 showed a number of high endothelial venules predominantly in the areas of heaviest mononuclear infiltration. Case 4 also showed variable numbers of these venules in the sclerotic areas (Fig. 3d); some were surrounded by laminations of dense collagen that formed a "targetoid" appearance. Factor VIII-related antigen also demonstrated a number of thin-walled vessels throughout the more diffuse and inflammatory portions.

A mixed inflammatory infiltrate was present in all cases. In fact, germinal centers were seen in the peripheral portions of cases 1 and 4 (Figs. 1a and 3a). There was a predominance of mononuclear cells, principally plasma cells, in the first two cases. A cutaneous lymphoma with plasmacytoid features was initially suspected in case 2, but immunohistochemical stains for immunoglobulins showed a polyclonal staining pattern with IgG, IgA, IgM, κ and λ. A small number of eosinophils were also present in the first two cases, but fewer of neutrophils were seen than in cases 3 and 4. In the latter two cases, however, a mixed-cell infiltrate, including many neutrophils, was noted; it was more prominent in case 4. Leder stain highlighted these focal collections of neutrophils; minimal numbers of mast cells were seen. Polarization microscopy for foreign material, and GMS, PAS (fungal control), and AFB stains on all cases were negative.

Additional immunohistochemical stains on case 4 revealed that the stroma was rich in vimentin-containing cells and showed considerable participation of S-100 protein-positive dendritic cells.

DISCUSSION

Lesions with such names as "inflammatory pseudotumor" or "plasma cell granuloma" have been reported for many years. These inflammatory masses can occur at any age. They are found most commonly in the lungs, where over 200 cases have been reported (10,58,60,77); but many other sites of visceral or deep soft-tissue involvement have been

TABLE 1. Cutaneous inflammatory pseudotumor: clinical features

Case	Age (yr)	Sex	Race	Site	Size (cm)	Duration	Clinical diagnosis	Follow-up
1	19	F	B	Posterior neck	1.3	1 yr	Keratinous cyst vs. pilar tumor	5 yr
2	39	M	W	Rt upper arm	0.8	Unknown	Lesion	5 yr
3	40	F	B	Rt calf	0.8	1 yr	Dermatofibroma	4 yr
4	64	M	W	Lt arm	1.3	8 mo	Pilomatricoma vs. vascular leiomyoma vs. metastatic carcinoma (colon primary, Dukes' A)	2 yr

TABLE 2. Cutaneous inflammatory pseudotumor: pathology

Case	Size (cm)	Location	Stroma	Germinal centers	Infiltrate	Vessels	Orig diagnosis
1	1.4	Dermis	Diffuse, fibrous	Peripheral	Mixed, plasma cell-rich	I, HE	Lymph node
2	0.8	Dermis	Diffuse, some storiform	No	Mixed, plasma cell-rich	I	Lymphoid hyperplasia
3	0.9	Dermis	Diffuse, storiform	No	Mixed, PC, E, N	HE	Dermatofibroma
4	1.3	Subcutis	Slight nodular pattern	Peripheral	Mixed, PC, E, L, N	HE	Inflammatory pseudotumor

PC = plasma cell, E = eosinophil, L = lymphocyte, N = neutrophil, HE = high endothelial venule, I = imperceptible vasculature.

described, mainly as single case reports or small series (Table 3) (24–29,53–62,71–73).

Whatever the anatomic site, the lesions share many common features; these include circumscription, variable amounts of sclerosis, and a mixed-cell infiltrate with plasma cells, lymphocytes, eosinophils, and neutrophils. Reactive germinal cells are present in some lesions, as are xanthoma cells. Some lesions are associated with myofibroblastic spindle cells resembling sarcoma, whereas others have dense sclerosis resembling localized fibromas or fibromatoses (70). Lymphomas are usually excluded after recognition of the polymorphous infiltrate.

The spectrum of histological variability was so obvious in the 32 pulmonary pseudotumors studied by Matsubara et al. that the lesions were subdivided into “organizing pneumonia-type,” “fibrocytic-histiocytic type,” and “plasma cell type” (58). The concept of a wide morphological spectrum in these lesions is certainly reasonable. When compared with individual case reports from extrapulmonary sites, it serves as useful nosological purpose, since virtually every lesion has been associated with an excellent prognosis. Most patients have had presenting signs related to a mass or obvious obstructive signs if a viscus was involved. The patients who had systemic illnesses associated with variable signs of fever, anemia, weight loss, hypergammaglobulinemia or hormone (41) abnormalities became asymptomatic after excision of the lesion.

A heterogeneous group of lesions including the progressive sclerotic, sometimes multifocal (21), inflammatory conditions of sclerosing retroperitonitis (14,59), sclerosing mesenteritis (48) and peritonitis (51), sclerosing cholangitis (9), sclerosing mediastinitis (52), Riedel’s thyroiditis (89), tumefactive fibro-inflammatory lesions of the head and neck (63,87), orbital pseudotumor (4), and so-called “systemic Weber-Christian disease” (12) share many of the purely histological features of the visceral inflammatory pseudotumors and the cutaneous

nodules described herein. The major difference with the progressive lesions is that they are not confined to solitary circumscribed masses; instead they have an infiltrative growth that encases vital structures at the site of involvement. The relationship of the progressive lesions to the solitary pseudotumors is not known, but in our opinion, the natural history indicates that the progressive lesions are clinically distinct processes that are biologically more aggressive than the solitary lesions.

We have been unable to find a cutaneous lesion comparable to the ones we describe; however, the cases reported by Hale and Mackenzie (38) are similar in some respects. They described five cases of idiopathic inflammatory nodules in the shoulders of young patients. None of the patients had any systemic signs or symptoms. All lesions were excised and none recurred. Their cases were all subcutaneous, and none involved the dermis. Histologically, they exhibited a mixed-cell infiltrate with germinal centers and an eosinophilic (not neutrophilic) infiltrate. These lesions differed from ours in location, circumscription, and neutrophilic infiltrate. Despite the lack of clinical signs and symptoms, we suspect that their cases represented examples of Kimura’s disease.

