Virchows Arch [Pathol Anat] (1981) 390:127–138

Virchows Archiv A

Original Articles

Mesenteric Lymphadenitis Due to *Yersinia enterocolitica*

R.F.M. Schapers[1,4], Renate Reif[2], K. Lennert[1], and W. Knapp[3]

[1] Lymph Node Registry, Institute of Pathology, Christian Albrechts University, D-2300 Kiel, Federal Republic of Germany
[2] Department of Pathology, Gov. Hospital "Assaf Harofe", Zrifin, Israel
[3] Department of Clinical Microbiology and Infection-Hygiene, University of Erlangen-Nürnberg, D-8520 Erlangen, Federal Republic of Germany
[4] Present address: Pathologisch Anatomisch Laboratorium (S.P.L.), 5900 BX Venlo, The Netherlands

Summary. The histopathological diagnosis of *Yersinia enterocolitica* infections in mesenteric lymph nodes is described on the basis of biopsy material from 14 cases collected at the Lymph Node Registry in Kiel. In all cases, the aetiological diagnosis was verified by demonstrating significant antibody titres in serological tests and, in two cases, by isolating *Yersinia enterocolitica* from faeces. The mesenteric lymph nodes showed a rather specific histological picture. In all cases, the capsule was thickened by oedema and stained metachromatically. The cortical and paracortical pulp was always hyperplastic owing to an increase in the number of immunoblasts, plasmablasts, and plasma cells. The sinuses were dilatated and filled with intensely basophilic cells that varied in size from small to large (plasmacytoid cells and precursors). Small, or relatively large accumulations of "immature histiocytes" (transformed lymphocytes) were seen in the sinuses in about two thirds of the cases. Occasionally, there were small foci of small histiocytes (emigrated monocytes) in the cortical pulp. An abscess similar to the abscesses found in abscess-forming reticulocytic lymphadenitis due to *Yersinia pseudotuberculosis* was evident in only one case.

The differential diagnosis is also discussed. The diseases to be considered are mesenteric lymphadenitis due to *Yersinia pseudotuberculosis* or salmonella infection, and nonspecific mesenteric lymphadenitis.

Key words: *Yersinia enterocolitica* – Mesenteric lymphadenitis – Histopathology

Introduction

The name "*Yersinia enterocolitica*" was proposed by Frederiksen (1964). Previously, this group of bacteria had been described under many different names,

Offprint requests to: Prof. Dr. K. Lennert, Institute of Pathology, University of Kiel, Hospitalstr. 42, D-2300 Kiel 1, Federal Republic of Germany

0340-1227/81/0390/0127/$02.40

e.g., *Bacterium enterocoliticum* (Schleifstein and Coleman 1939), *Pasteurella X* (Daniels 1963), and *germe X* (Mollaret and Destombes 1964). The new species within the genus *Yersinia* was placed in the family of Enterobacteriaceae. *Yersinia enterocolitica* infections can be diagnosed by isolating the organisms from cultures on selective or nonselective media or by demonstrating specific antibodies.

The first *Yersinia enterocolitica* infections in man were described in Europe in 1949 by Hässig et al. as infections with *Pasteurella pseudotuberculosis* (cf., Knapp and Thal 1963). Since that time, there have been many reports on the bacteriological and serological diagnosis of *Yersinia enterocolitica* infections (e.g., Mollaret 1966; Winblad 1968; Nilehn 1969; Wauters 1970; Ahvonen 1972; Wauters et al. 1972; Knapp et al. 1973). It has been shown that well-defined strains of *Yersinia enterocolitica* are important pathogens in man, causing acute abdominal disease (sometimes with symptoms of acute appendicitis, mesenteric lymphadenitis, or acute terminal ileitis), enteritis, enterocolitis, arthritis, erythema nodosum, sepsis, Reiter's syndrome, meningitis, or cutaneous lesions (e.g., Mollaret et al. 1964; Mollaret 1966; Winblad et al. 1966a, b; Nilehn et al. 1968; Winblad 1969; Vandepitte et al. 1970; Ahvonen 1972; Knapp et al. 1973; Leino and Kalliomäki 1974; Erikson and Olcén 1975; Laitinen et al. 1975; Aho et al. 1976; Spira and Kabins 1976; Knapp 1977). It has been suggested that the *porte d'entrée* of the infection is oral. Possible sources of infection are infected meat, drinking water, and other food. Sick animals may also be a source of infection, but, as yet, the epidemioloy of *Yersinia enterocolitica* infections has not been clarified.

Only very few authors have described the histopathology of mesenteric lymph nodes from patients with *Yersinia enterocolitica* infections (Mollaret et al. 1964; Winblad et al. 1966b; Ahlquist et al. 1971; Bradford et al. 1974; Vantrappen et al. 1977). These investigators usually described the lesions as nonspecific lymphadenitis. Only Winblad et al. (1966b) mentioned small collections of leukocytes within germinal centres in lymphoid tissue. He called the collections "microabscesses".

Material and Methods

In this paper, we discuss the histology of mesenteric lymph nodes from 14 patients; *Yersiniae enterocoliticae* were isolated from the faeces of two patients, and significant titres of specific antibodies were detected in all cases. Five of the patients were females, and nine were males. They were from 11 to 42 years old (mean age 20.8 years). Five of the 14 cases were diagnosed in the month of January; the others were evenly distributed throughout the year.

