대한외과학회지 : 제 62 권 제 3 호
Vol. 62, No. 3, March, 2002

□ Case Report □

Cervical Lymphadenitis Caused by Toxoplasma Gondii

Department of Surgery, [1]St. Vincent's Hospital and [2]Holy Family Hospital, The Catholic University of Korea, Seoul, Korea

Young Jin Suh,[1] Wook Kim,[2] Woo-Bae Park,[1] and Chung-Soo Chun[1]

경부임파선 포자충증

서영진 · 김 욱 · 박우배 · 전정수

We present a case of toxoplasma lymphadenitis diagnosed by histologic and serologic evaluation. A 40-year-old woman presented with a 4-month history of a unilateral cervical lymphadenopathy. An excisional biopsy specimen of the lymph node disclosed a toxoplasma cyst. Her IgG anti-Toxoplasma serotiter far exceeded the upper normal limit. Histological findings supporting a diagnosis of toxoplasmosis correlated with the results of serological studies. This report emphasizes the need for clinicians to consider toxoplasmosis in the differential diagnosis of lymphadenopathies in young patients with enlarged cervical lymph nodes and varying degrees of fatigue, malaise, cough, and fever. **(J Korean Surg Soc 2002;62:271-273)**

Key Words: Toxoplasmosis, Lymph nodes, Neck
중심 단어: 포자충증, 임파선, 경부

가톨릭대학교 성빈센트병원 외과

INTRODUCTION

Toxoplasma gondii was discovered in 1907 and found to induce a human disease in 1948. It has a worldwide distribution and has been noted to present with a wide spectrum of clinical symptoms. Toxoplasma lymphadenitis, in its typical form, involves the posterior cervical nodes of young women (so-called Piringer-Kuchinka lymphadenitis).(*1*) Local serological surveys have also demonstrated that anti-Toxoplasma antibodies are present in about a fifth of the domestic animals and of normal healthy adults.(*2*)

Correspondence : Young-Jin Suh, Department of Surgery, St. Vincent's Hospital, The Catholic University of Korea, 93 chi-dong paltal-gu, Suwon 442-723, Korea, (Tel) +82-31-249-7176, (Fax) +82-31-247-5247, (E-mail) yjsuh@vincent.cuk.ac.kr
Received February 7, 2002, Accepted February 20, 2002
*This was supported in part by the fund from Catholic Medical Center.

The diagnosis of toxoplasma lymphadenitis is traditionally based on a combination of lymph node excisional biopsy along with serological studies. We report a case of uncommon toxoplasma lymphadenitis.

CASE REPORT

A 40-year-old woman presented with a 4-month history of unilateral neck lymphadenopathy, appetite loss, and fatigue. She had received antibiotics with no physical or subjective improvement and the cervical lymph node even enlarged. On physical examination there was an enlarged lymph node in the posterior neck triangle unilaterally, measuring 2 cm in diameter, which was palpable in the upper portion of the right posterior triangle. There was no splenomegaly, and the physical examination was otherwise unremarkable.

The aforementioned lymph node was sampled by fine-needle aspiration, using a 22-gauge needle and 10-ml syringe attached to an aspiration gun. Cytologic changes included no specific findings but some lymphoid cells were observed. Excisional biopsy was done to confirm the exact histologic diagnosis that made us to suspect toxoplasmic lymphadenitis (Fig. 1). For the serologic confirmation of the toxoplasma lymphadenitis, IgG and IgM anti-toxoplasma serotiters were measured and were shown to be elevated at 146.5 IU/ml (reference range: <6 IU/ml) and 169.3 AU/ml (reference range: <100 AU/ml), respectively. After excision, she remained free of any cervical masses.

DISCUSSION

Toxoplasma gondii (*T. gondii*), a coccidian protozoon, infects various animal species and humans. It rarely harms its hosts including humans, pigs, and cattle, and most of the human infections remain occult throughout life, eliciting tissue damage only when cellular immunity is impaired.