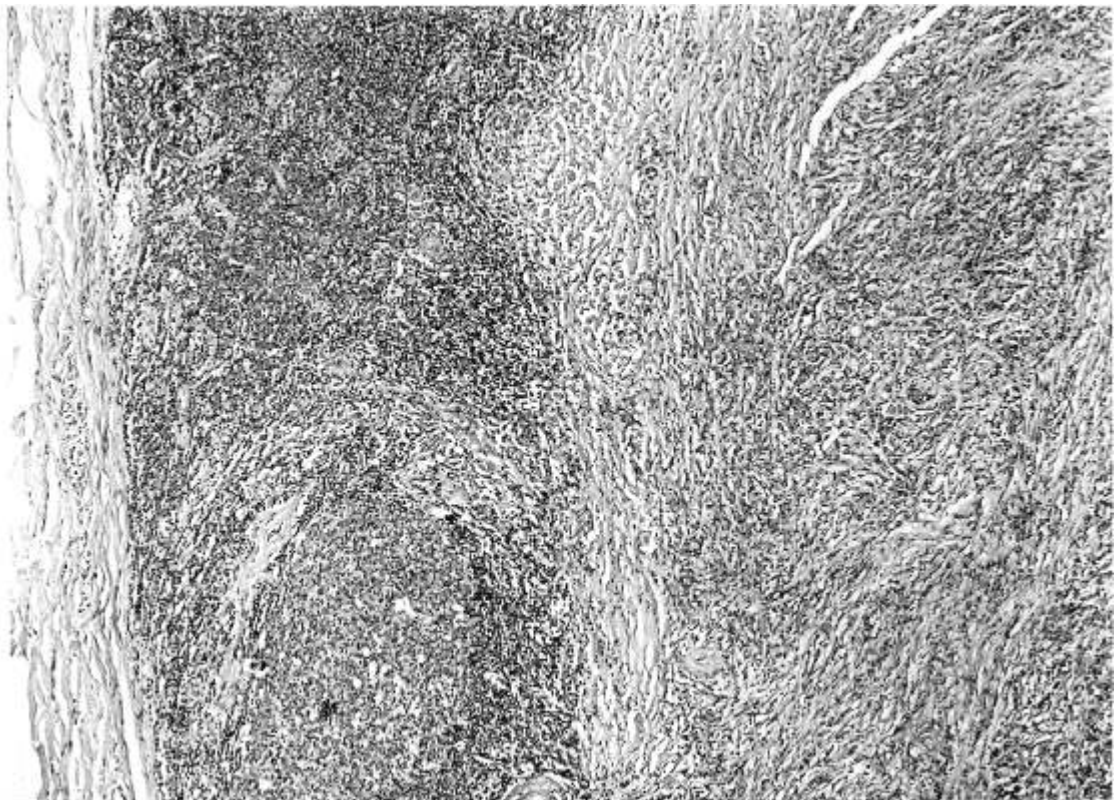
We believe that the cutaneous lesions described in this communication are morphologically similar to the circumscribed visceral lesions: the “plasma cell granulomas” or “inflammatory pseudotumors.” We have not observed as wide a range of histological changes as in the pulmonary lesions, but there appears to be at least some variation in our small series—namely, a spectrum of fibrosis, an abundance of mixed-cellular infiltrate, and variable numbers of peripherally located germinal centers. Our observations also indicate that the number and type of inflammatory cells are variable; some nodules displayed numerous eosinophils and neutrophils, whereas others contained a predominance of mononuclear cells. The high endothelial venules seen adjacent to the germinal centers and areas of

(a)



FIG. 1. (a) Case 1. Low-power view of lesion showing circumscription and peripheral germinal centers. The central portion appears homogeneous and consists of a mixed infiltrate. **(b)** Medium-power view showing the periphery with one germinal center and central sclerosis with mixed inflammation. No true subcapsular sinus can be seen.

(b)



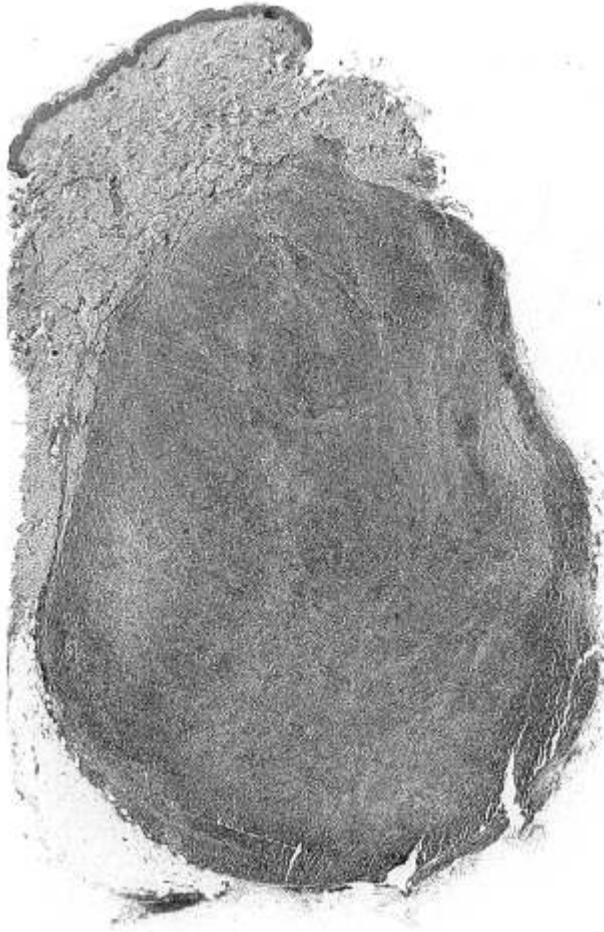


FIG. 2. Case 2. This small circumscribed dermal nodule lacks the germinal centers. It was characterized by lymphocytes and a heavy polyclonal plasma cell infiltrate confirmed by immunoperoxidase stains. Small numbers of eosinophils and neutrophils were present.