Agglutination tests were done with *Yersinia enterocolitica* strain Ye 75^{I} and Ye 373^{V}. These strains belong to the serogroups I and V, respectively, defined by Knapp and Thal (1973) or to groups 3 and 9, respectively, according to Winblad (1968). Cross reactions between *Yersinia enterocolitica* O-group V and the three *Brucella* species *Brucella abortus, Brucella melitensis,* and *Brucella suis* have to be mentioned. Our methods for the detection of antibodies and for the isolation of *Yersinia enterocolitica* from faeces have been described previously (Knapp et al. 1973).

The histological specimens were fixed in 10% phosphate-buffered neutral formalin, embedded in Paraplast®, and sectioned at 4 µm. All slides were routinely stained with haematoxylin and eosin, Giemsa, Bielschowsky-Gomori, and PAS.

Results

Serological and Bacteriological Findings

Antibodies against *Yersinia enterocolitica* were demonstrated in serum from all 14 patients. One sample was examined in six cases, two samples were examined in six cases, and three samples were examined in two cases. Antibody titres against O- or living antigen were demonstrated in sera diluted 1:20 to 1:320 or diluted 1:80 to 1:20,480. There was usually a significant decrease in the titre when antibody determinations were repeated. Details are shown in Table 1. Antibodies against *Yersinia enterocolitica* O-group I were detected in serum from nine patients. Antibodies against *Yersinia enterocolitica* O-group V and *Brucella abortus* could be demonstrated in serum from five patients. These five patients showed no clinical symptoms of brucellosis. Stool cultures were obtained from only four patients, and the organism could be isolated from two of the cultures.

Table 1. Serological and bacteriological findings

Patient	Age	Sex	Registry No.		Antibody titre against Yersinia enterocolitica antigen		Brucella abortus antigen	Stool examination
			Kiel	Erlangen	O group	O-/OH=living antigen		
1. P.S.	29	M	R 1455/71	Y 115/71 (20.10.)	V	160/640	160	ND[a]
				Y 136/71 (8.11.)	V	80/160	320	ND
2. K.E.	32	F	R 1579/72	Y 306/Cr/72 (21.8.)	V	160/320	640	+
				Y 332/Cr/72 (11.9.)	V	−/80	80	ND
3. M.H.	11	F	R 1313/73	Y 366/73 (26.6.)	V	160/320	320	ND
4. K.H.	42	M	R 1353/73	Y 386/73 (4.7.)	I	160/5120	ND	ND
				Y 398/73 (9.7.)	I	320/2560	ND	−
				Y 544/73 (11.10.)		−/40	ND	ND
5. G.H.	12	M	R 250/74	Y 143/74 (12.2.)	I	160/320	ND	ND
				Y 177/74 (19.2.)	I	80/320	ND	+
6. A.F.	15	F	R 2996/74	Y 1147/74 (30.11.)	I	>160/>2560	ND	ND
				Y 11/75 (8.1.)	I	80/320	ND	
7. C.L.	26	M	R 35/75	Y 44/75 (16.1.)	V	80/320	>640	ND
				Y 109/75 (23.1.)	V	320/160	80	−
8. T.A.	13	M	R 3882/75	Y 37/76 (8.1.)	I	320/1280	ND	ND
				Y 202/76 (2.2.)	I	40/640	ND	ND
				Y 791/76 (24.3.)	I	−/20	ND	ND
9. C.V.	26	F	R 58/76	Y 161/76 (28.1.)	I	20/160	ND	ND
10. B.R.	17	F	R 1359/76	Y 844/76 (6.5.)	V	20/80	160	ND
				Y 2086/76 (24.11.)	V	−/−	−	
11. H.M.	18	M	R 1689/76	Y 1050/76 (5.6.)	I	640/1280	ND	ND
12. K.N.	16	M	R 3854/76	Y 1984/76 (8.11.)	I	2560/20.480	ND	ND
13. T.B.	14	M	R 100/77	Y 118/77 (14.1.)	I	320/2560	ND	ND
14. N.K.	20	M	R 2013/77	Y 1452/77 (15.6.)	I	80/160	ND	ND

[a] ND=not done. −=negative. +=isolation of *Yersinia enterocolitica* group I or V

