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ORIGINAL PAPER

# Recurrent Pancreatic Arteritis and Vasculogenic Relapsing Pancreatitis in Rheumatoid Arthritis – A Retrospective Clinicopathologic and Immunohistochemical Study of 161 Autopsy Patients

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**Abstract** The aim of this study was to determine: the prevalence, and histological characteristics of vasculitis in the pancreas, and to follow the formal pathogenesis of multifocal pancreatitis due to arteritis and/or arteriolitis (multifocal vasculogenic pancreatitis). A randomized autopsy population of 161 in-patients with rheumatoid arthritis (RA) was studied. Systemic vasculitis (SV) complicated RA in 36 (22.36%) of 161 cases; tissue samples of pancreas were available for histologic evaluation in 28 patients. Pancreatitis and vasculitis were characterized histologically and immunohistochemically. Vasculogenic, multifocal pancreatitis was not recognized clinically. Vasculitis of the pancreatic arterioles and small arteries (branches of splenic artery, upper and lower gastroduodenal arteries) can lead to local ischaemia and to regressive changes in the pancreas. This vasculogenic process is more or less widespread and multifocal, depending on the number of involved vessels and is followed by reactive inflammation, depending on the stages of the pathological process. Because of the recurrent nature of vasculitis with time these regressive changes accumulate within the pancreas and may contribute to an unexpected circulatory failure and sudden death of the patient. Vasculogenic microinfarcts in the pancreas may be clinically characterized by unexplained recurrent abdominal symptoms and spontaneous remissions which insidiously may lead to metabolic failure resistant to therapy.

**Keywords** Rheumatoid arthritis · Vasculitis · Pancreatitis

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## Introduction

The vascular changes and consequences of pancreatitis are well known. Pancreatitis can erode adjacent vessels and cause hemorrhage, hemorrhagic pseudocysts or pseudoaneurysms may develop (with or without secondary rupture and intracystic, intraperitoneal or retroperitoneal hemorrhage). Pancreatitis may be accompanied by complications such as thrombosis of the splenic, splenoportal, mesenteric etc. veins [1, 2]. Vascular complications of pancreatitis are relatively infrequent but are clinically important because of their higher mortality.

Remarkably, only a few reports describe the role of vasculitis in the pathogenesis of pancreatitis. Vasculitis may be caused by several disorders [3]. Vasculitis may accompany chronic hepatitis C (HCV) and may involve multiple organs, including the pancreas [4] etc. Vasculitis also is basic manifestation of autoimmune diseases [4]. Pagnoux et al [5] reported 62 patients with systemic small and medium sized vessel vasculitides, distributed as follows: 38 with polyarteritis nodosa, 11 with Churg–Strauss syndrome, 6 with Wegener's granulomatosis, 4 with microscopic polyangitis and 3 with rheumatoid arthritis. In 3 of 21 patients who had a surgical abdomen, laparoscopic exploration revealed acute pancreatitis.

Christl et al [6] described a previously healthy 50-year-old woman with a mass in the tail of the pancreas which masqueraded as pancreatic tail carcinoma but was caused by granulomatosus vasculitis as the first manifestation of