mononuclear infiltration most likely correspond to the similar endothelium found in lymph node- and mucosa-associated lymphoid tissue; presumably its function is to mediate lymphocyte trafficking (49). It is conceivable that some of these lesions represent an obliterative fibrotic inflammatory process within a superficial lymph node, but we cannot be completely certain.

The differential diagnosis of these cutaneous nodules is wide, but for practical purposes, it can be restricted to cutaneous malignant lymphoma, a few selected tumors and hyperplasias, and the patterns seen in some foreign body reactions, insect bite reactions, and reactions to certain infectious agents.

Cutaneous malignant lymphoma can readily be differentiated from the cutaneous inflammatory pseudotumors principally by identifying the polymorphous mixture of the infiltrate, circumscription,

and sclerosis in the latter lesion. In cases where there is some question, anti-immunoglobulin stains may show the mono- or polyclonal nature of the infiltrate, especially if it is plasmacytoid, as was the situation in our case 2. It is distinctly uncommon for a cutaneous lymphoma, either of B- or T-cell type, to have sharp circumscription in the skin. These lesions are almost always infiltrative; often they have a "bottom-heavy" pattern that may involve both dermis and subcutis. In cases of non-mycosis fungoides cutaneous T-cell lymphoma, the lymphocytes are hyperconvoluted or obviously pleomorphic as opposed to the reactive appearance of our cases.

Cutaneous lymphoid hyperplasia or "pseudolymphoma," also contains germinal centers, but in general, it lacks the circumscription, fibrosis, and appropriate mixed-cell infiltrate found in all our cases. Castleman's disease is a particular type of lymph node hyperplasia that occurs in three forms (33): the hyaline-vascular type, the plasma cell type, and the transitional or mixed type. These conditions can be separated morphologically from cutaneous inflammatory pseudotumors principally because of the polymorphous infiltrate seen in the latter condition. In addition, our cases showed no obvious remaining lymph nodes.

Benign, so-called "fibrohistiocytic" or myofibroblastic tumors, such as dermatofibroma, adult myofibromatosis, nodular fasciitis, and dermatofibrosarcoma protuberans can be differentiated from our cases by several criteria. Dermatofibromas generally exhibit epidermal hyperplasia over the lesion, poor circumscription in the dermis, and, in some cases, peripheral "keloidization" of collagen and hemosiderin with aneurysmal areas. These features were not seen in our cases.

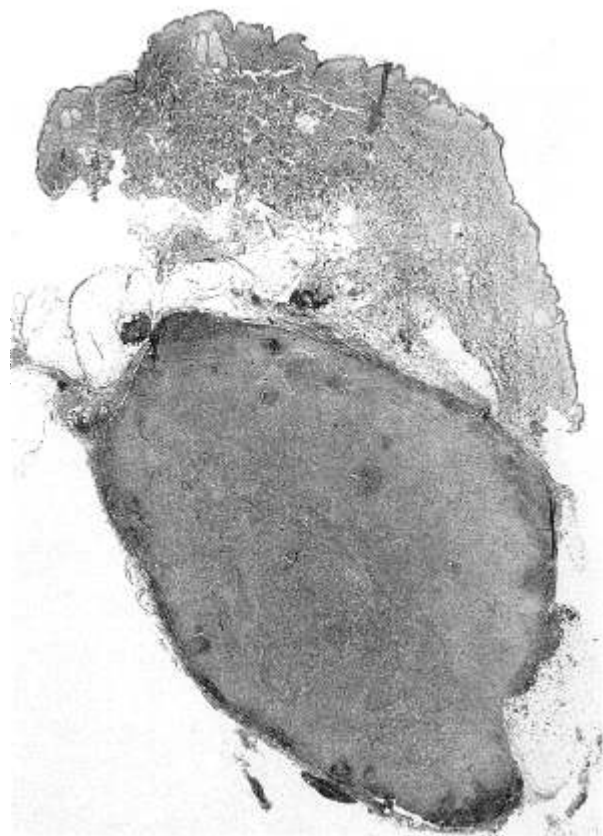
Adult myofibromatosis is a circumscribed multinodular lesion that occurs in the dermis or subcutis (23). It is composed of spindle cells, cellular mesenchymal cells, and areas resembling smooth-muscle cells. The sharp circumscription is similar to our lesion, but adult myofibromatosis lacks a significant inflammatory infiltrate.

Nodular fasciitis is typically a circumscribed subcutaneous lesion with a "tissue culture" growth pattern; it contains numerous spindle cells. Mitoses are common; by contrast, our cases showed bland cytology and sclerosis.

Dermatofibrosarcoma protuberans can be excluded due to its diffuse, infiltrative growth pattern from the dermis into the subcutis. Additionally, these lesions usually lack a significant inflammatory infiltrate.