Table 2. Histopathological findings

Case	Biopsy No.	Oedema	Capsule metachromasia	Inflammation	Germinal centres	Cells in sinuses	Pulp infiltration	Other findings
1.	R 1455/71	Slight	Marked	Marked	Small	Moderate	Moderate	–
2.	R 1579/72	Slight	Marked	Marked	Large	Many	Marked	–
3.	R 1313/73	Slight	Marked	Moderate	Large	Many	Marked	–
4.	R 1353/73	Moderate	Minimal	Marked	Small	Many (also erythrocytes)	Marked (also neutrophils)	–
5.	R 250/74	Moderate	Minimal	Minimal	Small	Many	Marked	Histiocytic foci in pulp with central necrosis
6.	R 2996/74	Moderate	Moderate	Moderate	Medium-sized	Moderate	Moderate	–
7.	R 35/75	Minimal	Marked	Marked	Large	Many	Slight/moderate	Histiocytic foci with small abscess and eosinophils
8.	R 3882/75	Moderate	Moderate	Minimal/ moderate	Medium-sized	Moderate	Moderate	Some Sternberg-Reed-like cells in pulp
9.	R 58/76	Minimal	Moderate	Moderate	Large	Few/moderate	Slight/moderate	Histiocytic foci with epithelioid cells
10.	R 1359/76	Minimal	Moderate	Moderate	Small	Few/moderate (also neutrophils)	Slight/moderate (also neutrophils)	–
11.	R 1689/76	Moderate	Moderate	Moderate		Moderate	Moderate	–
12.	R 3854/76	Moderate	Moderate	Marked	Medium-sized	Few	Marked	–
13.	R 100/77	Marked	Moderate	Marked	Medium-sized	Many	Marked	–
14.	R 2013/77	Moderate	Marked	Moderate/ marked	Medium-sized	Many	Marked (also neutrophils)	–

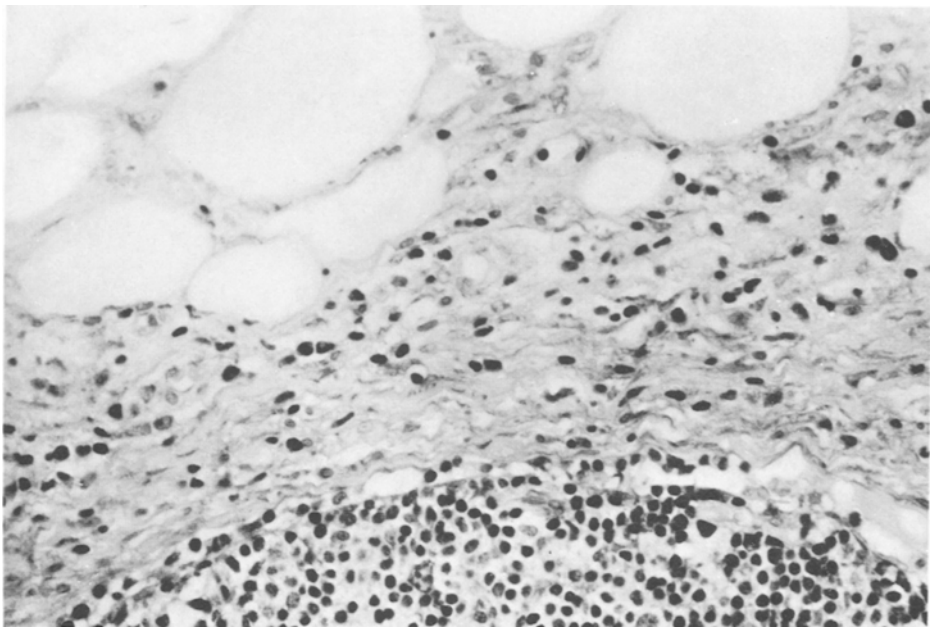

Fig. 1. Thickening of the capsule caused by oedema and by infiltration by lymphocytes and plasma cells. Case 13, Giemsa, ×350

Pathological Findings

Localisation. All of the lymph nodes were mesenteric and came especially from the ileocaecal region.

Macroscopical Findings. The lymph nodes were not larger than 1.5 cm in diameter and were soft in consistency. No necrotic areas were seen on cut section.

Histology (Table 2). One of the first discernible changes in all lymph nodes was thickening of the capsule (Fig. 1). The thickening occurred in varying degrees and was found to be caused, in part, by oedema and by an absolute increase in fibres and inflammatory infiltration. The infiltration was most pronounced in perivascular areas (Fig. 2). The infiltrating cells were mostly small lymphocytes, but also included eosinophils, plasma cells, a few mast cells, a few histiocytes, and occasionally some neutrophils. In some of the specimens, a few immunoblasts and plasmablasts could also be found. Giemsa staining disclosed metachromasia of the capsule.

All these changes were also evident in the trabeculae extending into the node. It might be easier to recognize the changes in the trabeculae in cases in which little capsular tissue is received.

The perilymphadenitis described here was present in all 14 cases. There were merely differences in degree and in cellular composition of the infiltrates from case to case.