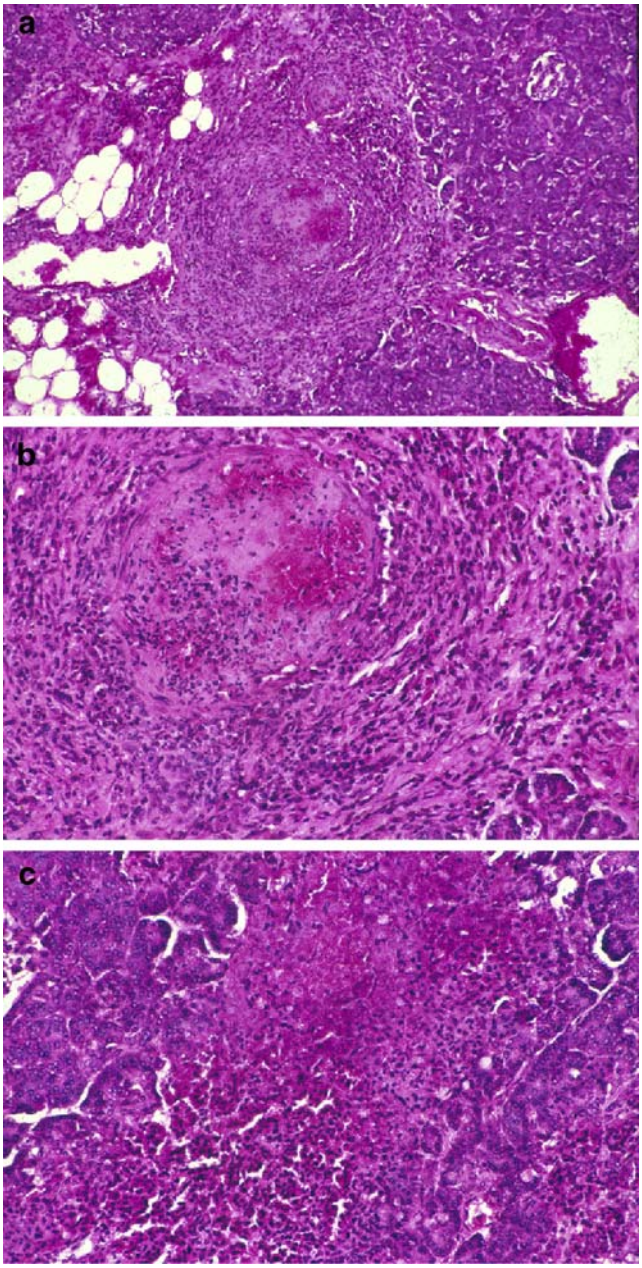


**Fig. 1** Pancreas. Arteriole. Acute necrotic thrombovasculitis, with microfocal pancreatitis. (**a**) HE, ×50 (**b**) Same as Fig. 1a, ×200 (**c**) Surrounding area of Fig. 1a, Vasculogenic focal pancreatitis, x200

Wegener's granulomatosis. Swol-Ben et al [7] reported a 59-year-old woman with SLE and vasculitis in the pancreas; the patient had pancreatitis revealed by abdominal ultrasound examination and by CT scan as pancreatic pseudotumor.

Iwasa and Katoh [8] presented a 84-year-old woman with crescentic glomerulonephritis, microscopic polyangitis and necrotizing pancreatitis due to acute vasculitis. The patient died of renal failure with disseminated intravascular coagulation (DIC). Haraguchi et al [9] published a 84-year-old woman with anti-myeloperoxidase(MPO)-antineutrophil cytoplasmic antibody(ANCA)-related microscopic polyangitis with systemic vasculitis of fibrinoid necrotic type involving the pancreas and pancreatitis. The patient died of cardiac arrest. Hidaka et al [10] described a malignant case of rheumatoid arthritis in a 15 year old boy, with mononeuritis multiplex, lung infiltration and pancreatitis. The patient died of disseminated intravascular coagulation (DIC) on 153 days after admission. Autopsy revealed vasculitis in heart, pancreas, liver and small large intestine. [10] O'Neil and colleagues [11] reported a similar case.

The aim of this study was to determine the prevalence and histological characteristics of vasculitis in the pancreas in rheumatoid arthritis (RA), and to follow the pathogenesis of multifocal pancreatitis due to arteritis and/or arteriolitis (multifocal vasculogenic pancreatitis).

## Patients and Methods

A randomized autopsy population of 161 in-patients (females 116, average age of 64.9 years; males 45, average age of 66.2 years at death) with rheumatoid arthritis (RA) was studied. Systemic vasculitis (SV) complicated RA in 36 (22.36%) of 161 cases (females 22, average age of 65.5 years; males 14, average age of 67.7 years at death);

