

# Chapter 11

## Panniculitis

**Keywords** Erythema nodosum • Nodular vasculitis • Erythema induratum • Lipodermatosclerosis • Lupus panniculitis • Factitial panniculitis

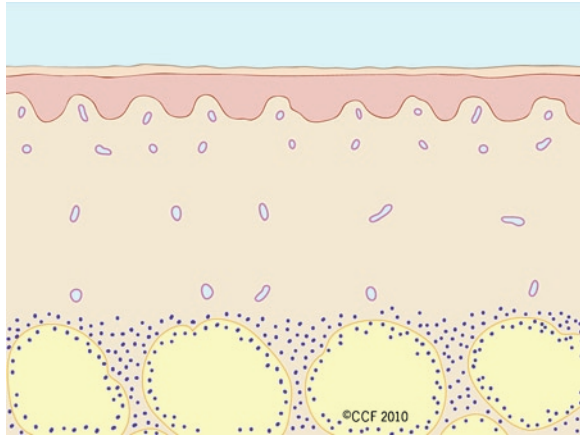
The panniculitides are a heterogeneous group of inflammatory disorders involving the subcutaneous adipose tissue. Diagnosis remains a challenge to clinicians and pathologists alike for several reasons. First, clinical monotony is common among the diseases. Second, there are often sampling issues, including inadequate superficial biopsies, which preclude optimal evaluation of the fat. Finally, as in all cutaneous inflammatory diseases, the panniculitides are dynamic processes that may demonstrate different histologic features at different stages of development. For example, early lesions of erythema nodosum are characterized by neutrophils permeating the connective tissue septa; in contrast, late stage lesions demonstrate granulomatous inflammation with prominent septal fibrosis.

Although several classification schemes for evaluation of have been proposed, the most commonly used and useful classification scheme divides panniculitides into septal or lobular patterns. That being said, essentially all cases of panniculitis demonstrate a mixed pattern of septal and lobular involvement. It is therefore critical to decide which is the predominant pattern typically best appreciated at low power. One then must look for additional histological features (composition of the inflammatory infiltrate, presence or absence of vasculitis) for definitive diagnosis.

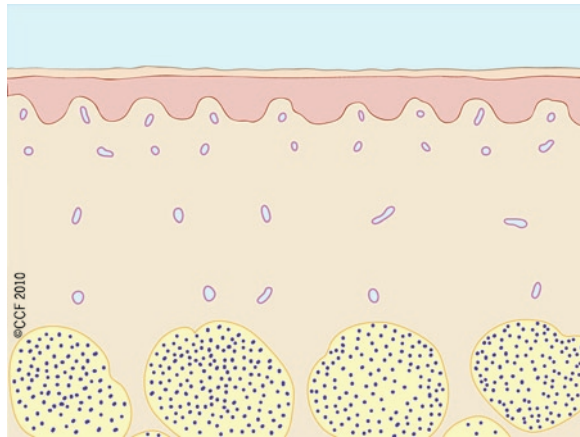
In summary, we suggest a stepwise approach when evaluating an inflammatory process in the subcutis:

- Determine the predominant location of the inflammatory cell infiltrate: septal (Fig. 11.1) vs. lobular (Fig. 11.2). This feature is best appreciated at scanning magnification
- Note the composition of the inflammatory infiltrate (neutrophilic, eosinophilic, granulomatous, or mixed)
- Examine blood vessels to determine whether there is vascular inflammation
- Note type of fat necrosis (lipophagic, enzymatic, hyaline, membranous, or ischemic)
- Finally, some diseases may require additional studies for definitive diagnosis (e.g., gene rearrangement studies to detect clonal T-cell or clonal B-cell populations)

**Fig. 11.1** *Schematic of septal panniculitis.* The pattern of panniculitis is characterized by inflammation and fibrosis that predominantly involves the septae that divided the subcutaneous lobules



**Fig. 11.2** *Schematic of lobular panniculitis.* The pattern of panniculitis is characterized by inflammation predominantly involving the fat lobules of the subcutis with relative sparing of the septae



Discussion of panniculitides in this chapter will highlight important clues to the diagnosis based on the histologic features outlined above.

## Erythema Nodosum

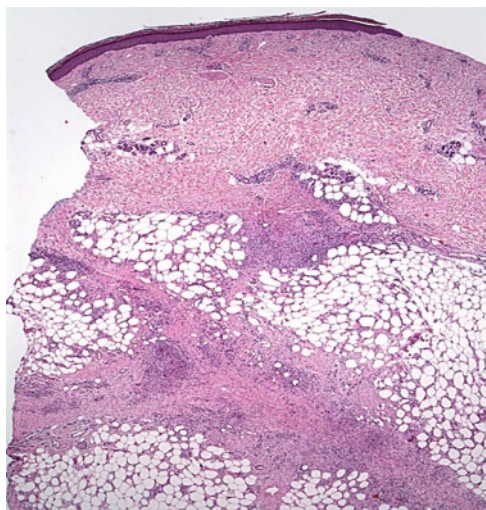
### *Clinical Features*

Erythema nodosum is the most common type of panniculitis, accounting for well over 80% of cases of panniculitis encountered in daily practice. It can occur at any age, with an incidence peak of 20–30 years of age. Erythema nodosum is characterized by the acute onset of tender, erythematous nodules or plaques most

commonly involving the shins. Lesions may be associated with fever, arthralgias and fatigue. The pathogenesis is unclear; it is probably a hypersensitivity response to underlying antigens, as it is associated with infections, drugs, malignancies and inflammatory disorders. In adults, the most frequent etiologic factors include drugs, sarcoidosis (Löfgren syndrome) and inflammatory bowel disease. In children, erythema nodosum is most often associated with streptococcal infections.