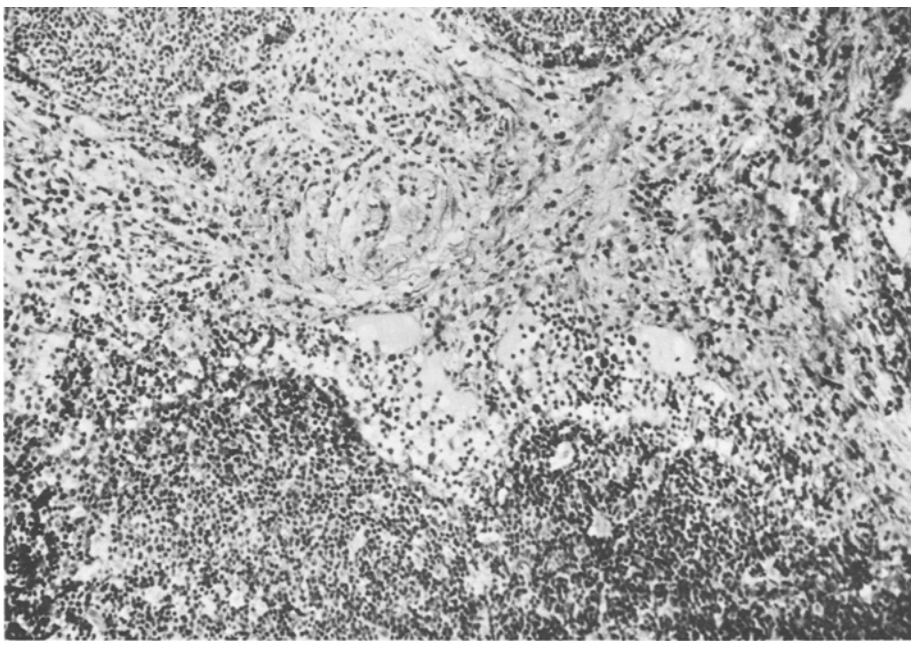

Fig. 2. Perivascular oedema and cellular infiltration of the connective tissue of the hilus. Case 13, Giemsa, ×350

The lymph node architecture was preserved. In most cases, germinal centres were small and inactive. Occasionally, they were medium-sized, and, in five cases, they were found to be hyperplastic. The T zones were sometimes hyperplastic and sometimes showed a moderate diffuse increase in the number of reticulum cells.

A relatively prominent finding was dilatation of the sinuses, which were filled to different degrees with lymphoid cells and cells of the plasma cell series. Sometimes it was easier to recognize the marginal sinuses, but similar changes also occurred within the node. The intrasinusoidal cells ranged from small lymphocytes and activated lymphocytes through immunoblasts to plasmablasts and, finally, some small plasmacytoid cells. The most conspicuous cells were the large immunoblasts, which were easily identified with Giemsa staining. They showed a moderate amount of intensely basophilic cytoplasm. Their nuclei were large and round or oval with chromatin that was condensed at the nuclear membrane and a fine chromatin network throughout the nucleus. One, or sometimes more than one, large nucleolus was found in the centre of the nucleus and was also basophilic. These cells correspond to the large basophilic stem cells described by Lennert in 1961, which are now called "immunoblasts" by our group or "large pyroninophilic cells" by Ahlquist et al. (1971). Immunoblasts occasionally resemble Hodgkin or Sternberg-Reed cells (case 8), but the general morphological picture of the cases presented here distinguish them from Hodgkin's disease. In addition to immunoblasts, there were blast cells that