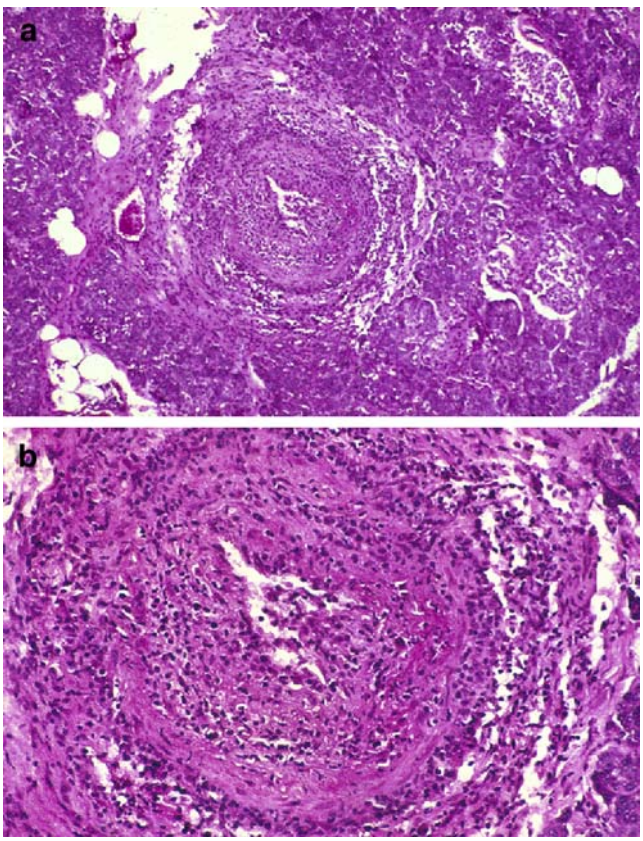


**Fig. 2** Pancreas. Small artery. Subacute vasculitis. (**a**) Same as Fig. 1a, ×125 (**b**) Same as Fig. 1a, ×200

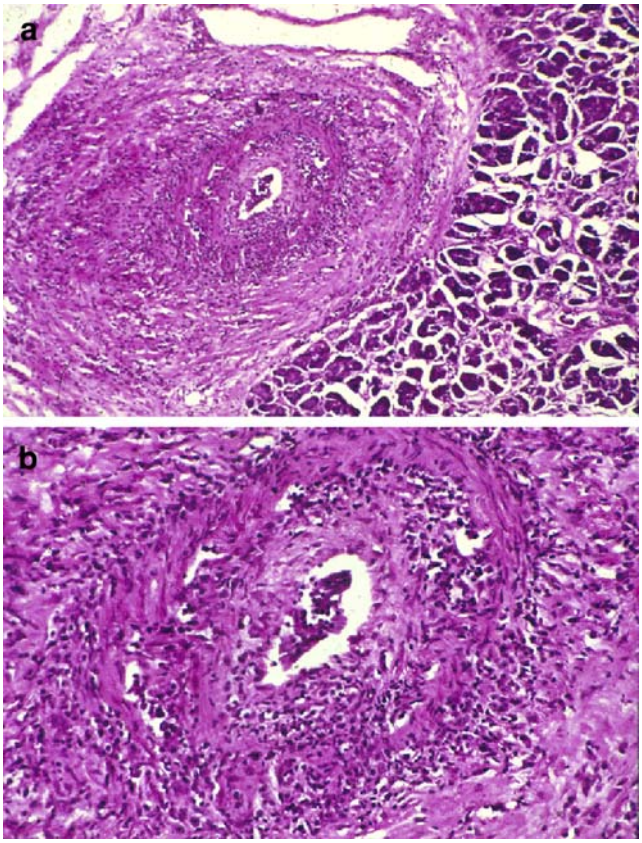


**Fig. 3** Pancreas. Small artery. Subchronic–chronic mixed, non-specific-vasculitis) (**a**) HE, ×50 (**b**) Same as (**a**), ×200

tissue samples of pancreas were available for histologic evaluation in 28 patients.

The tissue specimens were fixed in 8% formaldehyde solution at pH 7.6 and embedded in paraffin. Serial sections were cut and stained with haematoxylin-eosin, or the PAS-reaction, lightgreen-orcein, sirius red F3BA, or Congo red according to Romhányi [12], without alcoholic differentiation, and sealed with gum Arabic.

The amyloid deposits were determined histochemically by Congo red staining after performate pretreatment according to Romhányi [13], or by KMnO4 oxidation according to Wright [14, characterized according to Bély and Apáthy [15], or Bély [16], and viewed under polarized light.

Pancreatitis and vasculitis were determined and characterized histologically.

The link between vasculitis and pancreatitis was analyzed by $\chi$-test.

## Results

Arteritis and/or arteriolitis was observed in the pancreas in 10 of 28 cases (35.7 rel%). Three types of vasculitis were identified: non-specific (I) (Figs. 1, 2, 3, and 4a,b), fibrinoid necrotic (II) (Fig. 5a,b), and granulomatous (III). Different types of vasculitis existed simultaneously in different vessels or combined in the same vessel. Vessels of all sizes (arteriole, small artery, medium size artery, venule, small vein, and medium size vein) may be involved, with varying frequency (incidence) and severity (density) of vasculitis. The frequency of vasculitis in different blood vessels (according to the types of vasculitis) is summarized in Table 1. The severity of vasculitis in different blood vessels (according to the types of vasculitis) is summarized in Table 2. The vasculitis is usually severe in frequently involved vessels.