Other recently described lesions which have

(a)



(b)

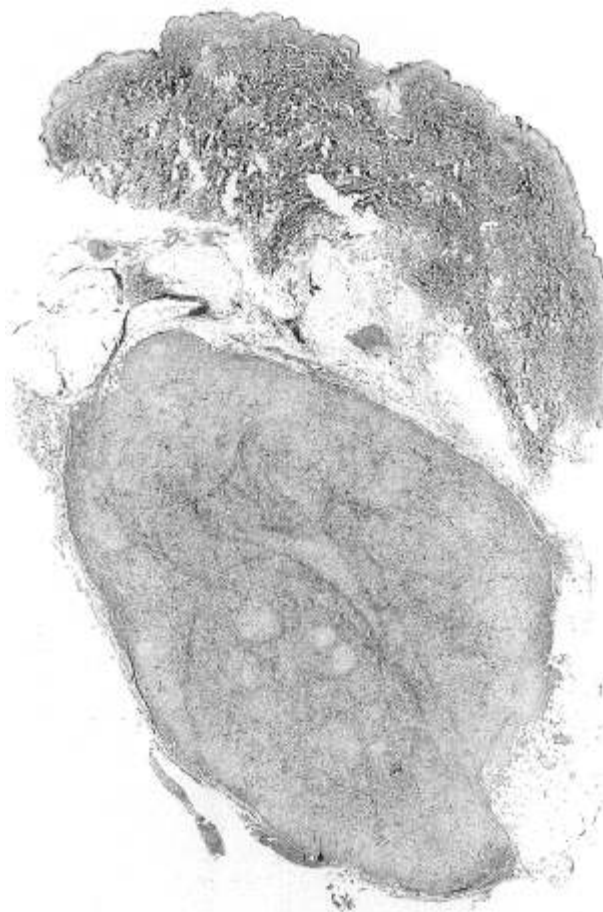


FIG. 3. (a) Case 4. This subcutaneous nodule is well circumscribed with peripheral lymphoid and germinal center formation and a central core of fibrosis. (b) Reticulin stain of this lesion accentuates the central sclerotic areas as hypodense areas separated by linear reticular bands.

been termed “sclerotic fibromas of the skin” (69) and “circumscribed storiform collagenomas” (57) have circumscription similar to our cases, but they lack any inflammatory infiltrate or prominent vascular changes.

Epithelioid (histiocytoid) hemangioma (angiolymphoid hyperplasia with eosinophilia) and Kimura’s disease, lesions which are often confused with each other (36,83), can both be separated from cutaneous inflammatory pseudotumors by applying several criteria. In epithelioid hemangioma, the lesion may affect the dermis, subcutis, or deep tissues. The vascular component is expressed as lobules of plump “epithelioid” endothelial cells protruding into conspicuous vascular lumina, sometimes obliterating the vascular space. The vascular component is proliferative and predominates over the cellular infiltrate. Additionally, although an eosinophilic infiltrate is present in some lesions, it is not characteristically mixed with a neutrophilic infiltrate. In contrast, in Kimura’s disease (44), the principal

sites of involvement, alone or in combination, are lymph nodes, subcutis, and head and neck salivary glands. Clinical eosinophilia is a common feature. Dermal and subcutaneous involvement may occur in some cases (83), but the lesions are somewhat ill defined and not necessarily circumscribed. The principal histological features include variable numbers of germinal centers associated with high endothelial venules and an eosinophilic infiltrate as well as sclerosis with or without sclerotic vascular lamination. In lymph nodes, other features have been documented, including proteinaceous germinal center deposits, germinal center necrosis, and eosinophilic abscesses (44). In our opinion, the sharp circumscription, polymorphous mixture of inflammatory cells, and lack of a proliferative endothelial component seen in the cutaneous inflammatory pseudotumor help distinguish our cases from epithelioid hemangioma and Kimura’s disease. In addition, none of our patients had any of the clinical features usually attributed to Kimura’s disease.