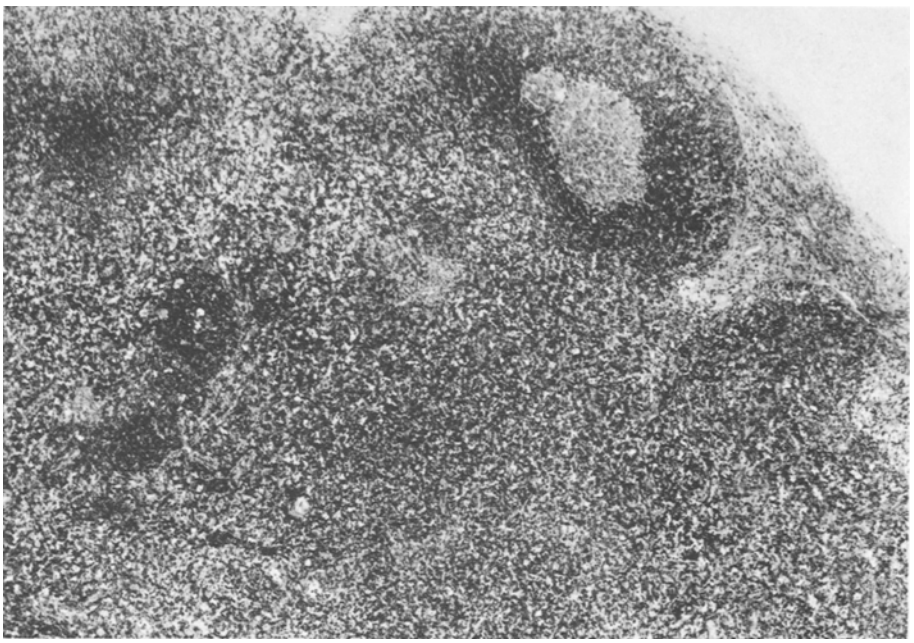

Fig. 3. Hyperplasia of the pulp. Small germinal centre. Case 13, Giemsa, × 56

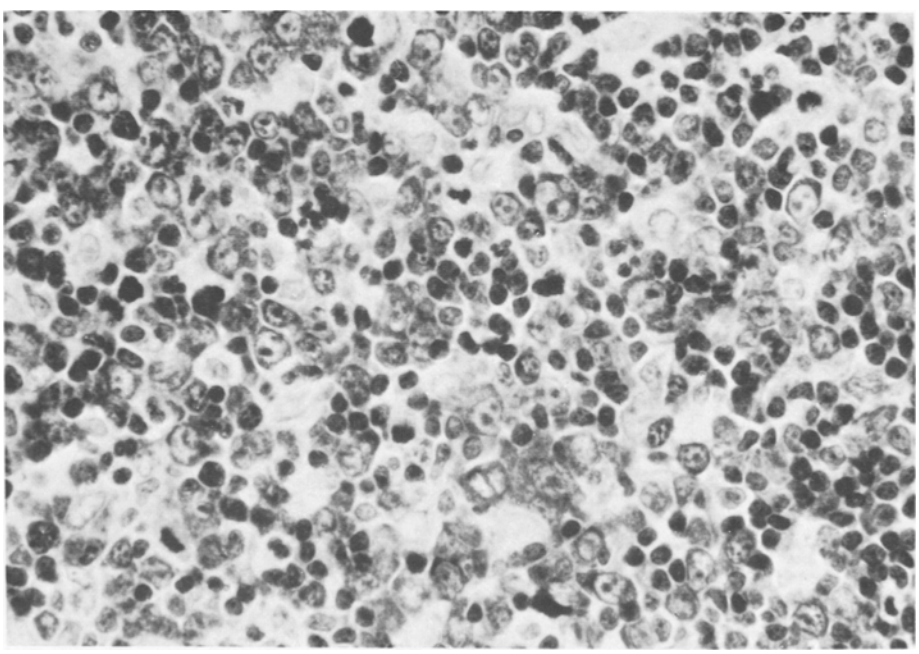

Fig. 4. Infiltration of the pulp by immunoblasts, plasmablasts, and plasma cells. Case 13, Giemsa, × 560

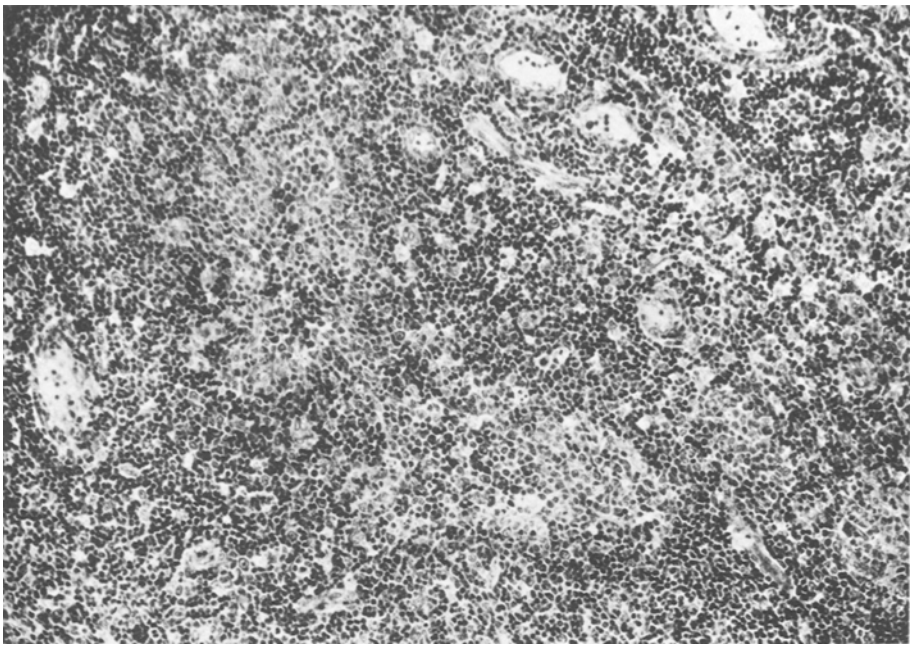

Fig. 5. Two (relatively pale) areas of "immature sinus histiocytosis". Case 13, Giemsa, ×140

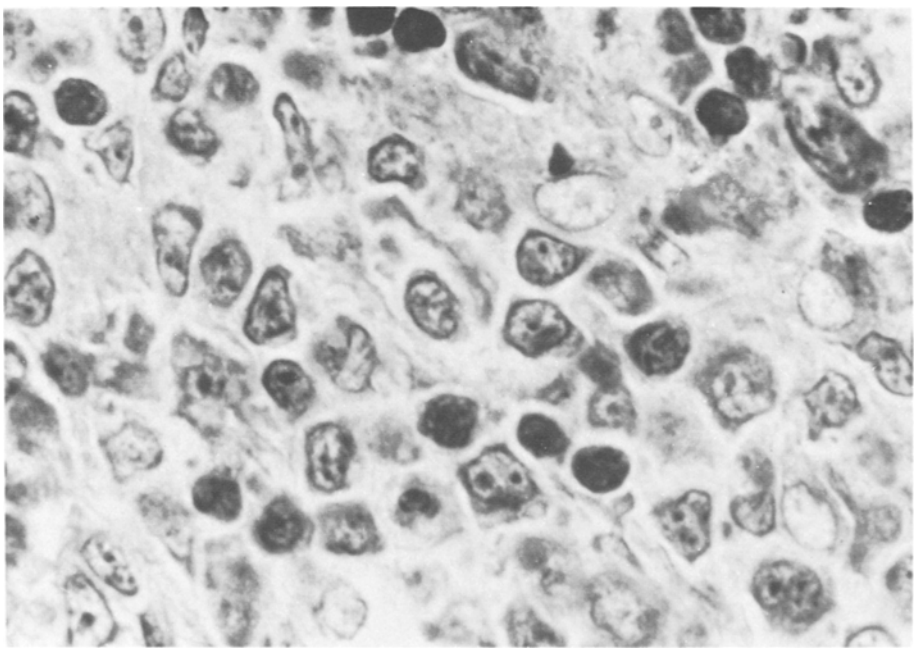

Fig. 6. Detail of Fig. 5 showing pleomorphic lymphoid cells ("immature histiocytes"). Giemsa, ×700