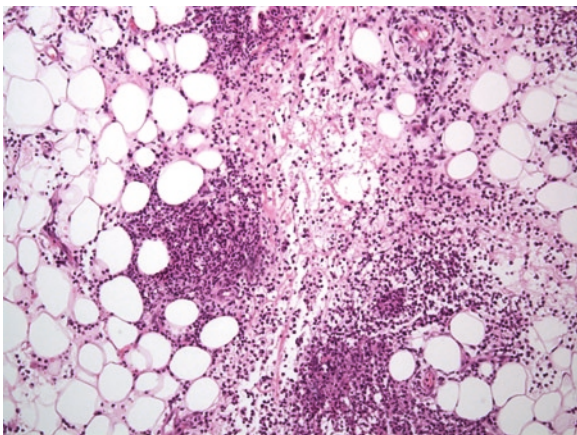
### ***Microscopic Features***

Erythema nodosum is the stereotypical example of a mostly septal panniculitis, with no vasculitis (Fig. 11.3). The composition of the inflammatory infiltrate varies with the stage of the lesions. In early lesions, the septal infiltrate is predominantly neutrophilic in nature (Fig. 11.4). Early lesions are less commonly biopsied and it is relatively uncommon to encounter the early phase of erythema nodosum. In later, well-developed lesions, there is septal fibrosis (Fig. 11.5) and the inflammatory region is composed of lymphocytes, histiocytes, eosinophils, and multinucleated giant cells (Fig. 11.6). There is usually some “spill over” of inflammatory cells into the periphery of the fat lobules. Sometimes, the central portion of the lobules may be involved, but the inflammation is still more prominent at the periphery. A histologic hallmark is the presence of so-called Miescher’s radial granulomas. These consist of small, well-defined aggregates around a central stellate cleft. Miescher’s granulomas appear in the septa sometimes surrounded by neutrophils (Fig. 11.7). These structures may be inconspicuous in some cases, seen only on examination of multiple levels. Diagnosis of erythema nodosum does not depend on the finding Miescher’s granulomas; the diagnosis depends on recognition of the predominant septal pattern of panniculitis.

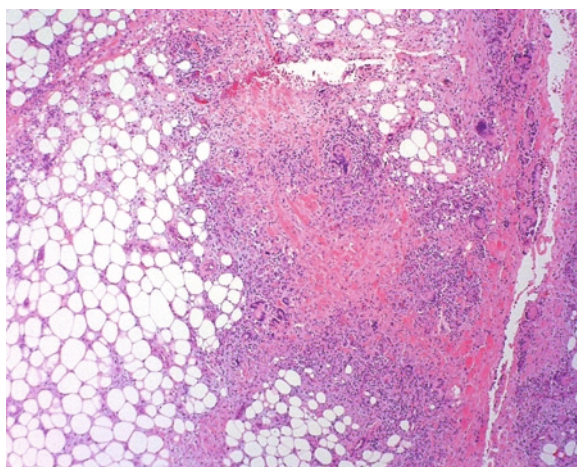


**Fig. 11.3** *Erythema nodosum.* Evaluation of any panniculitis should start at scanning magnification, to determine whether the process is septal, lobular or mixed. Erythema nodosum is the prototypic example of a septal panniculitis. There is no evidence of a vasculitis

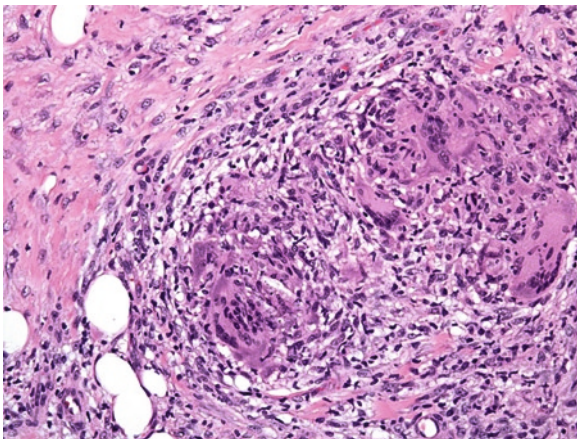
**Fig. 11.4** *Erythema nodosum* – early lesion. Early lesions of erythema nodosum are characterized by a neutrophilic inflammatory infiltrate and edema of the septum more than fibrosis, prompting consideration of an infectious process



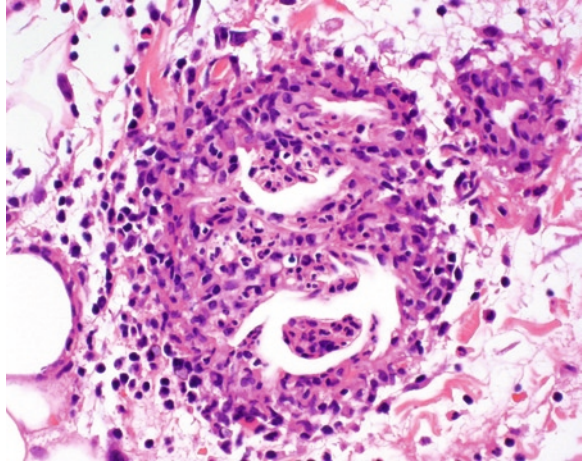
**Fig. 11.5** *Erythema nodosum* – well-developed lesion. There is striking septal fibrosis accompanied by a brisk lymphohistiocytic infiltrate with multinucleated giant cells



**Fig. 11.6** *Erythema nodosum* – well developed lesion. Multinucleated giant cells within fibrotic septae are characteristic of well developed lesions of erythema nodosum



**Fig. 11.7** *Miescher's granuloma of erythema nodosum.* Well-defined aggregates of histiocytes surrounding a central cleft, so-called Miescher's granulomas, are a histologic hallmark of erythema nodosum



**Table 11.1** Erythema nodosum: key microscopic features

- 
- Early lesions have more inflammation (neutrophils) and less fibrosis
  - Later lesions demonstrate septal thickening, lymphocytes, histiocytes, eosinophils, and multinucleated giant cells
  - Miescher's radial granulomas: aggregates of small histiocytes around central cleft
  - No vasculitis
- 

There may be a superficial deep perivascular lymphocytic infiltrate in the overlying dermis. Microscopic features are summarized in Table 11.1.

