Lymphomatoid Granulomatosis Involving the Central Nervous System: Complication of a Renal Transplant with Terminal Monoclonal B-Cell Proliferation

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Summary. A 33-year-old man, an immunosuppressed renal allograft recipient, developed fever of unknown etiology, multiple pulmonary densities, spaceoccupying lesions in the liver, confusion, disorientation, and seizures with cerebral infarcts. At autopsy, changes classical of active lymphomatoid granulomatosis (LYG) were found in the brain but those studied in the lungs and liver were in the healing phase.

In addition, one cerebral focus in the right temporooccipital lobe showed a lymphomatous transformation. Immunofluorescence studies done on this focus showed monoclonal IgG- λ cytoplasmic staining in the perivascular infiltrate. Immunoperoxidase studies confirmed the monoclonal nature of this focus but showed a polyclonal pattern in the other foci. This finding is discussed along with the relationship between LYG and lymphoma.

Key words: Lymphomatoid granulomatosis – Central nervous system – Immunofluorescence – Renal transplant – Lymphoma

Introduction

Lymphomatoid granulomatosis (LYG) described by Liebow et al. (1972) is morphologically defined as a necrotizing angiocentric and angiodestructive infiltrative disorder. The infiltrate is pleomorphic with lymphocytes, plasma cells, histiocytes, and a variable amount of atypical mononuclear cells. Lungs are predominantly involved. Extra-pulmonary involvement is frequent and is most often clinically manifested by cutaneous or neurologic symptoms, the latter bearing a grave prognosis. The evolution and prognosis are quite variable and unpredictable with long survival periods or, more often, rapid deterioration and death (Liebow et al. 1972; Katzenstein et al. 1979). "Polymorphous" lymphoma or immunoblastic sarcoma are sometimes part of the terminal picture (Liebow et al. 1972; Katzenstein et al. 1979; Cohen et al. 1979; Bender and Jaffe 1980). No satisfactory mode of therapy is yet available (Katzenstein et al. 1979).

We describe a case of LYG complicating the evolution of an immunosuppressed renal transplant patient. The central nervous system (CNS), lungs, and liver were involved although lesions were healing in the latter two sites.

In addition, one cerebral focus in the right temporooccipital lobe showed lymphomatous transformation. Immunofluorescence and immunoperoxidase studies of this focus revealed the presence of a monoclonal cytoplasmic immunoglobulin in the perivascular infiltrate. The other foci were, however, polyclonal by immunoperoxidase studies.

Case Report

Clinical History

A 33-year-old white man developed proteinuria at 15 years of age. Chronic renal failure followed, and a diagnosis of chronic glomerulonephritis was made 10 years later. He was placed on hemodialysis. At 30, a renal transplant had to be removed because of acute rejection. One year prior to the final admission, bilateral nephrectomy was done and was followed by another transplant. Multiple complications followed: cytomegalovirus pneumonia, signs of transplant rejection, hypertension, herpetic and monilial esophagitis, right lower lobe pulmonary infiltrate secondary to papovavirus and bleeding esophageal ulcer. He had no allergies and no history of alcohol abuse. He was a heavy smoker. The family history was non-contributory.

His last admission was for fever of a few days duration, anorexia, and abdominal discomfort of 1 month's duration. He also complained of a slight weight loss. The medications, at the time of admission, included Prednisone 20 mg daily, Inderal 160 mg b.i.d.,

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Aldomet 250 mg q.i.d., Imuran 75 mg daily, and Lasix 40 mg daily. Physical examination revealed a patient in no acute distress. The blood pressure was 120/90 mm Hg, temperature was 38.9° C. The head, neck, chest, and cardiovascular system were unremarkable. Abdominal palpation revealed a non-tender kidney in the right lower quadrant. The neurologic examination was totally unremarkable.

Laboratory investigation revealed a normochromic normocytic anemia with hemoglobin (Hb) of 8.9 mg/100 ml. The white cell count (WBC) was 3,800 cells/mm³ with a normal differential count. Rehydration corrected slightly elevated creatinine and BUN levels. Electrolytes, bilirubin, serum calcium, iron, vitamin B12, and serum and red cell (RBC) folate were normal. The blood coagulation studies were also normal. A renal scan and an i.v. pyelogram were unremarkable.