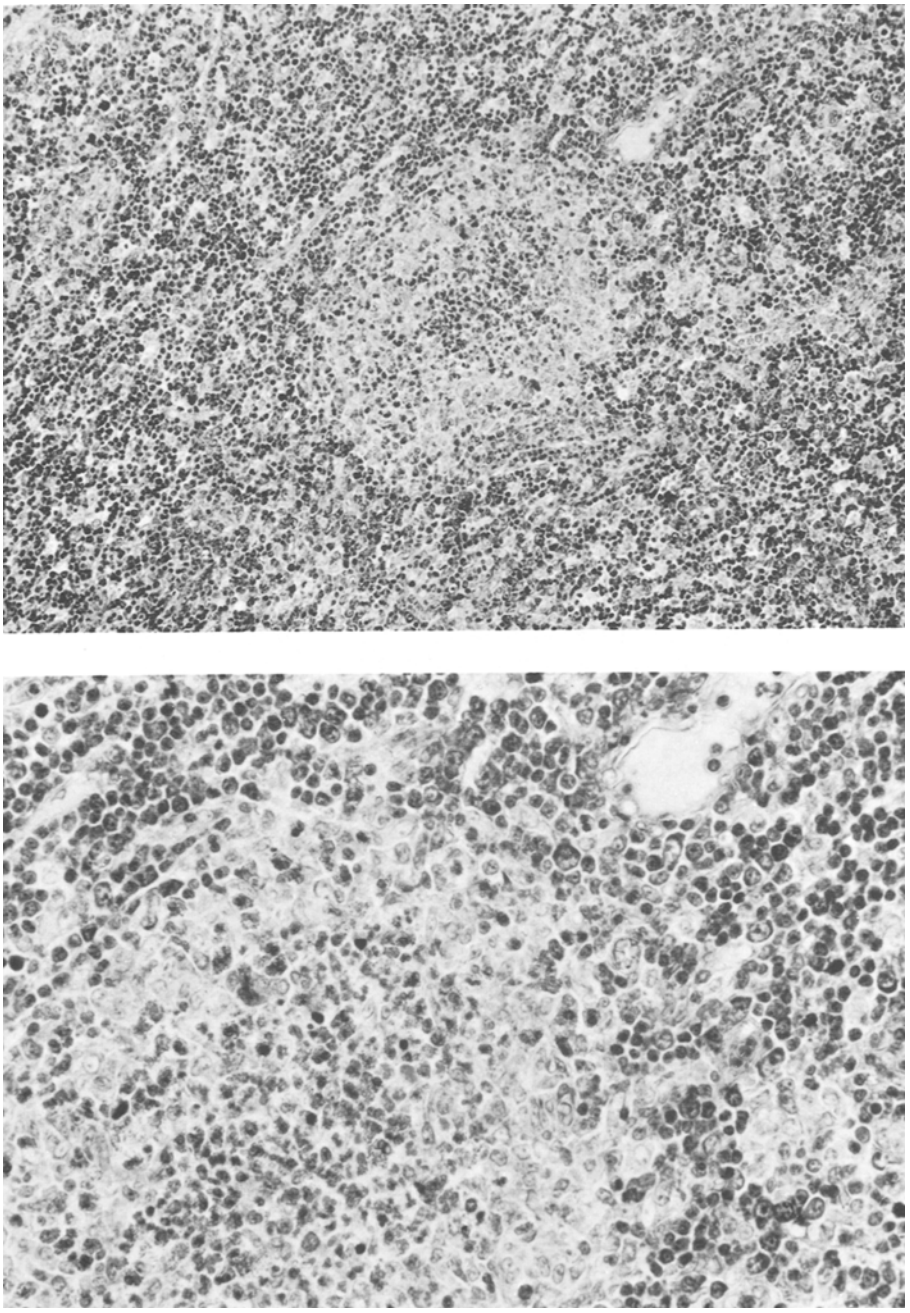

Fig. 7a and b. Small abscess with a "reticulocytic" border. Case 7, Giemsa, (a) ×140, (b) ×380

resembled plasmablasts. These cells were somewhat smaller and contained multiple, medium-sized nucleoli. Furthermore, some small basophilic cells were found. They showed a small to moderate amount of basophilic cytoplasm and a round nucleus with relatively fine chromatin. These cells were thus identified as plasmacytoid cells. The lymphoid cells and cells of the plasma cell series were occasionally interspersed with eosinophils and sometimes with erythrocytes or neutrophils, but seldom with mast cells.