Four stages of vasculitis were recognized histologically: acute, subacute, subchronic, and chronic. Different stages of vasculitis existed simultaneously in different vessels or combined (segmental, sectorial) in the same vessel. The (absolute value) relative frequency of different stages in various vessels is summarized in Table 3.

Pancreatic arteritis and/or arteriolitis led to multiple, focal pancreatitis in 5 of 10 cases (17.85 rel%) (Fig. 1c, and Fig. 9). In one of these five cases of vasculogenic pancreatitis was so severe, that it may be regarded as a main contributory factor of circulatory insufficiency and death. The direct cause of death was multifocal myocardiocytolysis due to coronary arteriolitis.

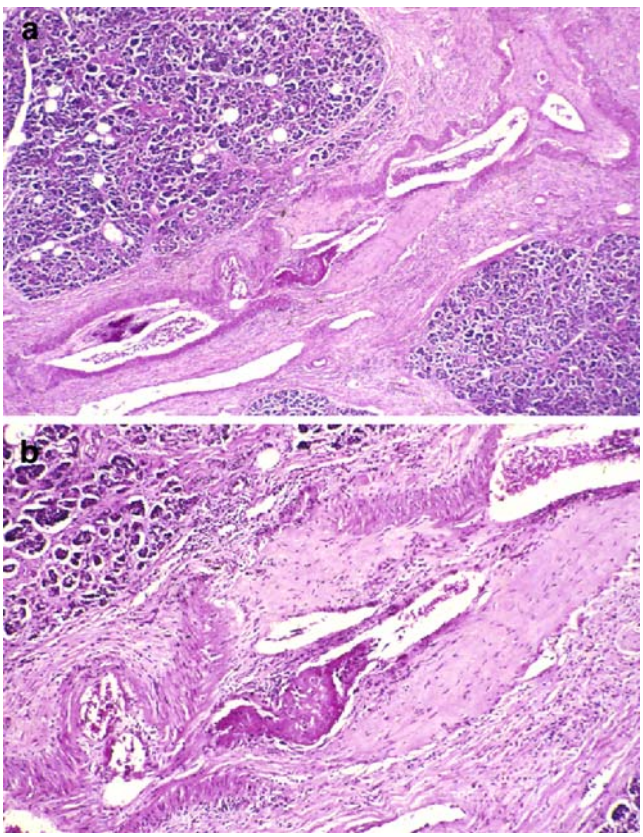


**Fig. 4** Pancreas. Medium size artery. Chronic recurrent, non-specific thrombo-vasculitis (**a**) HE, ×50 (**b**) Same as (**a**), ×125

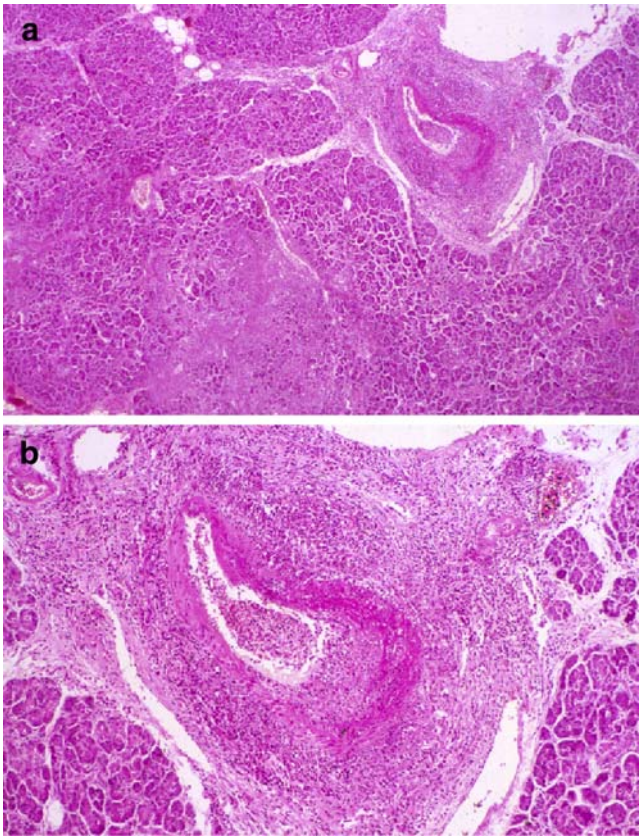


**Fig. 5** Pancreas. Small artery. Fibrinoid necrotis vasculitis. (**a**) HE, ×50 (**b**) HE, ×125

In a second case of severe vasculogenic pancreatitis, pancreatic arteritis and arteriolitis was accompanied by generalized secondary AA amyloidosis and uraemia, which was regarded to be direct cause of death (Figs. 6 and 7a,b).

