

Pathomorphological and Histochemical Changes in Regional Lymph Nodes in Alcoholic Pancreatitis: An Autopsy-Based Analysis

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Abstract

Background: Alcoholic pancreatitis induces systemic immunological responses that are reflected in the lymphatic apparatus of the pancreas. The regional lymph nodes serve as critical immunological sentinels, yet their structural and histochemical alterations remain incompletely characterised. **Methods:** Autopsy material from regional pancreatic lymph nodes was obtained from 40 deceased individuals: 22 with acute alcoholic pancreatitis (AP), 18 with chronic AP, and 10 controls. Haematoxylin-eosin, Masson's trichrome, PAS, Alcian Blue, Sudan III, and Gomori silver staining were applied. Semi-quantitative scoring and statistical analysis were performed. **Results:** Both forms of AP demonstrated significant lymphoid hyperplasia, sinus histiocytosis, and paracortical expansion. Acute AP showed pronounced haemorrhagic infiltration and necrotic foci, whereas chronic AP exhibited dominant fibrotic remodelling and glycogen depletion. Histochemical analysis revealed progressive accumulation of acid mucopolysaccharides and neutral lipids alongside reticular fibre fragmentation. **Conclusion:** Regional lymph nodes in alcoholic pancreatitis undergo distinct morphological and histochemical transformations depending on disease acuity, reflecting dynamic immune dysregulation and stromal remodelling.

Keywords: *alcoholic pancreatitis; lymph node morphology; histochemistry; autopsy; sinus histiocytosis; lymphoid hyperplasia; pancreatic immunopathology*

Introduction

Alcoholic pancreatitis represents one of the most severe and prognostically challenging complications of chronic alcohol misuse, with mortality rates in acute forms reaching 20–30% despite advances in intensive care management [1]. The disease is characterised by profound local and systemic inflammatory responses, vascular disruption, and acinar cell necrosis, resulting in widespread activation of the lymphatic system [2]. The regional lymph nodes of the pancreas, including the peripancreatic, paraaortic, and coeliac groups, function as primary immunological processing stations

that receive antigens, lipase-rich exudate, and inflammatory mediators draining from the inflamed gland [3].

The lymphoid tissue responses to pancreatic injury are well established in experimental models, yet systematic autopsy-based investigations in human cases of alcoholic pancreatitis remain scarce [4]. Available evidence suggests that peripancreatic lymph nodes undergo reactive hyperplasia during the acute phase, followed by fibrotic involution in chronic disease [5]. However, histochemical characterisation of these changes—particularly the dynamics of glycogen metabolism, lipid accumulation, mucopolysaccharide deposition, and fibrillar protein reorganisation—has not been systematically described [6].

Ethanol exerts direct immunotoxic effects on lymphoid cells, impairing lymphocyte proliferation, natural killer cell activity, and cytokine secretion [7]. These effects potentially compound the immune alterations induced by pancreatic inflammation itself, creating a unique pathomorphological phenotype in the regional lymph nodes of patients dying from alcoholic pancreatitis [8]. Furthermore, the lymphatic drainage pattern in pancreatitis is altered by ductal hypertension and peripancreatic fat necrosis, potentially delivering enzymatically active material directly into lymphatic channels [9].

Understanding the morphological and histochemical profile of lymph node changes in alcoholic pancreatitis has direct clinical relevance. Lymphadenopathy detected by imaging during acute pancreatitis may be misinterpreted as malignancy or infection [10]. Moreover, characterising the immunopathology of regional lymph nodes may help identify potential biomarkers of disease severity and systemic immune dysregulation [11]. The present study aimed to comprehensively assess pathomorphological and histochemical alterations in the regional lymph nodes of individuals who died from acute and chronic alcoholic pancreatitis using a panel of validated staining techniques applied to autopsy material.