The evolution was mostly characterized by a fever varying between 38.1° C and 39.2° C. All blood, throat urine, and fecal cultures remained negative for bacteria and fungi. Sputum was repeatedly negative for acid-fast bacilli. Numerous studies were done to rule out the following: adenovirus, herpes simplex, varicella-zoster, cytomegalovirus, influenza A/B, papopavirus, mycoplasma, toxoplasmosis, blastomycosis, coccidioidomycosis, cryptococcosis, histoplasmosis, psittacosis, rickettsia, and Q fever. All tests were negative. WBC remained between 2,200 and 3,800 cells/mm³ with a normal differential count.

The admission chest X-ray revealed a density in the right upper lobe, approximately 2 cm in diameter. Repeated chest X-rays were suggestive of a tuberculous process. Although repeated sputum examinations were negative for acid-fast bacilli, a trial of Isoniazid and Rifampin was begun in addition to the initial medication. Fever persisted and the trial was discontinued. A percutaneous needle biopsy of the right lung was not contributory. Subsequently, he developed a right lower lobe consolidation and several nodules in the left lower lobe. A bronchoscopy showed inflamed right middle and lower lobe bronchial mucosa. Cultures from washing and brushing specimens remained negative. An open lung biopsy showed focal necrotizing pneumonitis. Tissue cultures were negative. Tests for Legionnaire's disease were also negative.

Hepatomegaly and a slight elevation of the hepatic enzymes were also noted. A computerized axial tomography (CAT) body scan showed space-occupying lesions in the liver. A biopsy revealed only hemosiderosis and tissue cultures remained negative.

Three weeks prior to his death, he developed disorientation and confusion with a suggestion of hallucinations. Five days later, a grand mal seizure occurred with head and eyes deviated to the right but without other signs of localization. A CAT scan was negative. A lumbar puncture showed 82 RBC, 4 WBC, a glucose level of 80 mg/100 ml, and a protein level of 72 mg/100 ml. Cultures were negative. Six days later, another seizure occurred. The CAT scan showed a possible infarct in the right parieto-occipital region. He improved temporarily. Five days prior to his death, however, neck stiffness and disorientation occurred with a rapid deterioration. A lumbar puncture showed 5 RBC, 59 WBC (all mononuclears), a glucose level of 32 mg/100 ml, and a protein level of 103 mg/100 ml. Cultures remained negative. A brain needle biopsy was done in the right temporal lobe. The tissue was very scanty and smears only were done at the time. No organisms were found but a few atypical cells suggested the possibility of a neoplasm. Permanent sections revealed areas of necrosis, a few atypical cells, and two small vessels with a definite fibrinoid necrosis. A vasculitis was suggested without further characterization due to the small amount of tissue available. Tissue cultures were also negative. Profound coma preceded his death and a complete autopsy was performed.

Methods

Representative paraffin sections were studied with one or more of the following stains: hematoxylin-eosin (HE), Gordon and Sweet,



Fig. 1. Coronal section, left temporal lobe. Softening and petechiae involving the left hippocampus

Martius scarlet blue, methyl green-pyronin, luxol fast blue, Gram, periodic acid-Schiff (PAS), methenamine silver, Dieterle (for Legionnaire's disease), Ziehl-Nielson, and Warthin-Starry (for spirochetes).

For immunofluorescence, fresh tissue from the right temporal lobe was snap-frozen in a liquid nitrogen-isopentane slurry and stored at -80° C until processed. Frozen sections were cut, picked up on gelatinized slides, dried at 4°C overnight, and stained for immunoglobulins and C3 using a direct immunofluorescence method. Fluorescence was visualized with a Leitz Orthoplan fluorescent microscope with an HBO 100W high pressure mercury light source, using transmitted illumination. Immunoperoxidase studies were performed using the peroxidase-anti-peroxidase (PAP) method on formaldehyde-fixed paraffin-embedded cerebral tissue. This technique as well as its various control methods has been described before in detail by Taylor (1978a).

Autopsy Findings

Central Nervous System. The brain weighed 1,410 g. External examination revealed a mild flattening of gyri without significant uncal or cerebellar tonsil herniation. Several foci of softening with petechiae were seen over the lateral and inferior temporal and occipital lobes. One, at the tip of the left temporal lobe, was covered by a yellow green meningeal exudate.