### ***Differential Diagnosis***

Well-established lesions of erythema nodosum are generally not a diagnostic problem. Infectious panniculitis may be a consideration, especially in earlier lesions, when neutrophils may be prominent. Sarcoidosis (discussed in Chap. 7) falls in the differential diagnosis of well-developed lesions of erythema nodosum. However, unlike erythema nodosum, in subcutaneous sarcoidosis, there are well defined, lobular-based naked granulomas with minimal or no septal involvement. Nodular vasculitis (erythema induratum), discussed below, is distinguished from erythema nodosum by the presence of vasculitis and a predominantly lobular panniculitis. Membranocystic change, a feature of lipodermatosclerosis (see below), may be seen in well-established lesions of erythema nodosum; however, lesions of lipodermatosclerosis usually demonstrate dermal changes of stasis, are less inflammatory, and occur in a distinctive clinical setting. Cutaneous polyarteritis nodosa, discussed in Chap. 6, may sometimes resemble a septal panniculitis, but is defined by a vasculitis. Practical tips for the diagnosis of erythema nodosum are summarized in Table 11.2.

**Table 11.2** Erythema nodosum: practical tips

- 
- Evaluation of all panniculitides requires an adequate biopsy (preferably a deep wedge) for optimal visualization of the inflammatory pattern and involvement of blood vessels
  - Low power examination is crucial for dividing the inflammatory process into the septal or lobular patterns
  - Erythema nodosum is the prototypic example of a septal panniculitis
  - Remember erythema nodosum accounts for the >80% of all cases of panniculitis
- 

## Nodular Vasculitis (Erythema Induratum)

### *Clinical Features*

Recent consensus opinion is that erythema induratum and nodular vasculitis are related entities. Differences are probably related to etiologic factors: the former is regarded as a tuberculin hypersensitivity reaction (a form of tuberculid occurring on the legs), whereas the latter represents the nontuberculous counterpart. Lesions are present as recurrent painful nodules most frequently on the calves.

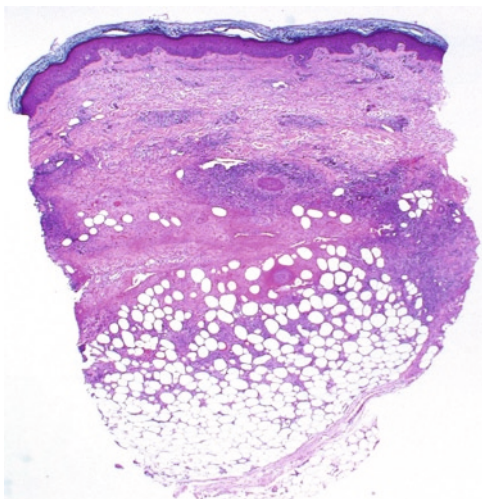
### *Microscopic Features*

Nodular vasculitis/erythema induratum are histologically identical. Nodular vasculitis is classically a lobular panniculitis, but secondary septal inflammation is commonly seen. Within the lobules, there is a granulomatous inflammation with some evidence of vasculitis (Fig. 11.8). Vascular inflammation may involve arteries, veins and venules. Vasculitic changes can show frank fibrinoid necrosis in early lesions (Fig. 11.9) to endothelial swelling and a mixed inflammatory infiltrate in the vessel walls in older lesions. In some cases, there is extensive necrosis of the panniculus with neutrophilic microabscesses. Special stains (AFB or Fite) do not demonstrate the presence of acid-fast bacilli (Table 11.3).