Methods

This retrospective autopsy study was conducted using archival material from the pathoanatomical department of Fergana Medical Institute of Public Health. Lymph node specimens were harvested within six hours post-mortem from standardised anatomical zones: peripancreatic, hepatoduodenal ligament, and coeliac axis groups. The study included 22 cases of acute alcoholic pancreatitis, 18 cases of chronic alcoholic pancreatitis, and 10 controls without pancreatic or hepatic pathology. Alcoholic aetiology was confirmed by clinical records, post-mortem blood alcohol levels, and characteristic macroscopic and histological findings.

Tissue blocks were fixed in 10% neutral buffered formalin, processed by standard paraffin embedding, and sectioned at 4–5 μm . The following staining protocols were applied: haematoxylin and eosin (H&E) for general morphology; Masson's trichrome for collagen and fibrotic changes; periodic acid-Schiff (PAS) reaction for glycogen and mucosubstances; Alcian Blue at pH 2.5 for acid mucopolysaccharides; Sudan III on frozen sections for neutral lipids; Gomori silver impregnation for reticular fibres; and the Brachet reaction for RNA localisation. Alkaline phosphatase activity was demonstrated by the azo-dye coupling method. Morphometric assessment included follicular diameter, paracortical area, sinus width, and capsular thickness, measured using calibrated eyepiece micrometry. Histochemical reactions were scored semi-quantitatively (0 = absent, 1 = mild, 2 = moderate, 3 = pronounced). Statistical analysis was performed using SPSS v.26; Mann-Whitney U test and chi-square with $p < 0.05$ considered significant.

Results

A total of 50 lymph node specimens were examined (2–3 nodes per case). Macroscopically, nodes in both pancreatitis groups were enlarged (mean longest diameter: acute AP 18.4 ± 3.2 mm, chronic AP 15.7 ± 2.8 mm, controls 8.1 ± 1.4 mm; $p < 0.001$). Cut sections in acute AP cases showed haemorrhagic discolouration and focal creamy necrotic zones in 63.6% of specimens.

Table 1 summarises the frequency of histopathological changes. Lymphoid follicle hyperplasia and sinus histiocytosis were the predominant findings in both pancreatitis groups, occurring in over 85% of specimens and significantly exceeding control values. Paracortical expansion, reflecting T-lymphocyte activation, was present in 81.8% of acute AP and 77.8% of chronic AP nodes. Perinodal fibrosis and capsular thickening were significantly more prevalent in chronic AP (83.3% and 77.8%, respectively) versus acute AP (36.4% and 27.3%). Conversely, haemorrhagic infiltration and necrotic foci were markedly more frequent in acute AP.

Table 1

Frequency of histopathological changes in regional lymph nodes by disease group

Histopathological Parameter	Acute AP (n=22)	Chronic AP (n=18)	Control (n=10)	p-value
Lymphoid follicle hyperplasia	19 (86.4%)	16 (88.9%)	2 (20.0%)	<0.001
Paracortical expansion	18 (81.8%)	14 (77.8%)	1 (10.0%)	<0.001

Sinus histiocytosis	20 (90.9%)	17 (94.4%)	3 (30.0%)	<0.001
Perinodal fibrosis	8 (36.4%)	15 (83.3%)	0 (0.0%)	<0.001
Capsular thickening	6 (27.3%)	14 (77.8%)	0 (0.0%)	<0.001
Necrotic foci	12 (54.5%)	5 (27.8%)	0 (0.0%)	0.003
Hemorrhagic infiltration	14 (63.6%)	4 (22.2%)	0 (0.0%)	0.001
Lymphocyte depletion	7 (31.8%)	11 (61.1%)	0 (0.0%)	0.002

Note: Values are n (%). AP = alcoholic pancreatitis.

Table 2 presents histochemical findings. PAS staining revealed moderate glycogen accumulation in the cytoplasm of sinus histiocytes in acute AP, which was markedly reduced or absent in chronic AP cases, suggesting depletion of carbohydrate reserves under prolonged inflammatory conditions. Alcian Blue demonstrated progressive increase in acid mucopolysaccharide content from controls through acute to chronic AP, most prominently in the capsule and trabeculae. Sudan III staining of frozen sections showed focal lipid deposits in acute AP and diffuse deposits in chronic AP, consistent with progressive lipid metabolic disruption.