Coronal sections of the brain showed multiple foci of petechiae with or without associated softening. They involved both hemispheres, all cerebral artery territories, and the cortex and white matter. The most prominent foci were located in the left occipital lobe and in the inferior portion of the right temporo-occipital lobe where the softening was significantly more extensive. The genu of the corpus callosum was involved on the right side and the hippocampus on the left (Fig. 1). The deep gray matter was spared. A small hematoma filled the biopsy site in the right temporal lobe.

External examination of the cerebellum, brainstem, and spinal cord was unremarkable. However, sagittal and parasagittal sections of the cerebellum revealed two large foci of hemorrhagic softening, one in the central vermis and the other in the left posterior hemisphere. Transverse sections of the brainstem and spinal cord were grossly normal.

Microscopic examination showed a patchy but rather dense infiltrate in the meninges and cerebral parenchyma giving a picture of meningo-encephalitis (Fig. 2). The infiltrate had a predominantly perivascular distribution. Several vessels showed complete fibrinoid necrosis of the wall with narrowing or occlusion of the lumen by

J. Michaud et al.: Lymphomatoid Granulomatosis Involving the CNS



Fig. 2. Meninges and cortex, right occipital lobe, with meningeal, perivascular and parenchymal infiltrate. One cortical vessel shows fibrinoid necrosis (*arrow*). HE, $\times 65$

fibrin. Small and medium-sized vessels were involved (Fig. 3). Surrounding necrosis of the cerebral parenchyma was seen more often in sections taken from the temporal and occipital lobes and the cerebellum.

The infiltrate was pleomorphic with small lymphocytes, plasma cells, histiocytes, and atypical mononuclear cells (Fig. 3). The latter varied in number from one field to another. In the right temporooccipital lobe, however, the atypical mononuclear cells were the majority (Fig. 4). Mitoses were seen along with bi- or trinucleated cells. No Reed-Sternberg cells were found. Pyroninophilia was seen in plasma cells and in some atypical mononuclear cells. Russell bodies were occasionally seen in plasma cells. Reticulin deposition was increased around vessels with a thick perivascular infiltrate, in the right temporo-occipital lobe (Fig. 4, insert).

Microscopically, the most severely involved structures were the meninges of the cerebral hemispheres and midbrain, and the cortex and subcortical white matter of the cerebellum and cerebrum. However, similar changes were seen focally in the left mammillary body, the substantia nigra, the left basis pontis, the pontine tegmentum and medulla dorsal to the pyramids, the fourth cranial nerve, and one cranial nerve at the medullary level. No organisms or viral inclusions were found.

The spinal cord, pituitary gland, eyes, common peroneal nerve, and gastrocnemius muscle were not involved. Finally, a mild anoxic encephalopathy was seen in both hippocampi and in the left frontal lobe.



Fig. 3. Photomicrograph of a cortical vessel, left frontal lobe, showing fibrinoid necrosis and perivascular pleomorphic infiltrate and necrosis. HE, $\times 120$



Fig. 4. Photomicrograph of a cortical vessel, right temporal lobe, showing numerous atypical mononuclear cells. HE, \times 190. *Insert*. Increased reticulin deposition, around vessel with thick perivascular infiltrate. Reticulin, \times 50

Other Systems. The right and left lungs weighed 950 and 970 g, respectively. All lobes were distended and firm. On cut sections, a few yellow-white nodules, measuring between 1.0 and 3.5 cm, were seen in both upper and lower lobes, bilaterally. Microscopically, they consisted of necrotic tissue surrounded by granulation tissue with occasional multinucleate giant cells. Some totally necrotic vessels surrounded by degenerated and ghost-like irregular mononuclear cells. The liver was markedly enlarged and weighed 2,800 g. On cut sections, several yellow-white irregularly circumscribed lesions varying from 1.0 to 2.0 cm were present. Microscopically, they were similar to the pulmonary lesions. Increased fibrosis was present in the surrounding periportal regions. No viral inclusions and no organisms were found in both liver and lung sections.

The lymph nodes showed a non-specific mild reactive hyperplasia. The spleen showed congestion, and the bone marrow was normal. The skin was normal. The other significant changes found at autopsy were: moderate chronic rejection of kidney transplant with arteriolonephrosclerosis and a few infarcts, thrombotic non-bacterial endocarditis of the mitral valve and acute bronchopneumonia.