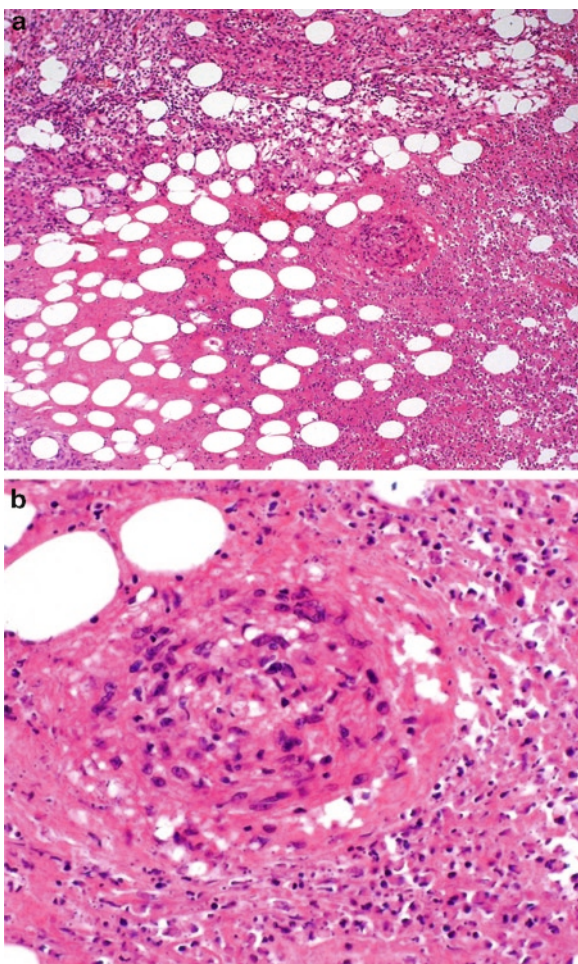
### *Differential Diagnosis*

Late stage lesions of erythema nodosum may be considered in the histologic differential diagnosis. However, erythema nodosum is a septal panniculitis and does not demonstrate features of a vasculitis. Polyarteritis nodosa is also in the differential diagnosis (see Chap. 6). Briefly, in polyarteritis nodosa, the inflammation of fat lobules is more restricted to the immediate area around damaged vessels rather than the more diffuse pattern of nodular vasculitis. Obviously, an infectious etiology should be excluded in those lesions of nodular vasculitis that demonstrate areas of neutrophilic inflammation. Special stains and cultures should be considered. In most infections, vasculitis is not a feature. See Table 11.4.

**Fig. 11.8** *Nodular vasculitis*. Scanning magnification demonstrates a lobular panniculitis associated with medium vessel vasculitis. Note how the central portion of the lobule is involved but the septum is relatively spared



**Fig. 11.9** *Nodular vasculitis*. (a) In this well-developed lesion of nodular vasculitis there is extensive necrosis of the subcutaneous fat throughout the lobule associated with vasculitis of medium-sized vessel. (b) There is fibrinoid necrosis of the affected vessel



**Table 11.3** Nodular vasculitis/erythema induratum: key microscopic features

- 
- Acute vasculitis in septae affecting artery and/or veins
  - Adjacent lobular panniculitis with granulomas and fat necrosis
  - Septae may be widened in older lesions
- 

**Table 11.4** Nodular vasculitis/erythema induratum: practical tips

- 
- Low power examination crucial
  - Inflammatory process involves the entire lobule (vs. polyarteritis nodosa in which inflammation is more restricted around vessels)
  - Look for evidence of vascular damage
  - Most common on calves
- 

## Lipodermatosclerosis (Sclerosing Panniculitis)

### *Clinical Features*

Lipodermatosclerosis is a form of long-term chronic panniculitis that presents as indurated plaques involving the lower extremities. It usually develops in middle-aged or elderly women, often with a history of venous/arterial insufficiency and previous thrombophlebitis. There is woody, erythematous induration of the lower extremities. Long-standing lesions of lipodermatosclerosis can result in a deformity of the leg that resembles an inverted bottle.

### *Microscopic Features*

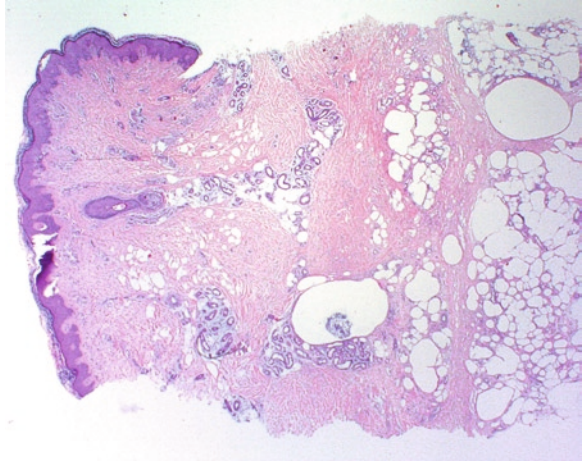
Lesions are relatively noninflammatory, an important clue to the diagnosis. At scanning magnification, there is septal and lobular fibrosis (Fig. 11.10). Within the lobules, there is formation of fatty microcysts (Fig. 11.11) and lipomembranous fat necrosis. The latter feature is characterized by cystic cavities lined with a crenulated, hyaline membrane that is PAS-positive (Fig. 11.12). Changes of stasis dermatitis may be seen in the overlying dermis (see Chap. 2) (Table 11.5).

### *Differential Diagnosis*

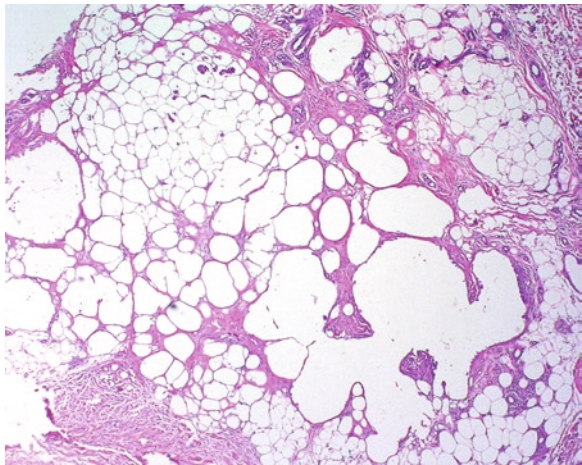
Membranocystic change is not unique to lipodermatosclerosis. It is considered to be a form of fat cell degeneration that has been described in a number of other panniculitides, including erythema nodosum, and subcutaneous morphea. However, in the appropriate clinical setting (venous insufficiency and sclerosing plaques on lower extremities) the findings are fairly diagnostic (Table 11.6).