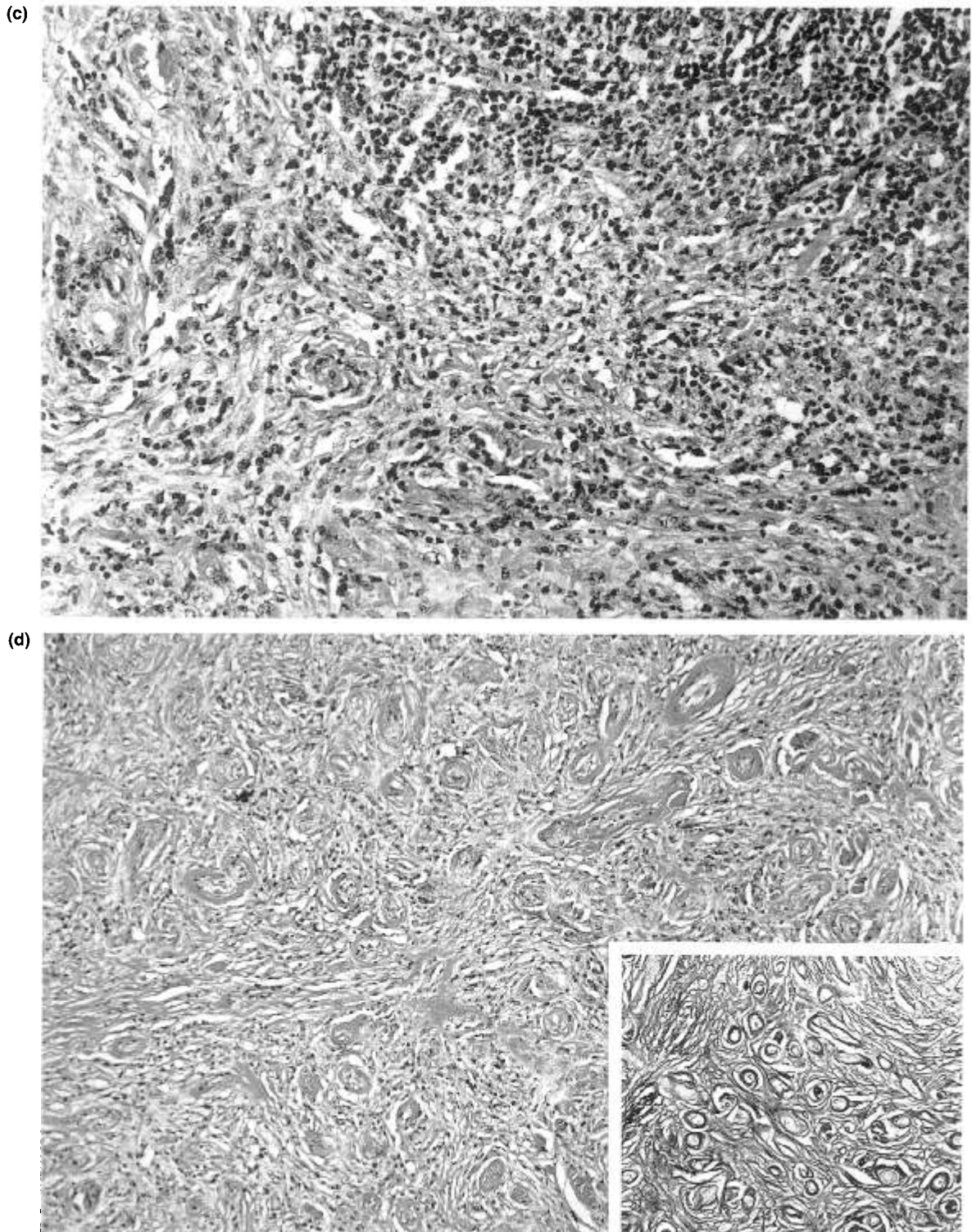


FIG. 3. (c) Higher-power view shows a storiform architecture with a mixed infiltrate of plasma cells, lymphocytes, eosinophils, and neutrophils. This type of infiltrate was seen, in varying degree, in all lesions. (d) The central portions show distinct venules with plump endothelial cells circumscribed by multiple layers of thickened collagen fibers ("targetoid" collagenosis) and associated with a mixed infiltrate. These circumscribed vessels are highlighted by reticulin stain (inset).

TABLE 3. *Extracutaneous inflammatory pseudotumors*

Involved site	Reference
Respiratory system, pleura, and mediastinum, and heart	
Oropharynx-larynx	(26), (27)
Trachea	(8)
Lung	(7), (58), (60), (77), (81), (82), (84)
Pleura	(13)
Mediastinum	(26), (27)
Heart	(18), (35), (66)
Gastrointestinal tract, mesentery, peritoneum, and retroperitoneum	
Esophagus	(53), (88)
Stomach	(45), (75), (79)
Small intestine	(25)
Large intestine	(54)
Appendix	(61)
Mesentery	(25), (26), (27), (47), (71), (72)
Peritoneum	(90)
Retroperitoneum	(90)
Hepatobiliary tract and pancreas	
Common bile duct	(37)
Gallbladder	(19)
Liver	(5), (17), (20), (42), (65), (76), (78)
Porta hepatis	(26)
Pancreas	(1), (71)
Genitourinary tract and pelvis	
Kidney	(32)
Renal pelvis	(24)
Urinary bladder	(26), (27), (46), (62), (71)
Ovary	(64)
Uterus	(34), (71)
Pelvis	(50)
Soft tissue, skeletal-muscular system, and breast	
Mandible (dental)	(92)
Parapharyngeal	(16)
Orbital (ocular)	(11), (40)
Upper limb	(70)
Lower limb	(26), (27), (70)
Breast	(68)
Lymphoreticular system	
Lymph nodes	(67)
Spleen	(3), (22), (55), (73)
Thymus	(39)
Tonsil	(85)
Central nervous system	
Parahypophyseal	(6)
Meninges, cranial	(86)
Meninges, spinal cord	(29)
Ventricle, fourth	(56)
Endocrine system	
Adrenal gland	(28)
Thyroid gland	(15), (28), (43), (80), (91)

In the skin, it is important to polarize specimens with mixed-cell infiltrates because foreign body reactions can produce a wide spectrum of histological patterns varying from almost entirely epithelioid cells (sarcoidosis-like) to neutrophilic and eosinophilic abscesses (some foreign-body reactions). A few cases in this "foreign body" spectrum have been described in detail (2). Notably, cutaneous

aluminum reactions share several features with our cases, mainly with regard to the composition of the infiltrate. The aluminum reactions differ from our cases in that they are poorly circumscribed, they have numerous germinal centers throughout the lesion (not mainly peripheral), and they contain aluminum particles (31,74). Similar changes may be produced and indeed are constant findings in reactions to infectious agents such as mycobacteria, bacteria, fungi, and mouth parts of arthropods. Most of these lesions are not circumscribed, and they seem to have a predominant cell type that may be histiocytic (epithelioid), neutrophilic, or mononuclear. Additionally, the infectious agent will explain the reaction if it is identified in tissue or cultured in the appropriate medium. The arthropod reactions often are granuloma-poor and eosinophil-rich. They may sometimes be focally intense, but they usually do not have circumscription and peripheral germinal centers.

The histological picture of chronic mixed-cell vasculitis might briefly be considered in the differential diagnosis of the cutaneous inflammatory pseudotumor because lesions of erythema elevatum diutinum and granuloma faciale have a similar inflammatory infiltrate and erythema elevatum diutinum may show considerable sclerosis (30). Both of these conditions, however, show true vasculitis and can also be excluded on clinical grounds.

Finally, superficial soft-tissue lymph nodes exist and can be readily differentiated from cutaneous inflammatory pseudotumors by the presence of subcapsular and medullary sinuses in the lymph nodes. Additionally, it is unlikely that a lymph node will be found in the dermis; however, we have seen occasional examples of small lymph nodes at the interface of the dermis and subcutis. The main difficulty in distinguishing the lymph node from the cutaneous inflammatory pseudotumor is at the initial low-power assessment.