There was a significant correlation between vasculitis and multifocal pancreatitis ($\chi^2$=7.81, p<0.005) (Fig. 8).

**Table 1** Frequency of vasculitis in the pancreas (according to the type of vasculitis in various vessels)

| Size of involved vessels | Types of vasculitis in absolute value | | | Total |
|---|---|---|---|---|
| | Non-specific | Fibrinoid necrotic | Granulomatous | |
| Arteriole (a) | 8 | 4 | 2 | 14 |
| Small artery (A) | 7 | 3 | 1 | 11 |
| Medium size artery (AA) | 3 | 0 | 0 | 3 |
| Venule (v) | 0 | 0 | 0 | 0 |
| Small vein (V) | 0 | 0 | 0 | 0 |
| Medium size vein (VV) | 0 | 0 | 0 | 0 |
| Total in absolute value | 18 | 7 | 28 | 28 |

**Table 2** Severity of vasculitis in the pancreas (according to the type of vasculitis in various vessels)

| Size of involved vessels | Types of vasculitis in absolute value | | | Total |
|---|---|---|---|---|
| | Non-specific | Fibrinoid necrotic | Granulomatous | |
| Arteriole (a) | 15 | 9 | 4 | 28 |
| Small artery (A) | 14 | 7 | 3 | 24 |
| Medium size artery (AA) | 4 | 0 | 0 | 4 |
| Venule (v) | 0 | 0 | 0 | 0 |
| Small vein (V) | 0 | 0 | 0 | 0 |
| Medium size vein (VV) | 0 | 0 | 0 | 0 |
| Total in absolute value | 33 | 16 | 7 | 56 |

## Discussion

The prevalence of vasculitis in the literature [17–31] is summarized in Table 4. The frequent involvement of arterioles and the small arteries is characteristic, but not pathognostic for RA. All size of the vessels may be involved in RA with SV.

Vasculitis is frequent and severe within the heart, skeletal muscle, and peripheral nerves. Skeletal muscle and peripheral nerve (for example peroneal nerve) biopsy are suggested for the histological diagnosis of vasculitis [29–31]. Different types of vasculitis may exist side by side in the same vessel or in different vessels.

Different stages of inflammation in the same vessel or in different vessels of a patient reflect the relapsing (recurrent) nature of immune vasculitis and their number corresponds to

**Table 3** Stages of vasculitis in the pancreas (in absolute value)

| Size of involved vessels | Acute | Subacute | Subchronic | Chronic | Total |
|---|---|---|---|---|---|
| Arteriole (a) | 2 | 8 | 12 | 10 | 32 |
| Small artery (A) | 1 | 6 | 9 | 11 | 27 |
| Medium size artery (AA) | 1 | 3 | 3 | 2 | 9 |
| Venule (v) | 0 | 0 | 0 | 0 | 0 |
| Small vein (V) | 0 | 0 | 0 | 0 | 0 |
| Medium size vein (VV) | 0 | 0 | 0 | 0 | 0 |
| Total in absolute value | 4 | 17 | 24 | 23 | 68 |