Fig. 5 A, B. Immunoperoxidase study of monoclonal focus. A Lambda staining is present in several cells (*arrow heads*): B no reaction is present with Kappa light chain. Immunoperoxidase: $A \times 630$, $B \times 400$

Gram-negative rods were seen in the foci of acute bronchopneumonia and cultures grew *Escherichia coli*.

Immunohistochemical Studies. The mononuclear infiltrate showed cytoplasmic staining for IgG- λ only, by immunofluorescence, and for light chain only by immunoperoxidase (Fig. 5). However, in the polyclonal foci, cytoplasmic staining was detected with both immunoglobulin light and heavy chains (Fig. 6). By immunofluorescence, deposits of IgG and C3 were found in affected vessels.

Electron Microscopy. The fibrinoid necrosis of blood vessel walls and the pleomorphic infiltrate were confirmed. No viral structures or other organisms were found. However, these studies did not contribute further to the evaluation of the morphological changes and did not provide new data.

Discussion

The present case demonstrated clinically and morphologically several features of lymphomatoid granulomatosis (LYG): fever of unknown origin; multiple organ involvement: lungs, liver, and brain (including cranial nerves); striking vasculitis with parenchymal necrosis and pleomorphic perivascular infiltrate including atypical mononuclear cells. These features were first described by Liebow et al. (1972). Clinical manifestations of LYG, prognostic factors, and results of various therapeutic regimens in 152 patients were further documented by Katzenstein et al. (1979). In about one of six of these patients, associated diseases were present before or during the development of LYG. The nature of these illnesses was quite variable. However, most of them either required the use of immunosuppressive drugs or were commonly associated with immunologic disturbances (Katzenstein et al. 1979).

Immunosuppressed renal transplant recipients would therefore fall in a group susceptible to develop LYG. However, only three previous reports of such a combination have been made (Hammar and Mennemeyer 1976; Gardiner 1979; Walter et al. 1979). If one excludes the problems of rejection, renal transplantation is more often complicated by infections or by secondary malignancies.

Malignancies complicating renal transplantation are rising in frequency. After cancers of the skin and lips, lymphomas are the most frequent. The majority



Fig. 6A, B. Immunoperoxidase study of one polyclonal area. Lambda (A) and Kappa (B) staining is seen in occasional cells (*arrow heads*). Immunoperoxidase, $\times 400$

are large-cell lymphomas (Penn 1977). Contrary to the lymphomas diagnosed in the general population, the renal transplant lymphomas have a high predilection for the CNS: 43 of 103 in Penn's series. Furthermore, this system is frequently the only one involved by the process (Penn 1977).

Lymphomatoid granulomatosis has also a predilection for the CNS (Liebow et al. 1972; Katzenstein et al. 1979; Pena 1977; Verity and Wolfson 1976; Simon et al. 1981). Sometimes, neurologic symptoms are its only manifestation (Kokman et al. 1977). More often, the CNS involvement arrives when the disease is well established in the other systems or while it is being treated. Bone et al. (1978) emphasized that point in reviewing 13 cases published in the literature, including their own. They also noted that in six of them, pulmonary lesions were clearing after the development of the CNS lesions. This feature was present in our case. Both pulmonary and hepatic lesions were healing at the time of autopsy and were therefore not diagnostic. The fact that CNS lesions develop so frequently during treatment indicates resistance to treatment at least in that system. It also emphasizes the bad prognosis of CNS involvement, as already indicated by Katzenstein et al. (1979).

The present case stresses the close relationship between LYG and lymphomas because one focus in the right temporo-occipital lobe was morphologically a lymphoma. This area, studied by immunofluorescence, showed monoclonal IgG- λ cytoplasmic staining. The PAP method confirmed this finding while showing the polyclonal nature of the other foci. Deposition of reticulin in the perivascular lymphomatous cuffing is also a classical feature of intracerebral lymphomas (reticulin cell sarcoma-microglioma). In a large series of LYG, 12% of patients developed malignant lymphoma (Katzenstein et al. 1979). Focal lymphomatous transformation in LYG has rarely been studied immunohistochemically.