It is our opinion that these lesions are all secondary expressions of some antigenic stimulus, but as yet there is no scientific verification. Thus, we can only offer speculation on the progression of "early" to "late" lesions. In a very liberal sense, the progression might even include some fibrohistiocytic lesions usually regarded as benign tumors. It is possible that many disparate causes lead to such inflammatory nodules; a persistent delayed hypersensitivity reaction is the most attractive hypothesis to us. □

Acknowledgment: For their assistance with photography, we thank Walter C. Claremont, Barnes Hospital-Washington University Department of Illustration, St.

Louis, MO; and Phred Petersen, University of Texas Health Science Center, Photographic Services, San Antonio, TX.

REFERENCES

1. Abrebanel P, Sarfaty S, Gal R, Chaimoff C, Kessler E. Plasma cell granuloma of the pancreas [Letter]. *Arch Pathol Lab Med* 1984;108:531-2.
2. Allen AC. Persistent "insect bites" (dermal eosinophilic granulomas) simulating lymphoblastomas, histiocytoses, and squamous cell carcinomas. *Am J Pathol* 1948;24:367-87.
3. Alpern HD, Olson JE, Kozak AJ. Inflammatory pseudotumor of the spleen. *J Surg Oncol* 1986;33:46-9.
4. Amorosa LF, Shear MK, Spiera H. Multifocal fibrosis involving the thyroid, face, and orbits. *Arch Intern Med* 1976;136:221-3.
5. Anthony PP, Telesinghe PV. Inflammatory pseudotumor of the liver. *J Clin Pathol* 1986;39:761-8.
6. Aozasa K, Iwanaga H, Kamata K. Plasma cell granuloma of central nervous system. *Histopathology* 1988;12:98-100.
7. Bahadori M, Liebow AA. Plasma cell granulomas of the lung. *Cancer* 1973;31:191-208.
8. Barker AP, Carter MJ, Matz LR, Armstrong JA. Plasma-cell granuloma of the trachea. *Med J Aust* 1987;146:443-5.
9. Bartholomew LG, Cain JC, Woolner LB, Utz DC, Ferris DO. Sclerosing cholangitis. Its possible association with Riedel's struma and fibrous retroperitonitis—report of two cases. *N Engl J Med* 1963;269:8-12.
10. Berardi RS, Lee SS, Chen HP, Stines GJ. Inflammatory pseudotumors of the lung. *Surg Gynecol Obstet* 1983;156:89-96.
11. Blodi FC, Gass JDM. Inflammatory pseudotumor of the orbit. *Br J Ophthalmol* 1968;52:79-93.
12. Brawn LA, Ramsay LE, Shortland JR, Williams JL. Systemic Weber-Christian disease with reversible bilateral ureteric obstruction. *Postgrad Med J* 1989;65:410-6.
13. Brown WJ, Johnson LC. Postinflammatory "tumors" of the pleura. *Milit Surg* 1951;109:415-24.
14. Buff DD, Bogin MB, Faltz LL. Retroperitoneal fibrosis. A report of selected cases and review of the literature. *NY State J Med* 1989;89:511-6.
15. Chan KW, Poon GP, Choi CH. Plasma cell granuloma of the thyroid. *J Clin Pathol* 1986;39:1105-7.
16. Chan Y-F, Tung Ma L, Yeung CK, Lam KH. Parapharyngeal inflammatory pseudotumor presenting as fever of unknown origin in a 3-year-old girl. *Pediatr Pathol* 1988;8:195-203.
17. Chen KTK. Inflammatory pseudotumor of the liver. *Hum Pathol* 1984;15:694-6.
18. Chou P, Gonzalez-Crussi F, Cole R, Reddy VB. Plasma cell granuloma of the heart. *Cancer* 1988;62:1409-13.
19. Christensen AH, Ishak KG. Benign tumors and pseudotumors of the gallbladder. Report of 180 cases. *Arch Pathol* 1970;90:423-32.
20. Collina G, Baruzzi G, Eusebi V. Inflammatory pseudotumor of the liver: report of two cases. *Tumori* 1987;73:407-12.
21. Comings DE, Skubi KB, Van Eyes J, Motulsky AG. Familial multifocal fibrosclerosis. Findings suggesting that retroperitoneal fibrosis, mediastinal fibrosis, sclerosing cholangitis, Riedel's thyroiditis, and pseudotumor of the orbit may be different manifestations of a single disease. *Ann Intern Med* 1967;66:884-92.
22. Cotelingam JD, Jaffe ES. Inflammatory pseudotumor of the spleen. *Am J Surg Pathol* 1984;8:375-80.