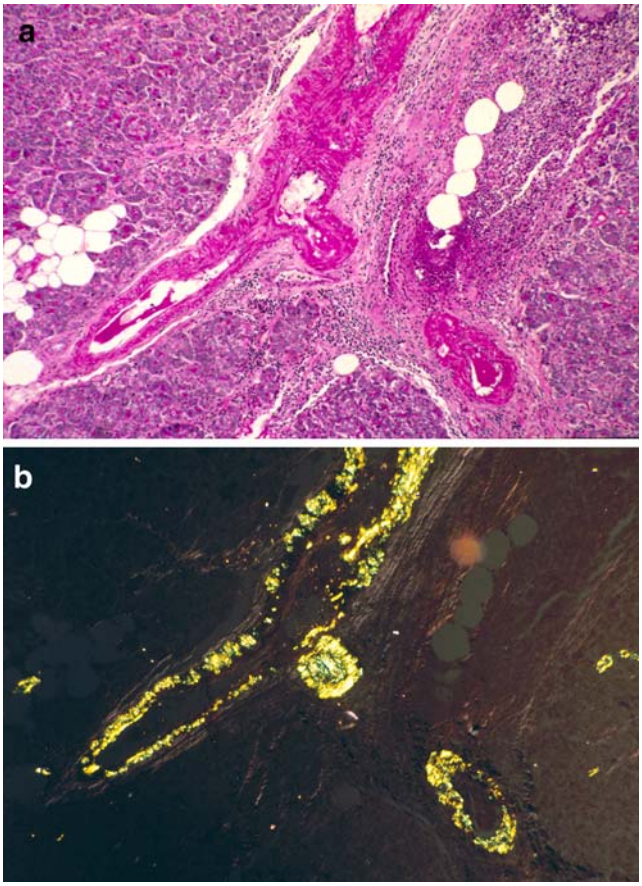


**Fig. 6** Pancreas. Small artery. AA amyloidosis and pancreatitis. (**a**) PAS ×50 (**b**) Congo red staining viewed under polarized light, Same as (**a**) ×50

the exacerbation of vasculitis in the past (subchronic–chronic stages) and more recently (acute–subacute stages) [29–31].

Vasculitis of the arterioles and small arteries can lead to local ischaemia and to regressive changes. This process is more or less widespread and multifocal, depending on the number of involved vessels (on the severity of vasculitis). Because of the recurrent nature of immune vasculitis the regressive changes accumulate within the involved organs in time and may lead to unexpected sudden death of the patient. Accumulated microinfarcts in the heart (vasculogenic multifocal myocardiocytolysis) may cause heart failure resistant to usual cardiac therapy or sudden death. Rheumatoid pneumonia represents a recurrent and migrant multifocal bronchopneumonia, which may be refractory to antibiotics. Multiple brain infarcts caused by SV may be accompanied by bizarre and inconstant neurological symptoms. Vasculogenic multifocal necrosis in the cortex of adrenal glands may depress their function [29–31].

Vasculitis is one of the main, and the most likely lethal, complication of *rheumatoid arthritis* to be missed clinically with high probability [31]. The mortality due to SV is determined by the location of the affected vessels (of involved organs), and not by the severity of SV. For example, mild SV involving the brain may be lethal; on the other hand, severe vasculitis of the skin may not be life threatening.

Arteritis and/or arteriolitis in the pancreas is less frequent and led directly never to death like vasculitis of other organs, e.g. in the myocardium, lungs, or brain. Vasculitis of the pancreatic arterioles and small arteries (branches of splenic artery, upper and lower gastroduodenal arteries) can lead to local ischaemia and to regressive changes in the pancreatic gland (Fig. 9). This process is more or less widespread and multifocal, depending on the number of involved vessels (Fig. 10). This vasculogenic multi(micro) focal necrosis of the pancreas is followed by reactive inflammation, depending on the stages of the pathological process. Because of the recurrent nature of vasculitis, with time the regressive changes accumulate within the pancreas and may contribute to an unexpected circulatory failure and sudden death of the patient.

Systemic vasculitis in rheumatoid arthritis was clinically recognised in about one fifth of lethal cases due to SV [31]. The clinical diagnosis of vasculitis was established mostly by visible skin involvement. It is important to recognize vasculitis in organs affected by RA, especially when the clinical complaints are recurrent.

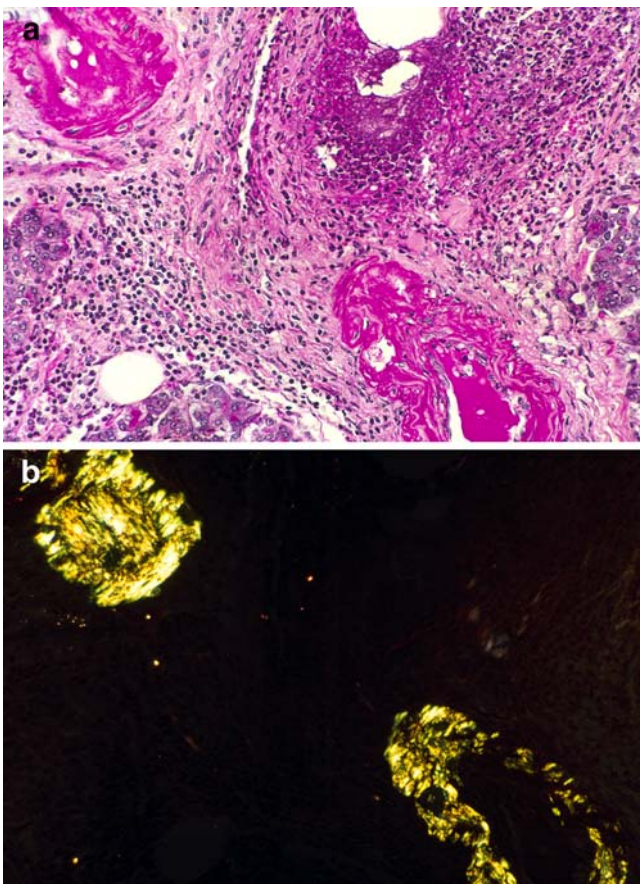


**Fig. 7** Pancreas. Small artery. AA amyloidosis and pancreatitis. (**a**) PAS x200 (**b**) Congo red staining viewed under polarized light, Same as Fig. 7a x200