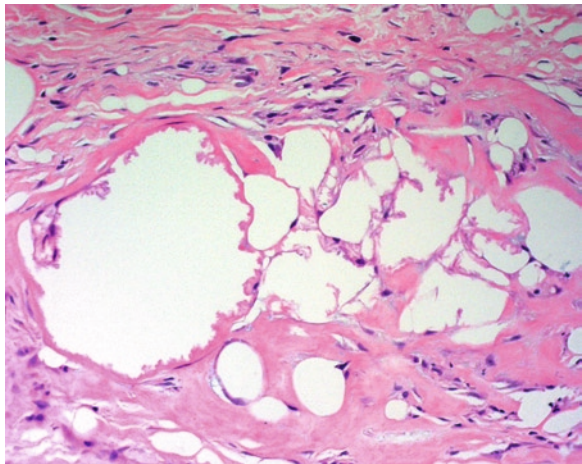
**Fig. 11.10** *Lipodermatosclerosis* appears relatively non-inflammatory and is a mixed septal and lobular panniculitis with membranocystic change. The deep reticular dermis is fibrotic



**Fig. 11.11** *Lipodermatosclerosis*. Within the lobule there is microcyst formation in association lipomembranous change accompanied by septal and lobular fibrosis



**Fig. 11.12** *Lipodermatosclerosis*. Lipomembranous fat necrosis is characterized by cystic cavities lined by a crenulated hyaline membrane



**Table 11.5** Lipodermatosclerosis: key microscopic features

- 
- Septae widened by fibrosis
  - Lipomembranous fat necrosis (cystic cavities lined by a crenulated hyaline membrane that is PAS-positive)
  - Mild perivascular lymphocytic infiltrate
  - Overlying features of stasis change in dermis and epidermis
- 

**Table 11.6** Lipodermatosclerosis: practical tips

- 
- Relatively non-inflammatory
  - Microcysts are the key diagnostic feature
  - Stasis changes of dermis
  - Clinical history of venous insufficiency
- 

## Lupus Panniculitis

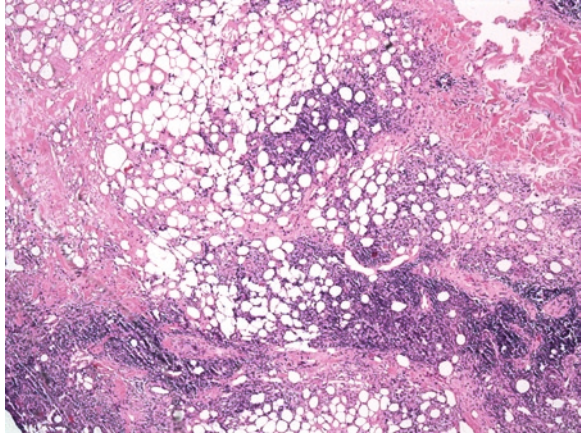
### *Clinical Features*

Lupus panniculitis, also called lupus profundus, is an unusual clinical variant of lupus erythematosus, which may occur as a separate entity in the lupus erythematosus spectrum, or be associated with discoid or systemic lupus erythematosus. Lupus panniculitis typically occurs in young to middle-aged women, and consists of deep nodules or plaques that may arise in crops. Usual involved sites are proximal extremities, particularly lateral arms and shoulders, buttocks, trunk, breast, face and scalp. The clinical presentation of a panniculitis in the upper half of the body should prompt consideration of lupus erythematosus panniculitis. Overlying erythema is commonly seen and, when clinical features of discoid lupus erythematosus are present, the skin surface may show scaling, follicular plugging, dyspigmentation or telangiectasia. Lipoatrophy may develop after resolution of the lesions. Patients with lupus panniculitis have a chronic and disabling course because of the scarring, pain and atrophy produced by the lesions.

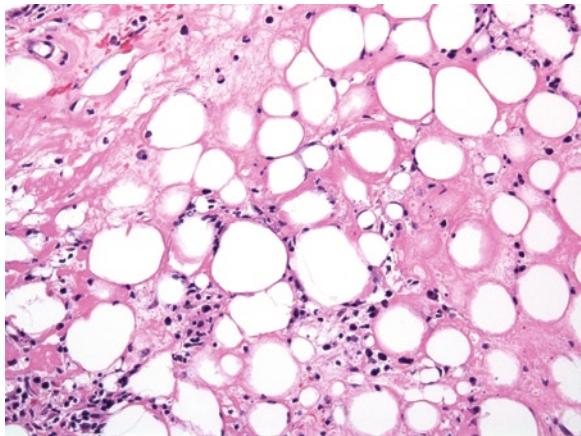
### *Microscopic Features*

Lupus panniculitis is considered a lobular panniculitis, but mixed septal and lobular involvement is common (Fig. 11.13). The inflammatory infiltrate is composed of lymphocytes accompanied by an admixture of histiocytes and plasma cells. Lymphoid aggregates, often with prominent germinal centers, are characteristic, but not pathognomonic. Perhaps, the most helpful diagnostic feature is the presence of hyaline fat necrosis, a form of fat necrosis in which fat cells undergo hyalinization resulting in a glassy eosinophilic appearance to the fat lobules (Fig. 11.14).

**Fig. 11.13** *Lupus erythematosus panniculitis*. Lupus panniculitis is predominantly lobular panniculitis, but septal involvement is common. The inflammatory infiltrate is composed predominantly of lymphocytes and plasma cells and there is hyaline fat necrosis



**Fig. 11.14** *Lupus erythematosus panniculitis*. Hyaline fat necrosis is a characteristic feature of lupus panniculitis. Hyalinization of fat cells gives a glassy appearance to the fat lobules. There are also foci of karyorrhexis, a feature often observed in lupus panniculitis



Foci of karyorrhexis in areas of necrosis are often seen. There may be vascular changes in the form of lymphocytic vasculitis (Table 11.7). Overlying histologic features of discoid lupus erythematosus may be seen in some cases; when present, they are an important clue to the diagnosis (see Chap. 3).