In all cases, the lymph node pulp was hyperplastic owing to a markedly increased number of immunoblasts and plasmablasts and some plasma cells (Figs. 3 and 4). In some cases, these cells were interspersed with eosinophils and sometimes with neutrophils. A number of blast cells in both the pulp and sinuses showed mitotic activity.

The lymph nodes from about two thirds of the cases exhibited so-called immature sinus histiocytosis (Lennert 1961; Mori and Lennert 1969), usually in small foci (Figs. 5 and 6). This "histiocytosis" consists of medium-sized cells with a somewhat pleomorphic nucleus, fine chromatin, one or two small nucleoli, and a small amount of greyish blue cytoplasm. The cell borders are usually distinct. Such cells were once thought to be immature histiocytes, but Mori and Lennert (1969) showed them to be transformed lymphocytes. In the cases presented here, foci of these cells could be found in peripheral or intermediate sinuses, underneath the capsule, or surrounding trabeculae.

In three cases (Nos. 5, 7 and 9), a few small foci of true small histiocytes (emigrated monocytes) were evident, usually in the cortical pulp. In one of these cases (No. 5), the foci showed central necrosis. In the second case (No. 7), there was central accumulation of neutrophils, corresponding to a small abscess (Fig. 7a and b). We found a few foci of epithelioid cells in the pulp in one case (No. 9). None of our cases exhibited the accumulation of leukocytes within germinal centres described by Winblad et al. (1966b).

In six cases, we examined appendix specimens obtained at the same time as the ileocaecal lymph node biopsies. Two of the appendices showed a slight to moderate increase in the same cellular elements as seen in the pulp of the lymph nodes, especially immunoblasts and small histiocytes, within the interfollicular lymphoid tissue and the lamina propria of the upper mucosa.

Discussion

It is possible to distinguish lymphadenitis caused by *Yersinia enterocolitica* infection from other kinds of lymphadenitis. The following findings are characteristic of the former: (1) thickening of the capsule by oedema, with or without infiltration by lymphocytes, plasma cells, and other cells; (2) hyperplasia of the cortical and paracortical pulp, with an increase in immunoblasts, plasmablasts, and plasma cells; (3) dilatation of the sinuses caused by an increased number of cells, especially those of the plasma cell series; (4) foci or relatively large areas of "immature sinus histiocytosis" (seen in about two thirds of our cases); (5) occasionally, small clusters of small histiocytes (emigrated monocytes) in the cortical pulp (only one of our cases showed an abscess similar to those

seen in abscess-forming reticulocytic lymphadenitis). When these features are found in mesenteric lymph nodes, one should suspect a *Yersinia enterocolitica* infection.

Infections with *Yersinia pseudotuberculosis* and *Salmonella* should always be considered in a differential diagnosis. In some cases, it is impossible to differentiate mesenteric lymphadenitis caused by *Yersinia enterocolitica* from that caused by *Yersinia pseudotuberculosis*. Especially in early stages of pseudotuberculosis, the histological picture may be quite similar to that of *Yersinia enterocolitica* infection (Lennert 1961). After a few days, however, mesenteric lymph nodes in *Yersinia pseudotuberculosis* infections show massive infiltration by neutrophils and, later, abscesses with a reticulocytic border. This lesion was called "abszedierende retikulozytäre Lymphadenitis" by Maßhoff (1953). Such abscesses were found in only one of our 14 cases of mesenteric lymphadenitis caused by *Yersinia enterocolitica*.

Salmonella infections may also present problems in a differential diagnosis, because "immature sinus histiocytosis" is also seen in such infections. True typhoid nodules (accumulations of monocytes) are found only in salmonella infections, however, and these nodules are usually more frequent than are the accumulations of "immature sinus histiocytes" in *Yersinia enterocolitica* infections.

Nonspecific mesenteric lymphadenitis can also be a diagnostic problem. In this type of lymphadenitis, a marked increase in plasma cell precursors is often seen in the sinuses; but there is no thickening of the capsule caused by oedema and no infiltration by lymphocytes and plasma cells. Furthermore, nonspecific mesenteric lymphadenitis does not show histiocytic nodules or foci of "immature sinus histiocytosis".

In enteric and pseudo-appendicitis diseases caused by *Yersinia enterocolitica*, therapy is usually unnecessary. If necessary, tetracycline has been used with success (Knapp et al. 1973). *Yersinia enterocolitica* is resistent to ampicillin, carbenicillin, and cephalosporin. It is sensitive to amikacin, chloramphenicol, gentamicin, polymyxin, sisomicin, sulfonamide, tobramycin, and trimethoprim-sulfamethoxazole (e.g., Knapp 1977).