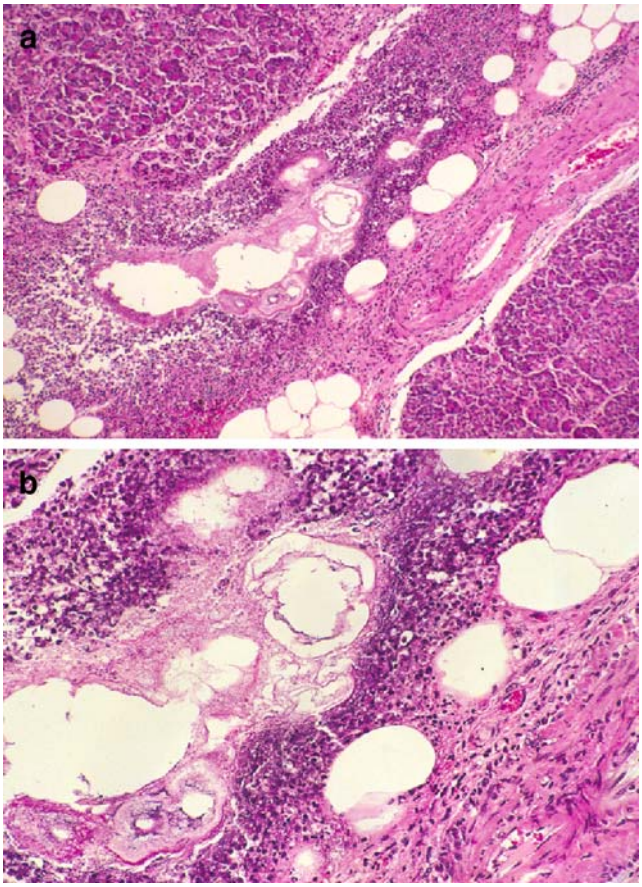


**Fig. 8** Pancreas. Vasculogenic microfocal pancreatitis. Surrounding area of Figs. 6 and 7a,b. (**a**) HE, x125 (**b**) Same as Fig. 9a, 200x

Pancreatitis due to vasculitis (vasculogenic, multifocal pancreatitis) was never recognized clinically. Vasculogenic microinfarcts in the pancreas may be clinically characterized by unexplained recurrent abdominal symptoms and spontaneous remissions which insidiously may lead to metabolic failure resistant to therapy.

**Glossary to Tables 1–3.**

| | |
|---|---|
| Vasculitis | was defined as non-specific inflammation (I) and/or fibrinoid necrosis (II), or granulomatous transformation (III) of blood vessels in different (acute, subacute, subchronic, chronic) stages of a pathological process. |
| Arteriole (a) | no internal or external elastic membrane, <500 micrometers in diameter. |
| Small artery (A) | only internal elastic membrane present, vessels 500–1000 μm in diameter. |
| Medium size artery (AA) | internal and external elastic membrane are present – vessel >1000 micrometers in diameter. |
| Venule (v), small vein (V), medium size vein (VV) | are accompanying (a), (A), or (AA). |

(I)

| | |
|---|---|
| Non-specific vasculitis | is characterized by non-specific leuko-, lympho-, plasmacytic infiltration. Fibrinoid necrosis is minimal or absent. |

(II)

| | |
|---|---|
| Fibrinoid necrotic vasculitis | is dominated by fibrinoid necrosis (by fresh or old (dense) fibrinoid deposits in the vessel wall). |