**Table 11.7** Lupus panniculitis: key microscopic features

- 
- The two most important histologic features are lobular lymphoplasmacytic inflammation accompanied by hyaline necrosis and nuclear dust
  - Lymphoid follicles in the subcutaneous fat are characteristic
  - Lymphocytic vasculitis may be seen in LEP
-

## ***Differential Diagnosis***

Subcutaneous morphea may demonstrate lymphoid aggregates like those seen in lupus panniculitis; however, germinal center formation is neither usually seen nor is hyaline fat necrosis or karyorrhexis. The most challenging and important entity to consider in the differential diagnosis is subcutaneous panniculitic-like T-cell lymphoma (SPTCL). Indeed, lupus panniculitis may be exceedingly difficult to differentiate from SPTCL. In brief, SPTCL is a mature T-cell  $\alpha/\beta$  lymphoma in which lymphomatous cells are positive for CD2, CD3, CD5, and negative for CD4 and CD56. Cytotoxic granular proteins TIA-1, perforin, and granzyme-B are present in almost all cases. Histologically, well-developed lesions of SPTCL are characterized by a brisk lobular infiltrate of pleomorphic, small-medium to medium-large atypical T lymphocytes. Rimming of individual fat cells by atypical lymphocytes, fat necrosis, and karyorrhexis of lymphocytes are characteristic features. Cytophagocytosis and erythrophagocytosis by macrophages (bean-bag cells) may be seen. Early cases of SPTCL may have minimal atypia; these are the cases that may be particularly difficult to differentiate from lupus panniculitis. The presence of hyaline fat necrosis, prominent mucin deposition, germinal center formation and vacuolar change at the dermal–epidermal junction all favor lupus panniculitis (Table 11.8). However, difficult cases may require a battery of immunohistochemical stains as well as gene rearrangement studies.

**Table 11.8** Lupus panniculitis: practical tips

- 
- Consider lupus panniculitis in cases of panniculitis presenting in the upper half of the body
  - Unlike other forms of lupus erythematosus, ANA serology is typically negative to low titer positive; other autoantibodies are uncommon
  - There is considerable clinical and histologic overlap with subcutaneous panniculitis-like T-cell lymphoma
    - Hyaline fat necrosis, mucin deposition, lymphoid follicle formation and interface change favor lupus panniculitis
    - Immunophenotypic and gene rearrangement studies may be needed to completely exclude lymphoma – borderline cases should be followed clinically!
  - The presence of histopathologic features of discoid lupus erythematosus in the overlying epidermis and dermis are helpful clues to the diagnosis when present
- 

## **Artifactual Panniculitis (Including Factitial, Traumatic, and Cold Panniculitis)**

### ***Clinical Features***

Artifactual panniculitides can be produced by chemical (injection of foreign material), mechanical (traumatic) or physical (cold/heat) means. The inciting event may be accidental, purposeful, or iatrogenic.

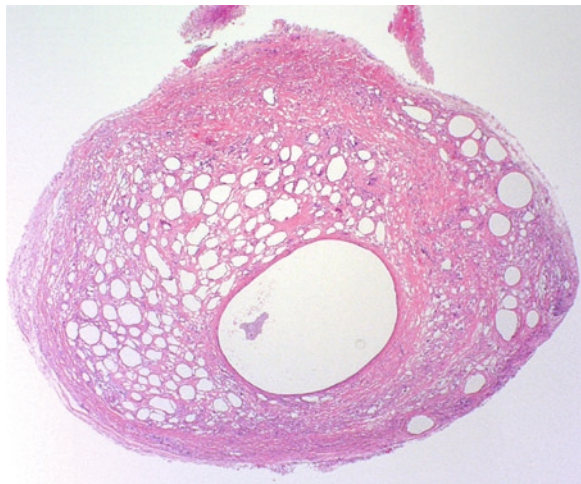
Factitial panniculitis is often characterized by confounding clinical and histologic features that defy diagnosis until self-inoculation is suspected. Most patients with factitial panniculitis are in a health care field profession with access to syringes and needles, and it is more common in women. Lesions are often localized to areas that are accessible to the hands, including buttocks and thighs. Agents implicated in factitial panniculitis include oily materials (paraffin), tissue fillers, and therapeutic agents including phytonadione (vitamin K). Some patients inject biological material such as saliva or feces.

Traumatic panniculitis does not have a specific clinical presentation; however, in adults it is most commonly seen as breast masses in women. The lesions are often indurated, warm erythematous subcutaneous nodules.

Cold panniculitis is a form of traumatic panniculitis caused by direct exposure to the cold. Infants and children are more commonly affected than adults. In children, the cheeks and chin are the most common sites of involvement. In adults, cold panniculitis usually appears in women, associated with obesity or certain sports activities including cycling or horseback riding.

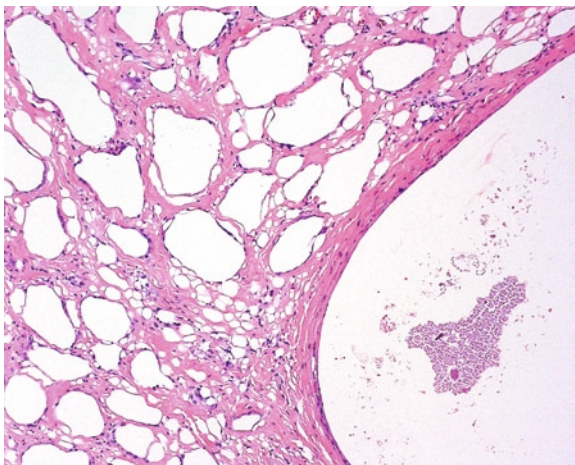
### ***Microscopic Features***

Factitial panniculitis usually shows a lobular panniculitis associated with prominent fat necrosis and a neutrophil-predominant inflammatory infiltrate. In some cases, polarization may identify birefringent material causing the panniculitis. In factitial panniculitis caused by injectable material, the substance may be seen in the overlying dermis, a feature that is not seen in other forms of panniculitis. Paraffinoma (mineral oil) is a classic example of factitial panniculitis characterized by the presence of empty spaces in the dermis and subcutaneous fat (Fig. 11.15), giving a so-called