Gametogenesis and oocyst formation takes place exclusively in the gut of feline hosts. Sporozoites are liberated in the feces of the feline hosts and then may infect various host species,

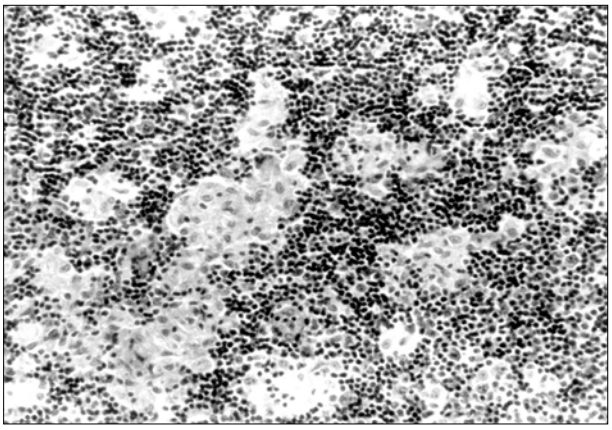

Fig. 1. Toxoplasmosis of lymph node (H&E stain, ×200). Small noncaseating granulomas composed of epithelioid cells, located at the periphery of hyperplastic follicle. This picture is almost pathognomonic of toxoplasma lymphadenitis.

in which cysts containing dormant bradyzoites are formed. When ingested through contaminated meat, cysts infect hosts by releasing the organisms that become motile, called tachyzoites or trophozoites. Such active motile forms are also released from dormant cysts in tissue upon faltering of the host resistance, swarming through the organs, entering inflammatory or tissue cells, causing cell damage and inflammation, and giving rise to clinical toxoplasmosis. On entering cells, the tachyzoites multiply intracellularly and sometimes fill the cytoplasm of parasitized cells, forming a new generation of cysts known as terminal colonies or pseudocysts.(*3*) Cysts containing bradyzoites can persist in the lesions long after resolution of active infection; therefore, their presence in specimens may not be diagnostic of active disease.(*4*)

Clinically, acquired toxoplasmosis presents itself with or without constitutional symptoms, such as fatigue, malaise, and fever.(*5*) The most common clinical feature is enlarged cervical lymph nodes, commonly unilateral.(*6*) The clinical presentations vary greatly, and the lymphadenopathy may be bilateral in the neck, generalized, intraabdominal, intraparotid, or intraoral, and can be confused with other clinical syndromes, including lymphoma.(*7,8*) The disease can be extranodal, presenting as tonsillar enlargement with neck lymphadenopathy, pneumonitis, chorioretinitis, papulonodular dermatitis, myocarditis, or encephalitis, taking a grave form in immunocompromised hosts; especially those with acquired immunodeficiency syndrome (AIDS) and following organ transplants, in which pulmonary forms have been reported, with identification of the organisms in bronchoalveolar lavage specimens.(*9-12*)

The congenital form is transmitted from a mother having active infection via the placenta and has a devastating effect on the newborn, presenting with psychomotor and mental retardation, chorioretinitis, and encephalitis frequently accompanied by internal hydrocephalus.(*5*)

On palpation, the nodes are firm and only moderately enlarged. Microscopically, the nodal architecture is rather well preserved. The histologic picture of toxoplasma lymphadenitis is characterized by follicular hyperplasia with fair preservation of normal lymph node architecture, focal proliferation of transformed monocytoid B-lymphocytes, and small scattered clusters of large and epithelioid-like histiocytes, with larger aggregates of such cells, defined as granulomas, being rare.(*13*) At times, necrotic foci and granulocytic collections are present, and occasionally there are extensive necrotizing changes simulating Kikuchi lymphadenitis or other conditions, although there have been cases in which entities such as these have coexisted with toxoplasmosis even in the same lymph node.(*14,15*) It is extremely rare to find *Toxoplasma* organisms by morphologic examination and just as difficult to detect the *Toxoplasma gondii* genome by polymerase chain reaction (PCR).(*16,17*) However, the combination of microscopic features described correlates remarkably well with serologic studies. Of 31 cases studied by Dorfman and Remington,(*18*) the Sabin-Feldman dye test was positive in all, and the IgM immunofluorescent antibody test was positive in 97% of the cases.

If the diagnosis of toxoplasmic lymphadenitis is suspected from the microscopic pattern, it should be confirmed serologically, keeping in mind that these tests might be normal in the early stages of the disease.(*19*) For this purpose, the enzyme-linked immunosorbent assay (ELISA) and the indirect immunofluorescence method are available for titration of the parasite-specific IgM antibody.(*20*)

The differential diagnosis of toxoplasmosis includes other infectious diseases and the lymphocyte predominant form of Hodgkin's disease. In this regard, Miettinen and Franssila(*21*) have made an interesting point that occurrence of collections of epithelioid cells within germinal centers seems to be a nearly specific feature for toxoplasmosis.

The disease is usually self-limited and resolves in weeks to months without any residual complication. The prognosis of our case was excellent.

