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Original Article

Ovarian Teratoma Associated With Anti-*N*-Methyl D-Aspartate Receptor Encephalitis: A Report of 5 Cases Documenting Prominent Intratumoral Lymphoid Infiltrates

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Summary: Anti-*N*-methyl D-aspartate receptor (NMDAR) encephalitis is a recently described severe neurological disorder predominantly affecting young women, which presents with psychosis, memory deficits, seizures, and encephalopathy, often requiring prolonged hospitalization. The condition is frequently associated with an underlying neoplasm, most often an ovarian teratoma, and in such cases appears to be a paraneoplastic, immune-mediated encephalopathy. The histologic features of the teratomas associated with anti-NMDAR encephalitis have seldom been described in detail. Therefore, in this report, we have compared ovarian teratomas (4 mature and 1 immature) from 5 patients with anti-NMDAR encephalitis with 22 sporadic control teratomas (14 mature and 8 immature) that included neuroglial elements. The encephalitis-associated tumors ranged from 0.7 to 9.5 cm diameter, and 1 case was bilateral; the second teratoma was discovered 13 mo after the first when symptoms recurred. In comparison with control teratomas, the anti-NMDAR-associated tumors showed a more marked intratumoral lymphoid infiltrate that colocalized to the mature neuroglial elements. Reactive germinal centers (3 cases) and diffuse lymphoplasmacytic infiltrates within the neuroglial matrix (4 cases), and degenerative neuronal changes (2 cases), were seen only in the anti-NMDAR-positive cases. Pathologists encountering ovarian teratomas with these distinctive reactive lymphoid elements should consider the possibility of anti-NMDAR encephalitis, particularly because the neurological symptoms may develop after tumor resection. Careful histopathologic examination may be required to identify small, radiologically occult teratomas, and to demonstrate the presence of subtle neoplastic neuroglial components in teratomas associated with anti-NMDAR encephalitis. **Key Words:** Ovary—Teratoma—NMDA receptor—Encephalitis—Lymphoid—Histopathology.

Anti-*N*-methyl D-aspartate receptor (NMDAR) encephalitis is an uncommon but probably under-diagnosed variant of limbic encephalitis. Patients frequently present with psychosis-like symptoms including hallucinations, agitation, and personality alterations such that initial admission to psychiatric units is common. Disease progression is characterized by seizures, dyskinesis, autonomic dysfunction, and a reduced conscious level, often requiring ventilatory support. Anti-NMDAR encephalitis has been reported in patients between 5 and 76 yr of age, but most commonly affects females in

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the second to fifth decades. In between 20% and 60% of cases, anti-NMDAR encephalitis is associated with an underlying tumor (1,2), most commonly an ovarian teratoma, and interestingly, immature teratomas are disproportionately represented, accounting for 14/49 (29%) cases in the largest series published to date (1). This contrasts with the estimated 3% incidence of immature tumors among ovarian teratomas overall (3). Although a neoplastic association is less frequent in younger patients and in males, occasional cases have also been associated with an immature teratoma of the testis (1,4).

At present, the management of anti-NMDAR encephalitis includes general supportive therapy, a variety of immunomodulatory approaches such as plasmapheresis, corticosteroids, and rituximab, and excision of any associated tumor. The latter appears to improve patient outcomes, and therefore, it is important that appropriate screening for an underlying neoplasm is performed (1). However, it is worth noting that clinically and radiologically occult tumors may be discovered only at autopsy or, in the case of ovarian teratoma, after oophorectomy. Overall, 75% of patients make a full or substantial recovery, although this may take many months while the remainder suffer persisting moderate to severe neurological defects; approximately 4% of cases have a fatal outcome (5,6).

To date, most studies on anti-NMDAR encephalitis have been reported in the clinical literature and the histologic features of the associated tumors have seldom been described in detail. In the past 3 yr, we have encountered 5 patients with ovarian teratomas (1 with bilateral teratomas) and anti-NMDAR encephalitis and have noted an unusually prominent lymphoid infiltrate within some of these tumors. As it seems possible that such lymphoid reactions may be pathogenetically related to the associated encephalitis, and could provide a histologic marker of this association, we present the pathology findings of these 5 cases together with a comparison of lymphoid infiltrates in sporadic (non-anti-NMDAR associated) ovarian teratomas.

CASES AND METHODS

The histologic findings of 5 cases of ovarian teratoma associated with anti-NMDAR encephalitis were reviewed with specific emphasis on the nature and distribution of the intratumoral lymphoid elements. The clinical histories are summarized below. All pathologic specimens were fixed in neutral-buffered formalin and processed routinely to paraffin wax. Immunohistochemistry for glial fibrillary acidic protein, CD3, and CD20 was performed on selected sections to demonstrate the association between the neuroglial tissue and the lymphoid elements. The immunohistochemical stains were performed according to local laboratory protocols with staining of appropriate control sections.

To determine whether the lymphoid populations in the encephalitis-associated tumors differed from those observed in sporadic ovarian teratomas, 22 control cases including 14 consecutive mature cystic teratomas in which neuroglial elements were identified and 8 immature teratomas (2 Grade 1, 2 Grade 2, and 4 Grade 3) were also reviewed.

Anti-NMDAR Encephalitis Case Histories

Case 1

A 17-year-old female presented 4 mo postpartum with acute right iliac fossa pain. Ultrasound examination revealed a complex right ovarian mass, and laparoscopic right ovarian cystectomy was performed. The tumor measured up to 9.5 cm and histology showed a Grade 1 immature teratoma. The neuroglial component of the tumor was predominantly mature. Twelve days after surgery, she represented with acute psychotic symptoms including auditory hallucinations, agitation, insomnia, and aggression. Initially, the patient was admitted to a psychiatric hospital, but following the development of seizures and general deterioration, she was transferred to an intensive care unit (ICU). After the diagnosis of anti-NMDAR encephalitis, right salpingo-oophorectomy and pelvic lymph node biopsies were performed, and these samples were negative for neoplasia. The patient received corticosteroids and rituximab therapy. After a 51-d ICU admission, she was discharged with nearly complete neurologic recovery after 4 mo.