**Table 4** Prevalence of systemic vasculitis in autopsy material of rheumatoid arthritis

| Authors | Reference Published in the year | Autopsy *n* = | Prevalence of vasculitis N (%) | Mortality of vasculitis N (%) |
|---|---|---|---|---|
| Cruickshank | 1954 (17) | 72 | 18 (25) | ND |
| Sinclair and Cruickshank | 1956 (18) | 16 | 9 (56.3) | ND |
| Cruickshank[a] | 1958 (19) | 100 | 20[a] (20) | ND |
| Lebowitz | 1963 (20) | 62 | 6 (10) | ND |
| Sokoloff | 1964 (21) | 19 | 2 (10.5) | ND |
| Karten[b] | 1969 (22) | 102 | 6[b] (6) | ND |
| Gardner | 1972 (23) | 142 | 7 (4.9) | ND |
| Davis and Engleman | 1974 (24) | 62 | 6 (10) | ND |
| Eulderink | 1976 (25) | 111 | ND | 7 (6.3) |
| Albada-Kuipers et al. | 1986 (26) | 173 | 17 (10) | ND |
| Boers et al. | 1987 (27) | 132 | 18 (13.6) | ND |
| Suzuki et al. | 1994 (28) | 81 | 25 (30.8) | ND |
| Bély and Apáthy | 1994 (29–30) | 161 | 36 (22..4) | 19 (11.8) |
| Bély and Apáthy | 2005 (31) | 234 | 51 (21.8) | 23 (9.8) |

*ND* no data
[a] Coronaritis
[b] 102 patients with RA – partially autopsied (Karten)

**Fig. 9** Pancreas. Vasculogenic microfocal pancreatitis

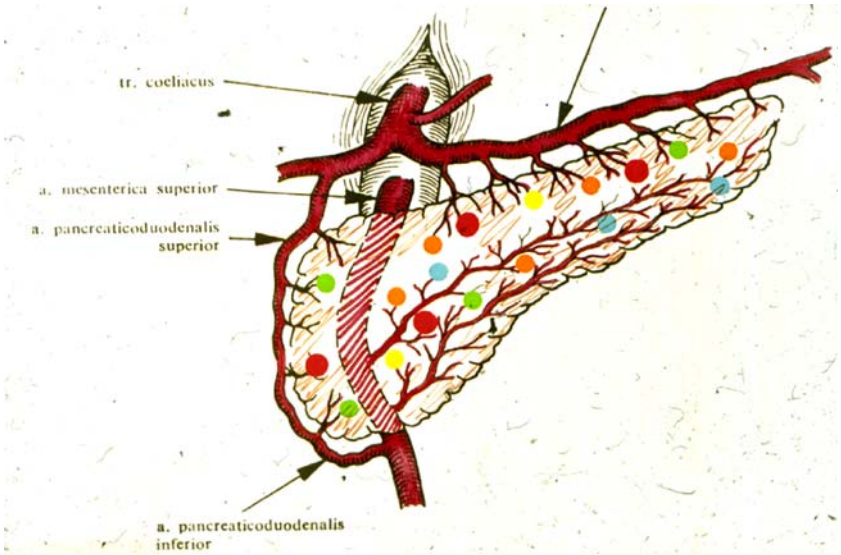


**Fig. 10** Schematic drawing of pathogenesis of multiple microfocal pancreatitis

(III)

| | |
|---|---|
| Granulomatous vasculitis | the original structure of the vessel wall disappears, and granulomatous, more or less cellular infiltrate replaces it. In the early stage of vasculitis the infiltration is dominated by histiocytes, with, or without multinucleated giant cells, and later by fibroblasts. In the end stage of granulomatous vasculitis the vessel wall becomes less cellular and more fibrotic. |
| Acute stage of vasculitis | is characterized by edema, leukocytic infiltration with or without fresh fibrinoid necrosis of the vessel wall |
| Subacute-Subchronic stage | is characterized by lymphocytic, plasmacytic infiltration, with or without old fibrin deposits in the vessel wall. Histiocytes, multinucleated giant cells, fibroblasts may be present. There is no hyalinization, or fibrosis. |
| Chronic | structural changes of the vessel wall are characterized by fibrosis and/or hyaline degeneration of the vessel wall. Inflammatory cellular infiltration is sporadic or absent, |

Semi-objective score system of "Severity"

| | |
|---|---|
| "0" | no vasculitis |
| "1" | Sporadically, scattered located blood vessels with vasculitis |
| "2" | less than 5 involved blood vessels per microscopic field with a x20 objective. (Remark: in case of AA, or VV it corresponds to the absolute number of involved medium size vessels of a tissue sample, e.g. less than five medium size vessels/tissue sample) |
| "3" | 5, or more involved blood vessels/microscopic field with a ×20 objective (Remark: in case of AA, or VV it corresponds to the absolute number of involved medium size vessels of tissue a sample, e.g. 5, or more than five medium size vessels/tissue sample) |

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