Reddick et al. (1978) studied a case of systemic LYG with immunoblastic sarcoma in the brain. The cerebral lesion showed only a few cells staining with IgG or IgM and both k and λ chains. The lymph nodes showed a polyclonal pattern, but the typical LYG

lesions in the lungs were not studied. Bender and Jaffe (1980), using an indirect immunoperoxidase method, demonstrated a predominantly mixed pattern of all immunoglobulins in two typical cases of pulmonary LYG. One of these had one focus consistent with immunoblastic sarcoma and this one showed only IgG/k. Two atypical cases were also studied, one showing a mixed pattern and the other no staining at all. They noticed that these results were similar to other benign lymphoproliferative disorders with malignant potentials, e.g., angioimmunoblastic lymphadenopathy, with an original polyclonal pattern sometimes evolving in a monoclonal proliferation. These few studies do not permit, however, definite conclusions.

Interestingly, primary intracranial lymphomas, without organ transplantation or immunosuppressive therapy, seem to be more consistently monoclonal (Houthoff et al. 1978; Taylor et al. 1978b; Hassoun et al. 1981; Jardon-Jeghers and Reznik 1982). Even if a certain number of cases show results difficult to interpret or no staining at all, these results are in accordance with those of a large series of extra-cranial B-cell lymphomas (Taylor 1979).

Distinction between LYG and lymphomas has been already commented upon several times (Liebow et al. 1972; Bone et al. 1978; Katzenstein et al. 1979; Pasquier et al. 1980; Bender and Jaffe 1980; Colby and Carrington 1982). Most authors agree so far that there are significant differences and that both entities should be kept separate until new information is obtained on the etiology and pathogenesis of these diseases. The striking vasculitis and the tendency for LYG to spare lymph nodes, spleen, and bone marrow are important differentiating features. It is possible, however, that some cases of LYG could be lymphomas from the beginning. De Remee et al. (1978) and Stamemkovic et al. (1981) who compared LYG with polymorphic reticulosis and more recently Colby and Carrington (1982) who studied 20 primary lung lymphomas simulating LYG suggested that the latter is probably a lymphoma defying recognition by the usual criteria. The polymorphic infiltrates and the polyclonal pattern by immunofluorescence in some reported cases could then be explained by the presence of a secondary inflammatory response to the tumor. Even the vasculitis could be the result of local antigen-antibody (Ag-Ac) complexes in the presence of complement (C^1) . Deposits of IgG and C¹ were found in the vessel walls in our case and these are consistent with an Ag-Ac-C¹ reaction.

Whether LYG is a lymphoproliferative disorder resulting from a chronic antigenic stimulation with lymphoma as a possible outcome or a lymphoma from the beginning remains to be settled. It stresses a need for not only more immunohistochemical studies of large series of LYG but also for prospective in vivo immunologic evaluation. The role of Epstein-Barr virus (EBV) as a possible etiologic factor should also be evaluated as a case of LYG was recently linked with this virus. Veltri et al. (1982) reported a patient with LYG presenting initially as a reactivated EBV infection.

EBV is an ubiquitous B-cell-tropic virus which has been linked with a variety of lymphoproliferative conditions including those arising in renal transplant recipients (RTR). Approximately 80% of RTR secrete EBV in saliva compared to 15-20% in the general population. A large collaborative studies recently demonstrated EBV genome in tissues of all RTR tested (Purtilo 1981). Furthermore, Hanto et al. (1981) showed evidence that EBV was the probable cause of the lymphoproliferative disorders found in six RTR. Although not demonstrated, this is a very likely possibility in our case.