23. Daimaru Y, Hashimoto H, Enjoji M. Myofibromatosis in adults (adult counterpart of infantile myofibromatosis). *Am J Surg Pathol* 1989;13:859-65.
24. Davides KC, Johnson III SH, Marshall Jr M, Price Jr SE, Stavrides A. Plasma cell granuloma of the renal pelvis. *J Urol* 1972;107:938-9.
25. Day DL, Sane S, Dehner LP. Inflammatory pseudotumor of the mesentery and small intestine. *Pediatr Radiol* 1986;16:210-5.
26. Dehner LP. Extrapulmonary inflammatory myofibroblastic tumor: the inflammatory pseudotumor as another expression of the fibrohistiocytic complex [Abstract 86]. *Lab Invest* 1986;54:15A.
27. Dehner LP, Kaye V, Levitt C, Askin FB. Cellular inflammatory pseudotumor in young individuals: a lesion distinguishable from fibrous histiocytoma of myosarcoma? [Abstract]. *Lab Invest* 1981;44:14A.
28. De Mascarel A, Vergier B, Merlio J-P, Goussot JF, Coindre J-M. Plasma cell granuloma of the adrenal gland and the thyroid: report of two cases. *J Surg Oncol* 1989;41:139-42.
29. Eimoto T, Yanaka M, Kurosawa M, Ikeya F. Plasma cell granuloma (inflammatory pseudotumor) of the spinal cord meninges. Report of a case. *Cancer* 1978;41:1929-36.
30. English JSC, Smith NP, Kersy PJW, Levene GM. Erythema elevatum diutinum—an unusual case. *Clin Exp Dermatol* 1985;10:577-80.
31. Fawcett HA, Smith NP. Injection-site granuloma due to aluminum. *Arch Dermatol* 1984;120:1318-22.
32. Fisch AE, Brodey PA. Plasma cell granuloma of kidney. *Urology* 1976;8:89-91.
33. Frizzera G. Castleman's disease and related disorders. *Semin Diagn Pathol* 1988;5:346-64.
34. Gilks CB, Taylor GP, Clement PB. Inflammatory pseudotumor of the uterus. *Int J Gyn Pathol* 1987;6:275-86.
35. Gonzalez-Crussi F, Vanderbilt BL, Miller JK. Unusual intracardiac tumor in a child. Inflammatory pseudotumor or "granulomatous" variant of myxoma? *Cancer* 1975;36:2214-26.
36. Googe PB, Harris NL, Mihm Jr MC. Kimura's disease and angiolymphoid hyperplasia with eosinophilia: two distinct histopathological entities. *J Cutan Pathol* 1987;14:263-71.
37. Haith EE, Kepes JJ, Holder TM. Inflammatory pseudotumor involving the common bile duct of a six-year-old boy: successful pancreaticoduodenectomy. *Surgery* 1964;56:436-41.
38. Hale JE, Mackenzie DH. Subcutaneous nodular lymphohistiocytic hyperplasia. *Br J Surg* 1972;59:375-6.
39. Harpaz N, Gribetz AR, Krellenstein DJ, Marchevsky AM. Inflammatory pseudotumor of the thymus. *Ann Thorac Surg* 1986;42:331-3.
40. Heathcoate JG, Allen LH, Willis NR. Plasma cell granuloma of the lacrimal sac. *Can J Ophthalmol* 1987;22:387-90.
41. Helikson MA, Havey AD, Zerwekh JE, Breslau NA, Gardner DW. Plasma-cell granuloma producing calcitriol and hypercalcemia. *Ann Intern Med* 1986;105:379-81.
42. Hertzner NR, Hawk WA, Hermann RE. Inflammatory lesions of the liver which simulate tumor: report of two cases in children. *Surgery* 1971;69:839-46.
43. Holck S. Plasma cell granuloma of the thyroid. *Cancer* 1981;48:830-2.
44. Hui PK, Chan JKC, Ng CS, Kung ITM, Gwi E. Lymphadenopathy of Kimura's disease. *Am J Surg Pathol* 1989;13:177-86.
45. Isaacson P, Buchanan R, Mephram BL. Plasma cell granuloma of the stomach. *Hum Pathol* 1978;9:355-8.
46. Jufe R, Molinolo AA, Fefer SA, Meiss RP. Plasma cell granuloma of the bladder: a case report. *J Urol* 1984;131:1175-6.
47. Keen PE, Weitzner S. Inflammatory pseudotumor of mesentery: a complication of ventriculoperitoneal shunt. Case report. *J Neurosurg* 1973;38:371-3.
48. Kelly JK, Hwang W-S. Idiopathic retractile (sclerosing) mesenteritis and its differential diagnosis. *Am J Surg Pathol* 1989;13:513-21.
49. Kraal G, Duijvestijn AM, Hendriks HH. The endothelium of the high endothelial venule: a specialized endothelium with unique properties. *Exp Cell Biol* 1987;55:1-10.