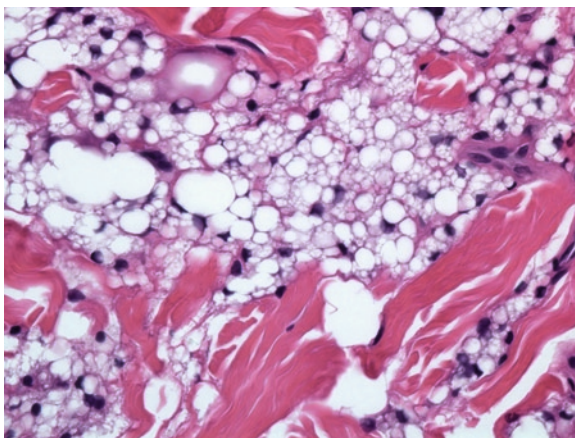


**Fig. 11.15** *Paraffinoma.*  
A form of factitial panniculitis, paraffinoma is characterized by prominent empty spaces in the dermis and subcutaneous tissue

**Fig. 11.16** *Paraffinoma*. Variably sized cystic spaces give the so called “swiss cheese” appearance of paraffinoma



**Fig. 11.17** *Silicone granuloma*. Histiocytes with multiple cytoplasmic vacuoles are characteristic of silicone granulomas



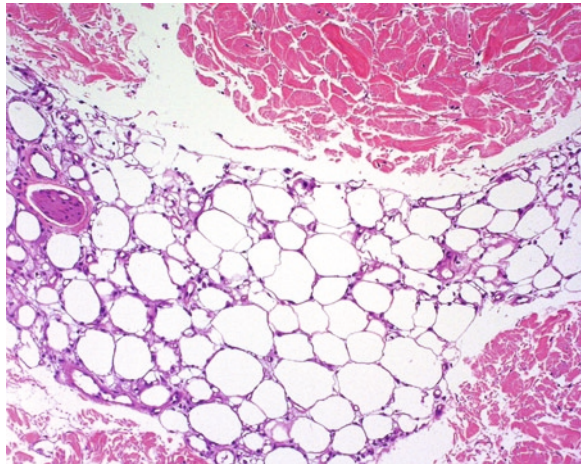
“Swiss cheese” appearance to the specimen (Fig. 11.16). The pattern of panniculitis secondary to esthetic implants varies with the material. For example, silicon granuloma is characterized by prominent foamy histiocytes with multiple vacuoles (Fig. 11.17). When biological material is injected, there is often a lobular panniculitis with abscess formation. Bacterial organisms may be identified, but their absence does not exclude the diagnosis, as cultures are more sensitive (Table 11.9).

The histological findings of traumatic and cold panniculitis are nonspecific and require some degree of clinical suspicion. Early lesions demonstrate a septal and lobular nonspecific inflammatory infiltrate of lymphocytes and macrophages (Fig. 11.18). In late lesions, there is lipotrophy with variable pseudocystic change accompanied by macrophages, fibrosis, and foreign body giant cells.

**Table 11.9** Artifactual panniculitis (factitial, traumatic/cold): key microscopic features

- Histologic features depend on the cause of the trauma. Self-inflicted injections with contaminated material produces an acute suppurative panniculitis, resembling an infection-induced panniculitis
- Paraffinoma (mineral oil) injection results in a classic “swiss cheese” appearance to the fat lobules (pseudocystic spaces surrounded by giant cells)
- Later stage lesions may demonstrate nonspecific fibrosis, lipomembranous changes, granulomas and hemorrhage
- Histologic findings of traumatic panniculitis are generally nonspecific (septal and lobular inflammation, fat necrosis, mixed inflammatory infiltrate)

**Fig. 11.18** *Traumatic fat necrosis.* The histologic features are relatively nondescript. There is often lipoatrophy with variable fat necrosis with foamy macrophages, chronic inflammation and fibrosis



## Differential Diagnosis

The histological features of the artifactual panniculitides are not always specific. In cases demonstrating acute inflammation and necrosis, infection should be excluded by antimicrobial stains (Gram’s, PAS, and Fite/AFB) and/or tissue culture. Positive cultures or the presence of microorganisms do not exclude a factitial process. In fact, cultures with more than one type of bacteria should prompt consideration of a factitial process due to injection of biologic material. Another important clue to the diagnosis is that the histologic findings do not match the clinical presentation. Frequently, the patient may have had multiple previously nonspecific biopsies. That should raise the suspicion of a factitial process (Table 11.10).