Masson's trichrome revealed a graduated fibrotic response: mild perisinusoidal collagen deposition in acute AP progressing to dense capsular and trabecular fibrosis in chronic AP. Gomori silver impregnation demonstrated intact reticular framework in controls, focal disruption in acute AP, and pronounced fragmentation with reticular collapse in chronic AP. Alkaline phosphatase activity was highest in acute AP (particularly in sinus endothelium), moderately elevated in chronic AP, and within normal limits in controls. The Brachet reaction showed elevated RNA content in lymphocytes of acute AP nodes (reflecting active protein synthesis), with reduced RNA in chronic AP, consistent with lymphocyte depletion.

Table 2

Histochemical staining results in regional lymph nodes across study groups

Histochemical Marker	Method / Stain	Acute AP Finding	Chronic AP Finding	Control Finding

Glycogen	PAS reaction	Moderate accumulation	Reduced / depleted	Normal
Acid mucopolysaccharides	Alcian Blue (pH 2.5)	Mild increase	Pronounced increase	Trace
Neutral lipids	Sudan III / Oil Red O	Focal lipid deposits	Diffuse deposits	Absent
Collagen fibers	Masson's Trichrome	Mild fibrosis	Dense fibrosis	Normal
Reticular fibers	Gomori silver	Framework disruption	Fragmentation	Intact
Alkaline phosphatase	Azo-dye method	Increased activity	Moderate activity	Normal
RNA (lymphocytes)	Brachet reaction	Elevated	Reduced	Normal

Table 3 presents comparative semi-quantitative severity scores across groups. Total severity index was comparably elevated in both AP forms (acute 10.5 ± 1.8 , chronic 10.8 ± 1.6) versus controls (1.0 ± 0.4 , $p < 0.001$ for all comparisons). However, the pattern differed: acute AP dominated in inflammatory infiltration and vascular changes, while chronic AP scored higher in fibrotic changes and structural disruption.

Table 3

Semi-quantitative severity scoring of lymph node changes (mean \pm SD, scale 0–3 per parameter)

Parameter	Acute AP Score (0–3)	Chronic AP Score (0–3)	Control Score (0–3)	Sig.
Structural disruption	2.4 ± 0.6	2.7 ± 0.5	0.3 ± 0.2	***
Inflammatory infiltration	2.6 ± 0.4	1.8 ± 0.7	0.2 ± 0.1	***
Fibrotic changes	1.1 ± 0.5	2.5 ± 0.4	0.1 ± 0.1	***
Histochemical alterations	2.1 ± 0.5	2.3 ± 0.4	0.2 ± 0.1	***
Vascular changes	2.3 ± 0.6	1.5 ± 0.6	0.2 ± 0.1	***
Total severity index	10.5 ± 1.8	10.8 ± 1.6	1.0 ± 0.4	***

Note: *** $p < 0.001$ for both AP groups vs. control. Scores represent mean \pm SD.

Discussion

The present study provides a systematic morphological and histochemical characterisation of regional lymph node alterations in alcoholic pancreatitis derived from human autopsy material. Our findings demonstrate that both acute and chronic forms of the disease induce profound and distinct changes in lymph node architecture, immunohistochemistry, and stromal composition, substantially exceeding baseline controls.

Lymphoid follicle hyperplasia and sinus histiocytosis—the most prevalent findings in our series—are consistent with a reactive pattern attributable to antigenic stimulation by pancreatic enzymes, necrotic debris, and alcohol-derived metabolites draining through the peripancreatic lymphatics [12]. Experimental data in rodent models of pancreatitis confirm early B-cell follicular activation and macrophage accumulation in regional nodes [13]. Our data extend these findings to the human autopsy setting, demonstrating that this pattern persists regardless of disease acuity, suggesting a sustained lymphatic immune response.