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References

- Bender BL, Jaffe R (1980) Immunoglobulin production in lymphomatoid granulomatosis and relation to other "benign" lymphoproliferative disorders. Am J Clin Pathol 73:41-47
- Bone RC, Vernon M, Sobonya RE, Rendon H (1978) Lymphomatoid granulomatosis. Report of a case and review of the literature. Am J Med 65:709-716
- Cohen ML, Dawkins RL, Henderson DW, Sterrett GF, Papadimitriou JM (1979) Pulmonary lymphomatoid granulomatosis with immunodeficiency terminating as malignant lymphoma. Pathology 11:537-550
- Colby TV, Carrington CB (1982) Pulmonary lymphomas simulating lymphomatoid granulomatosis. Am J Surg Pathol 6:19-32.
- DeRemee RA, Weiland LH, McDonald TJ (1978) Polymorphic reticulosis, lymphomatoid granulomatosis. Two diseases or one? Mayo Clin Proc 53:634-640
- Gardiner GW (1979) Lymphomatoid granulomatosis of the larynx in a renal transplant recipient. J Otolaryngol 8:549-555
- Hammar S, Mennemeyer R (1976) Lymphomatoid granulomatosis in a renal transplant recipient. Hum Pathol 7:111-116
- Hanto DW, Frizzera G, Purtilo DT, Sakamoto K, Sullivan JL, Saemundsen AK, Klein G, Simmons RL, Najarian JS (1981) Clinical spectrum of lymphoproliferative disorders in renal transplant recipients and evidence for the role of Epstein-Barr virus. Cancer Res 41:4253-4261
- Hassoun J, Andrac L, Gambarelli D, Toga M (1981) Lymphomes malins primitifs du système nerveux central. Etude anatomoclinique, ultrastructurale et immunocytochimique. A propos de 23 cas. Ann Anat Pathol (Paris) 1:193-203
- Houthoff HJ, Poppema S, Ebels EJ, Elema JD (1978) Intracranial malignant lymphomas. A morphologic and immunocytologic study of twenty cases. Acta Neuropathol (Berl) 44:203-210
- Jardon-Jeghers C, Reznik M (1982) Etude immuno-histochimique de 16 lymphomes primitifs du système nerveux central. J Neurol Sci 53:331-346
- Katzenstein ALA, Carrington CRB, Liebow AA (1979) Lymphomatoid granulomatosis. A clinico-pathologic study of 152 cases. Cancer 43:360-373

- Kokman E, Billman JK, Abell MR (1977) Lymphomatoid granulomatosis clinically confined to the CNS. A case report. Arch Neurol 34:782-784
- Liebow AA, Carrington CRB, Friedman PJ (1972) Lymphomatoid granulomatosis. Hum Pathol 3:457-558
- Pasquier B, Pasquier D, Gueulle JM, N'Golet A, Panh MH, Couderc P (1980) Granulomatose lymphomatoïde et système nerveux. Aspects anatomo-cliniques et problèmes nosologiques. Rev Neurol 136:205-220
- Pena CE (1977) Lymphomatoid granulomatosis with cerebral involvement. Light and electron microscopic study of a case. Acta Neuropathol (Berl) 37:193–197.
- Penn I (1977) Malignancies associated with renal transplantation. Urology [Suppl] 10:57-63
- Purtilo DT (1981) Malignant lymphoproliferative diseases induced by Epstein-Barr virus in immunodeficient patients, including X-linked, cytogenetic, and familial syndromes. Cancer Genet Cytogen 4:251-268
- Reddick RL, Fauci AS, Valsamis MP, Mann RB (1978) Immunoblastic sarcoma of the central nervous system in a patient with lymphomatoid granulomatosis. Cancer 42: 652-659
- Simon RH, Abeles M, Farber NJ, Grunnet M, Brennan TG (1981) Lymphomatoid granulomatosis with multiple intracranial lesions. Case report. J Neurosurg 55:293-298

- Stamenkovic I, Toccanier MF, Kapanci Y (1981) Polymorphic reticulosis (lethal midline granuloma) and lymphomatoid granulomatosis: identical or distinct entities? Virchows Arch [Pathol Anat] 390:81-91
- Taylor CR (1978a) Immunoperoxidase techniques: theoretical and practical aspects. Arch Pathol Lab Med 102:113-121
- Taylor CR, Russell R, Lukes RJ, Davis RL (1978b) An immunohistological study of immunoglobulin content of primary central nervous system lymphomas. Cancer 41:2197–2205
- Taylor CR (1979) Results of multiparameter studies of B-cell lymphomas. Am J Clin Pathol 72:687-698
- Veltri RW, Raich PC, McClung JE, Shah SH, Sprinkle PM (1982) Lymphomatoid granulomatosis and Epstein-Barr virus. Cancer 50:1513-1517
- Verity MA, Wolfson WL (1976) Cerebral lymphomatoid granulomatosis. A report of two cases, with disseminated necrotizing leukoencephalopathy in one. Acta Neuropathol (Berl) 36: 117-124
- Walter M, Thomson NM, Dowling J, Fox R, Atkins RD (1979) Lymphomatoid granulomatosis in a renal transplant recipient. Aust NZ J Med 9:434-436

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