References

Ahlquist J, Ahvonen P, Räsänen JA, Wallgren GR (1971) Enteric infections with Yersinia enterocolitica. Acta Pathol Microbiol Scand Sect A 79:109–122

Aho K, Ahvonen P, Lassus A, Sievers K, Tiilikainen A (1976) Yersinia arthritis and related diseases: clinical and immunogenic implications. In: Dumonde DC (ed) Infection and immunology in the rheumatic diseases. Blackwell, Oxford, p 341–344

Ahvonen P (1972) Human yersiniosis in Finland. Ann Clin Res 4:30–48

Bradford WD, Noce PS, Gutman LT (1974) Pathological features of enteric infection with Yersinia enterocolitica. Arch Pathol 98:17–22

Daniels JJHM (1963) Untersuchungen an als Pasteurella pseudotuberculosis diagnostizierten Stämmen von Chinchillas. Zentralbl Vet Med 10:413–419

Erikson M, Olcén P (1975) Septicaemia due to Yersinia enterocolitica in a non-compromised host. Scand J Infect Dis 7:78–80

Frederiksen W (1964) A study of some Yersinia pseudotuberculosis like bacteria ("Bacterium enterocoliticum" and "Pasteurella X"). Proc XIV Scand Congr Path Microbiol 103. Norw Univ Press, Oslo

Hässig A, Karrer J, Pusterla F (1949) Über Pseudotuberculose beim Menschen. Schweiz Med Wochenschr 79:971–973

Knapp W (1977) Yersiniosen: bakteriologisch-serologische Gesichtspunkte. Med Welt 28:1586–1590

Knapp W, Lysy J, Knapp Ch, Stille W, Goll U (1973) Enterale Infektionen beim Menschen durch Yersinia enterocolitica und ihre Diagnose. Infection I:113–125

Knapp W, Thal E (1963) Untersuchungen über die kulturell-biochemischen, serologischen, tierexperimentellen und immunologischen Eigenschaften einer vorläufig "Pasteurella X" benannten Bakterienart. Zentralbl Bakteriol [Orig A] 190:472–484

Knapp W, Thal E (1973) Die biochemische Charakterisierung von Yersinia enterocolitica (syn. "Pasteurella X ") als Grundlage eines vereinfachten Antigenschemas. Zentralbl Bakteriol [Orig A] 223:88–105

Laitinen O, Leirisalo M, Allander E (1975) Rheumatic fever and Yersinia arthritis: criteria and diagnostic problems in a changing disease pattern. Scand J Rheumatol 4:145–157

Leino R, Kalliomäki JL (1974) Yersiniosis as an internal disease. Ann Intern Med 81:458–461

Lennert K (1961) Lymphknoten. Diagnostik in Schnitt und Ausstrich. Bandteil A: Cytologie und Lymphadenitis. In: Lubarsch O, Henke F, Rössle R, Uehlinger E (Hrsg) Handbuch der speziellen pathologischen Anatomie und Histologie, Bd I/3A. Springer, Berlin Göttingen Heidelberg

Maßhoff W (1953) Eine neuartige Form der mesenterialen Lymphadenitis, Dtsch Med Wochenschr 78:532–535

Mollaret HH (1966) L'infection humaine à Yersinia enterocolitica. Pathol Biol (Paris) 14:981–990

Mollaret HH, Chevalier A, Deplanche MC (1964) Contribution à l'étude d'un nouveau groupe de germe (Yersinia enterocolitica) proches du bacille de Malassez et Vignal. Ann Inst Pasteur 107:121–127

Mollaret HH, Destombes P (1964) Les germes "X" en pathologie humaine. Presse méd 72:2913–2915

Mori Y, Lennert K (1969) Electron microscopic atlas of lymph node cytology and pathology. Springer, Berlin Heidelberg New York

Nilehn B (1969) Studies on Yersinia enterocolitica. Acta Pathol Microbiol Scand Suppl 206:48

Nilehn B, Sjöström B, Damgaard K, Kindmark C (1968) Yersinia enterocolitica with the symptoms of infectious disease. Acta Pathol Microbiol Scand 74:101–113

Schleifstein J, Coleman M (1939) An unidentified microorganism resembling B. lignieri and Pasteurella pseudotuberculosis and pathogenic for man. NY State J Med 39:1749–1753

Spira TJ, Kabins SA (1976) Yersinia enterocolitica septicemia with septic arthritis. Arch Intern Med 136:1305–1308

Vandepitte J, van Noyen R, Isebaert A (1970) Yersinia enterocolitica: its incidence in patients with infectious diarrhoe. Abstr VI Congr Int Mal Infect Wien Acad Sci, p 119–123

Vantrappen G, Agg HO, Ponette E, Geboes K,, Bertran Ph (1977) Yersinia enteritis and enterocolitis: gastroenterological aspects. Gastroenterology 72:220–227

Wauters G (1970) Contribution à l'étude de Yersinia enterocolitica. Thése d'agregation, Louvain

Wauters G, Le Minor L, Chalon AM, Lassen J (1972) Supplément en schéma antigénique de Yersinia enterocolitica. Ann Inst Pasteur 122:951–956

Winblad S (1968) Studies on O-antigen factors of "Yersinia enterocolitica". Symp Series Immunobiol Stand 9:337–343

Winblad S (1969) Erythema nodosum associated with infection with Yersinia enterocolitica. Scand J Infect Dis 1:11–16

Winblad S, Nilehn B, Jonsson M (1966a) Two further cases bacteriologically verified, of human infection with Pasteurella X (syn. Yersinia enterocolitica). Acta Pathol Microbiol Scand 66:537–541

Winblad S, Nilehn B, Sternby NH (1966b) Yersinia enterocolitica ("Pasteurella X") in human enteric infections. Br Med J 2:1363–1366

Accepted August 23, 1980