In conclusion, toxoplasmosis may not be uncommon cause of cervical lymphadenopathies in pork-eating area and thus should be considered in the differential diagnosis of lymphadenopathy. The distinctive histopathological changes in affected lymph node might prevent the misdiagnosis of more serious

conditions like Hodgkin's disease. The morphology, however, should correlate with high serological titer to make a definitive diagnosis.

REFERENCES

1) Piringer-Kuchinka A, Martin I, Thalhammer O. Ueber die vorzuglich cerviconuchale Lymphadenitis mit kleinherdinger Epitheloid-zellwucherung. Virchows Arch [A] 1958;331:522-35.
2) Singh M, Zaman V, Goh TK, Chew M. A report on the prevalence of toxoplasmic antibodies in Singapore. Singapore Med J 1968;9:108-10.
3) Woods GL, Gutierrez Y. Blood and tissue protozoa. In: Diagnostic pathology of infectious diseases. Philadelphia: Lea & Febiger; 1993. p.497-511.
4) Wheeler RR, Bardales RH, North PE, Vesole DH, Tricot G, Stanley MW. Toxoplasma pneumonia: cytologic diagnosis by bronchoalveolar lavage. Diagn Cytopathol 1994;11:52-5.
5) Ahmad M, Iqbal J, Mansoor A, Khan AH. Toxoplasmic lymphadenitis. A clinico-pathological study. JPMA 1991;41: 303-5.
6) McCabe RE, Brooks RG, Dorfman RF, Remington JS. Clinical spectrum in 107 cases of toxoplasmic lymphadenopathy. Rev Infect Dis 1987;9:754-74.
7) Chizynski Z, Dworniak D, Tkacz B. Case of generalized lymphonodular form of acquired toxoplasmosis. Wiad Lek 1997;32:399-401.
8) Akiner MN, Saatci MR, Yilmaz O, Erekul S. Intraglandular toxoplasmosis lymphadenitis of the parotid gland. J Laryngol Otol 1991;105:860-2.
9) Kouba K, Lasovska J, Mirejovsky P, Stehlikova J, Kramarova K. Tonsillar-glandular form of acquired acute toxoplasmosis. J Laryngol Otol 1977;91:601-4.
10) Mojon M, Garin JP, Francois R, Hermier M. Pure pulmonary form of toxoplasmosis. Pediatrie 1972;27:80.
11) Renschler HE, Schumacher LK. Streptococcus angina, myocarditis and toxoplasmosis following splenectomy. Internist (Berlin) 1970;11:150-1.
12) Midana A, Zina G, Pileri A. Acquired toxoplasmosis in adults with cutaneous papulo-nodular manifestations. Bull Soc Dermatol Syphiligr 1970;77:333-4.
13) Sheibani K, Fritz RM, Winberg CD, Burke JS, Rappaport H. Monocytoid cells in reactive follicular hyperplasia with and without multifocal histiocytic reactions: an immunohistochemical study of 21 cases including suspected cases of toxoplasmic lymphadenitis. Am J Clin Pathol 1984;81:453-8.
14) Cherin P, Leger-Ravet B, Aznar C, D'oiron R, Delfraissy JR. Kikuchi's histiocytic necrotizing lymphadenitis of toxoplasmic origin may be due to a local reactional mechanism in lymph nodes. Clin Infect Dis 1995;20:481-2.
15) Kikuchi M, Yoshizumi T, Nakamura H. Necrotizing lymphadenitis: possible acute toxoplasmic infection. Virchows Arch [A] 1977;376:247-53.
16) Weiss LM, Chen YY, Berry GJ, Strickler JG, Dorfman RF, Warnke RA. Infrequent detection or Toxoplasma gondii genome in toxoplasmic lymphadenitis. A polymerase chain reaction study. Hum Pathol 1992;23:154-8.
17) Voglino G, Arisio R, Novero D, Marchi C, Fessia L. Lymphadenopathy caused by toxoplasma in an intramammary lymph node: role of molecular biology in the diagnosis. Pathologica 1997;89:446-8.
18) Dorfman RF, Remington JS. Value of lymph-node biopsy in the diagnosis of acute acquired toxoplasmosis. N Engl J Med 1973;289:878-81.
19) Frenkel JK. Toxoplasmosis. Mechanisms of infection, laboratory diagnosis and management. Curr Top Pathol 1971;54: 28-75.
20) Johnson AM, Gu QM, Roberts H. Antibody patterns in the serological diagnosis of acute lymphadenopathic toxoplasmosis. Aust N Z J Med 1987;17:430-4.
21) Miettinen M, Franssila K. Malignant lymphoma simulating lymph node toxoplasmosis. Histopathology 1982;6:129-40.