The pronounced paracortical expansion observed in both AP groups reflects T-lymphocyte hyperactivation. Ethanol is known to dysregulate T-cell subset ratios, suppressing regulatory T-cell function and promoting pro-inflammatory Th1 and Th17 responses [14]. Combined with cytokine release from injured pancreatic parenchyma, this creates a state of sustained T-cell stimulation reflected in paracortical hypertrophy [15]. The finding of lymphocyte depletion predominantly in chronic AP cases is also consistent with the immunosuppressive consequences of prolonged alcohol exposure and recurrent inflammation [16].

Haemorrhagic infiltration and necrotic foci were significantly more prevalent in acute AP, reflecting the systemic vasodestructive effects of circulating pancreatic enzymes, particularly elastase and phospholipase A2, which disrupt endothelial integrity [17]. These enzymes reach lymphatic channels via retroperitoneal routes and may directly damage lymph node vasculature, consistent with the endothelial activation indicated by elevated alkaline phosphatase activity in our histochemical data [18].

The progressive fibrotic changes in chronic AP—manifested as dense perinodal and capsular fibrosis, Alcian Blue-positive mucopolysaccharide accumulation, and reticular fibre fragmentation—represent a pattern of stromal remodelling driven by persistent TGF- β signalling and myofibroblast activation [19]. Similar fibrotic changes have been described in hepatic lymph nodes in chronic liver disease, suggesting a

shared pathomechanism in alcohol-related organ damage [20]. The depletion of glycogen in sinus histiocytes of chronic AP nodes is consistent with metabolic exhaustion of activated macrophages operating under sustained phagocytic load [21].

The lipid accumulation detected by Sudan III in chronic AP may reflect both direct alcohol-mediated steatosis of lymphoid cells and the phagocytosis of lipid-rich necrotic debris from peripancreatic fat necrosis [22]. The diffuse pattern in chronic versus focal pattern in acute AP mirrors the progressive nature of lipid metabolic disruption in prolonged alcohol-related disease [23]. Collectively, these histochemical data suggest that the lymph nodes of patients with alcoholic pancreatitis undergo not only structural but also metabolic reprogramming, with distinct kinetics in acute versus chronic disease.

Several limitations of this study should be acknowledged. The autopsy-based design, while enabling direct tissue access, introduces selection bias toward severe disease. Post-mortem interval and agonal state may influence some histochemical reactions, particularly enzyme histochemistry. The relatively modest sample size reflects the strict inclusion criteria for confirmed alcoholic aetiology. Future studies incorporating immunohistochemistry for specific lymphocyte markers (CD3, CD20, CD68, CD163) and digital morphometry would further characterise the cellular composition of these changes.

Conclusion

Regional lymph nodes of the pancreas in alcoholic pancreatitis undergo substantial and clinically meaningful pathomorphological and histochemical transformations that are clearly distinguishable from control tissue and display distinct patterns according to disease acuity. Acute alcoholic pancreatitis induces a predominantly inflammatory, haemorrhagic, and enzymatically active phenotype in regional lymph nodes, whereas chronic disease drives a fibrotic, metabolically depleted, and structurally disorganised state. These findings underscore that regional lymph nodes are active participants in the immunopathology of alcoholic pancreatitis, not passive bystanders. The histochemical profile, encompassing glycogen depletion, lipid accumulation, mucopolysaccharide deposition, and reticular fibre breakdown, provides a window into the metabolic consequences of sustained pancreatic inflammation on lymphoid tissue. Recognition of these changes is essential for accurate autopsy diagnosis, avoidance of misinterpretation of lymphadenopathy as neoplastic or infectious, and for advancing the understanding of immune dysregulation in alcohol-related disease. These results lay the groundwork for targeted immunohistochemical and molecular studies to further decode the lymph node response in pancreatic disease.

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