**Table 11.10** Artifactual panniculitis: practical tips

- Consider factitial etiology when there are confounding clinical and histologic features
- When acute inflammation and necrosis, are present, the differential diagnosis includes an area adjacent to a ruptured follicle/cyst, or an infectious process. Special stains and/or tissue culture may be useful in these cases
- Polarization of the slide is a cheap and quick way to identify birefractile foreign material

## Sample Reports: Erythema Nodosum

- Example 1: (Early lesion of Erythema Nodosum)  
*Clinical history:* Bilateral erythematous nodules on the legs of a 13-year-old boy.  
*Diagnosis:* Septal panniculitis with neutrophils. See comment.  
*Comment:* Initial and level sections were examined. The connective tissue septa of the subcutis are slightly thickened and expanded by an inflammatory infiltrate composed predominantly of neutrophils. Within the neutrophilic infiltrate, there are small, scattered aggregates of histiocytes around a central cleft (Miescher's radial granulomas). There is no evidence of vasculitis. The overlying dermis demonstrates a slight superficial and deep perivascular lymphocytic infiltrate. Epidermis is unremarkable. The histological findings are consistent with an early stage of erythema nodosum. If there is clinical suspicion of an infectious process, tissue culture is recommended.
- Example 2: (Well established lesion of Erythema Nodosum)  
*Clinical history:* Adult woman with ulcerative colitis presents with painful nodules on the legs. Rule out erythema nodosum.  
*Diagnosis:* Erythema nodosum. See comment.  
*Comment:* Scanning power demonstrates thickening of the connective tissue septa by an inflammatory infiltrate. In areas the inflammatory cells spill over into the fat lobules. At higher magnification, the infiltrate is composed of lymphocytes, histiocytes, and well-developed septal granulomas with prominent multinucleated giant cells. There is no evidence of vasculitis. These findings are compatible with erythema nodosum.
- Note to reader:* In cases where the clinical history is less precise, the diagnosis could be stated as "septal panniculitis consistent with erythema nodosum."

## Sample Report: Nodular Vasculitis

- Clinical history:* Painful nodules on the calf of a middle-aged woman.  
*Diagnosis:* Lobular panniculitis with vasculitis. See comment.  
*Comment:* There is a diffuse lobular inflammatory infiltrate composed of lymphocytes and neutrophils accompanied by fat necrosis with foamy histiocytes and occasional giant cells. Large areas of necrosis are observed. Medium-sized vessels in the septa demonstrate fibrinoid necrosis and intramural inflammation. Stains for microorganisms (AFB, gram, PAS) are negative. The findings are consistent with nodular vasculitis/erythema induratum. Recommend complete clinical work-up to exclude an underlying infectious process.



## Sample Report: Lipodermatosclerosis

*Clinical history:* Older woman with presenting with erythematous, indurated plaques on the lower extremities.

*Diagnosis:* Septal and lobular panniculitis with prominent membranocystic change and overlying dermal changes of stasis dermatitis. See comment.

*Comment:* There is a sparse inflammatory infiltrate of lymphocytes in the connective tissue septa, which are otherwise fibrotic. A mixed inflammatory infiltrate composed of lymphocytes, histiocytes and foamy macrophages is noted in the adjacent fat lobules. Fatty microcysts lined by amorphous eosinophilic material (membranocystic change) are a prominent feature. In the papillary and mid dermis, there is a lobular proliferation of capillaries accompanied by hemosiderin deposition and fibrosis, consistent with changes of stasis dermatitis. The clinical presentation together with the histologic pattern is consistent with lipodermatosclerosis.

## Sample Report: Lupus Erythematosus Panniculitis

*Clinical history:* Young woman with poorly circumscribed breast nodule.

*Diagnosis:* Mixed septal and lobular panniculitis with extensive hyaline fat necrosis and lymphoid follicles. See comment.

*Comment:* There is a brisk, predominantly a lobular panniculitis composed of lymphocytes accompanied by extensive hyaline fat necrosis. Lymphoid follicles surrounded by plasma cells are observed in the connective tissue septa. Foci of karyorrhexis are noted in areas of hyaline necrosis. There is increased interstitial mucin in the dermis. No vasculitis is observed. Epidermis is unremarkable. The findings are most compatible with lupus panniculitis. Lupus panniculitis may demonstrate significant overlapping features with subcutaneous, panniculitis-like T-cell lymphoma. Recommend clinical correlation and follow-up.

## Sample Reports: Artfactual Panniculitis

Example 1:

*Clinical history:* Nodule above upper lip in older woman.

*Diagnosis:* Lobular panniculitis with pseudocystic cavities and surrounding multinucleated giant cells. See comment.

*Comment:* The subcutaneous fat lobules are replaced by pseudocystic spaces surrounded by histiocytes and multinucleated giant cells. There is associated dense fibrosis. No polarizable material is seen. The findings are compatible with injection of some type of foreign material (paraffinoma). Clinical correlation recommended.

Example 2:

*Clinical history:* Nodule on thigh of a middle-aged woman.

*Diagnosis:* Mixed septal and lobular panniculitis. See comment.

*Comment:* There is a moderately brisk inflammatory infiltrate involving the septa and fat lobules with numerous neutrophils. There is also fat necrosis with coalesced fat cells forming pseudocysts lined by eosinophilic material. No vasculitis is observed. No polarizable material is demonstrated. Special stains for microorganisms (GMS, Fite, and gram stains) are negative. The findings do not fit into a traditional pattern of panniculitis. An infectious process could be considered despite negative stains. In the appropriate clinical context, the possibility of a factitial process could be considered. Clinical correlation is recommended.

## Selected References

1. Requena L. Panniculitis. *Dermatologic Clinics*. 26:4, 2008.
2. Cascajo CD, Borghi S, Weyers W. Panniculitis. *American Journal of Dermatopathology*. 22:530–549, 2000.
3. Requena L, Yus ES. Panniculitis. Part I. Mostly septal panniculitis. *Journal of the American Academy of Dermatology*. 45:163–183, 2001.
4. Requena L, Yus ES. Panniculitis. Part II. Mostly lobular panniculitis. *Journal of the American Academy of Dermatology*. 45:325–361, 2001.
5. Dahl PR, Su WPD. Panniculitis. In: Hood AF, Farmer ER (eds) *Pathology of the Skin*, 2nd ed. New York: McGraw Hill, 2000.