50. Kunakemakorn P, Ontai G, Balin H. Pelvic inflammatory pseudotumor: a case report. *Am J Obstet Gynecol* 1976;126:286-7.
51. Lee RG. Sclerosing peritonitis. *Dig Dis Sci* 1989;34:1473-6.
52. Light AM. Idiopathic fibrosis of mediastinum: a discussion of three cases and review of the literature. *J Clin Pathol* 1978;31:78-88.
53. LiVolsi VA, Perzin KH. Inflammatory pseudotumors (inflammatory fibrous polyps) of the esophagus. A clinicopathologic study. *Dig Dis* 1975;20:475-81.
54. McGee HJ. Inflammatory fibroid polyps of the ileum and cecum. *Arch Pathol* 1960;70:203-7.
55. McMahon RFT. Inflammatory pseudotumor of spleen. *J Clin Pathol* 1988;41:734-6.
56. Maeda Y, Tani E, Nakano M, Matsumoto T. Plasma-cell granuloma of the fourth ventricle. *J Neurosurg* 1984;60:1291-6.
57. Maize J, Leidel G, Mullins S, Metcalf J. Circumscribed storiform collagenoma [Abstract]. *Am J Dermatopathol* 1989;11:287.
58. Matsubara O, Tan-Liu NS, Kenney RM, Mark EJ. Inflammatory pseudotumors of the lung. Progression from organizing pneumonia to fibrous histiocytoma or to plasma cell granuloma in 32 cases. *Hum Pathol* 1988;19:807-14.
59. Mitchinson MJ. The pathology of idiopathic retroperitoneal fibrosis. *J Clin Pathol* 1970;23:681-9.
60. Monzon CM, Gilchrist GS, Burgert Jr EO, et al. Plasma cell granuloma of the lung in children. *Pediatrics* 1982;70:268-74.
61. Narasimharao KL, Malik AK, Mitra SK, Pathak IC. Inflammatory pseudotumor of the appendix. *Am J Gastroenterol* 1984;79:32-4.
62. Nochomovitz LE, Orenstein JM. Inflammatory pseudotumor of the urinary bladder—possible relationship to nodular fasciitis. Two case reports, cytologic observations, and ultrastructural observations. *Am J Surg Pathol* 1985;9:366-73.
63. Olsen KD, DeSanto LW, Wold LE, Weiland LH. Tumefactive fibroinflammatory lesions of the head and neck. *Laryngoscope* 1986;96:940-4.
64. Pace EH, Voet RL, Melancon JT. Xanthogranulomatous oophoritis: an inflammatory pseudotumor of the ovary. *Int J Gynecol Pathol* 1984;3:398-402.
65. Pack GT, Baker HW. Total right hepatic lobectomy. *Ann Surg* 1953;138:253-8.
66. Pearson PJ, Smithson WA, Driscoll DJ, Banks PM, Ehman RL. Inoperable plasma cell granuloma of the heart: spontaneous decrease in size during an 11-month period. *Mayo Clin Proc* 1988;63:1022-5.
67. Perrone T, De Wolf-Peters C, Frizzera G. Inflammatory pseudotumor of lymph nodes. A distinctive pattern of nodal reaction. *Am J Surg Pathol* 1988;12:351-61.
68. Pettinato G, Manivel JC, Insabato L, De Chiara A, Petrella G. Plasma cell granuloma (inflammatory pseudotumor) of the breast. *Am J Clin Pathol* 1988;90:627-32.
69. Rapini RP, Golitz LE. Sclerotic fibromas of the skin. *J Am Acad Dermatol* 1989;20:266-71.
70. Rosenthal NS, Abdul-Karim FW. Childhood fibrous tumor with psammoma bodies. Clinicopathologic features in two cases. *Arch Pathol Lab Med* 1988;112:798-800.
71. Scott L, Blair G, Taylor G, Dimmick J, Fraser G. Inflammatory pseudotumors in children. *J Pediatr Surg* 1988;23:755-8.
72. Scully RE, Mark EJ, McNeely BU. Case records of the Massachusetts General Hospital. Case 13-1984. Cellular inflammatory pseudotumor, involving ileal mesentery. *N Engl J Med* 1984;310:839-45.
73. Sheahan K, Wolf BC, Neiman RS. Inflammatory pseudotumor of the spleen: a clinicopathologic study of three cases. *Hum Pathol* 1988;19:1024-9.
74. Slater DN, Underwood JCE, Durrant TE, Gray T, Hopper IP. Aluminium hydroxide granulomas: light and electron microscopic studies and X-ray microanalysis. *Br J Dermatol* 1982;107:103-8.
75. Soga J, Saito K, Suzuki N, Sakai T. Plasma cell granuloma of the stomach. A report of a case and review of the literature. *Cancer* 1970;25:618-25.
76. Someren A. "Inflammatory pseudotumor" of liver with occlusive phlebitis. Report of a case in a child and review of the literature. *Am J Clin Pathol* 1978;69:176-81.
77. Spencer H. The pulmonary plasma cell/histiocytoma complex. *Histopathology* 1984;8:903-16.
78. Standiford SB, Sobel H, Dasmahapatra KS. Inflammatory pseudotumor of the liver. *J Surg Oncol* 1989;40:283-7.
79. Tada T, Wakabayashi T, Kishimoto H. Plasma cell granuloma of the stomach. A report of a case associated with gastric cancer. *Cancer* 1984;54:541-4.
80. Talmi YP, Finkelstein Y, Gal R, Zohar Y. Plasma cell granuloma of the thyroid gland. *Head & Neck* 1989;11:184-7.
81. Titus JL, Harrison EG, Clagett OT, Anderson MW, Knaff LJ. Xanthomatous and inflammatory pseudotumors of the lung. *Cancer* 1962;15:522-38.
82. Umiker WO, Iverson L. Postinflammatory "tumors" of the lung. *J Thorac Surg* 1954;28:55-63.
83. Urabe A, Tsuneyoshi M, Enjoji M. Epithelioid hemangioma versus Kimura's disease. *Am J Surg Pathol* 1987;11:758-66.
84. Warter A, Satge D, Roeslin N. Angioinvasive plasma cell granulomas of the lung. *Cancer* 1987;59:435-43.
85. Weilbaecher TG, Sarma DP. Plasma cell granuloma of the tonsil. *J Surg Oncol* 1984;27:228-31.
86. West SG, Pittman DL, Coggin JT. Intracranial plasma cell granuloma. *Cancer* 1980;46:330-5.
87. Wold LE, Weiland LH. Tumefactive fibro-inflammatory lesions of the head and neck. *Am J Surg Pathol* 1983;7:477-82.
88. Wolf BC, Khettry U, Leonardi HK, Neptune WB, Bhattacharyya AK, Legg MA. Benign lesions mimicking malignant tumors of the esophagus. *Hum Pathol* 1988;19:148-54.
89. Woolner LB, McConahey WM, Behrs OH. Invasive fibrous thyroiditis (Riedel's struma). *J Clin Endocrinol Metab* 1957;17:201-20.
90. Wu JP, Yunis EJ, Fetterman G, Jaeschke WF, Gilbert EF. Inflammatory pseudo-tumours of the abdomen: plasma cell granulomas. *J Clin Pathol* 1973;26:943-8.
91. Yapp R, Linder J, Schenken JR, Karrer FW. Plasma cell granuloma of the thyroid. *Hum Pathol* 1985;16:848-50.
92. Zegarelli DJ, Rankow RM, Zegarelli EV. A large dental granuloma (? inflammatory pseudotumor) with unusual features: report of a case. *J Am Dent Assoc* 1974;89:891-4.