Case 2

A 37-year-old female presented 3 mo postpartum with pyrexia of unknown origin, headache, agitation, and behavioral disturbance. Her general condition deteriorated requiring ICU admission and ventilatory support. On examination, she had generalized lymphadenopathy but no other abnormal clinical or radiologic findings. After the diagnosis of anti-NMDAR encephalitis, she received plasmapheresis together with corticosteroids and intravenous (IV) immunoglobulin therapy. There was no improvement and therefore

exploratory laparotomy and bilateral oophorectomy were performed 6 wk after presentation. Histologic examination of the right ovary showed a solid mature teratoma measuring 0.7 cm in which there was a single focus of neuroglial tissue. The left ovary was normal. The patient remained encephalopathic and in ICU for a total of 77 d, but she gradually improved and was discharged after 100 d with partial neurological deficit.

Case 3

A 27-year-old female was referred with multiple generalized tonic clonic seizures over the preceding 10 d and a 2-wk history of abnormal behavior, agitation, and uncharacteristic anxiety. Within days, she became encephalopathic with orofacial dyskinesias, and anti-NMDAR encephalitis was diagnosed. A right ovarian teratoma was found on computed tomography of the pelvis, the left ovary appearing normal at this stage. She underwent right oophorectomy and immunomodulatory therapy including IV and oral corticosteroids, IV immunoglobulin, and plasmapheresis. Histopathology revealed a 3.5 cm mature cystic teratoma but neuroglial elements were not identified. During treatment, she required ICU support over a 4-wk period for autonomic instability and airway management. The anti-NMDAR antibody titer dropped in response to treatment in accordance with clinical improvement, and she was discharged 3 mo after presentation. At this stage, she was independently mobile, communicating, and self-caring, and she subsequently returned to work. Thirteen months after her initial presentation, the patient again developed abnormal behavior, agitation, and anxiety, progressing to a catatonic state within a week. A computed tomography scan of the pelvis revealed an abnormal left ovary suggestive of a teratoma and she underwent partial oophorectomy and further IV immunoglobulin and plasmapheresis. Histology revealed a mature cystic teratoma with a single focus of neuroglial tissue. She remains in ICU 2 mo after representation, but is beginning to improve.

Case 4

A 19-year-old female presented with a 4-d history of confusion, disorientation, memory loss and agitation, progressing to encephalopathy that required admission to ICU for ventilatory support. A diagnosis of anti-NMDAR encephalitis was made. Pelvic magnetic resonance imaging identified a right ovarian lesion and the patient underwent a right salpingo-oophorectomy 11 d after her initial presentation. Histopathology showed a 2.5 cm mature cystic teratoma including neuroglial elements. Encephalopathy persisted after removal of the tumor and the patient received plasmapheresis, rituximab, corticosteroids, and IV immunoglobulin therapy. She remained in hospital for 143 d, but slowly improved and was eventually discharged to a rehabilitation unit with a moderate residual cognitive defect.

Case 5

A 17-year-old female presented with headache, fever, photophobia, and neck stiffness. She deteriorated rapidly, requiring ICU admission after 4 d, and thereafter, her course was characterized by emotional instability, abnormal behavior, increasing confusion, and agitation. She was commenced on IV immunoglobulin and corticosteroids, but then developed recurrent generalized tonic-clonic seizures and severe dyskinesia in the face and arms. Seven days after admission, laparoscopic right salpingo-oophorectomy was performed and histologic examination showed a 1.8 cm mature solid teratoma including neuroglial tissue. Rituximab therapy was introduced after the diagnosis of anti-NMDAR encephalitis. The patient remained in ICU for more than 6 mo and made a slow partial recovery with residual neurological deficits including dysphagia, bowel and bladder incontinence, and dystonic movement disorder. She also had severe cognitive impairment, emotional lability, and impaired behavior control. Although these symptoms had improved significantly 2 y later, the patient still suffered from oral dyspraxia requiring percutaneous enterostomy feeding and she also had persisting communication and behavioral difficulties.

RESULTS

The pathologic findings in the five anti-NMDAR-positive cases and the 22 sporadic teratomas are summarized in Table 1.

All 5 anti-NMDAR-associated teratomas included neuroglial tissue although this was identified only in the second tumor in the patient with bilateral teratomas (Case 3) after recurrence of the neurological symptoms after initial surgery. Four of the teratomas showed only mature elements, whereas 1 tumor (Case 1) included a focal immature neuroglial component consistent with a Grade 1 immature teratoma. The most striking histologic feature in these tumors was the presence of prominent lymphoid aggregates, which in 3 cases included large reactive germinal centers

TABLE 1. *Comparison of lymphoid infiltrates in 5 ovarian teratomas associated with anti-NMDAR-associated encephalitis and 22 control tumors*

Case/pathology	LA adjacent to NG tissue	Germinal centers in NG tissue	Diffuse LP infiltrates in NG tissue	Degenerative neuronal changes
Anti-NMDAR cases				
1. Grade 1 immature teratoma	Yes (mature NG elements only)	Yes	Yes	Yes
2. Mature solid teratoma	Yes	No	Yes	No
3. Mature cystic teratoma (bilateral)	Yes	No	No	No
4. Mature cystic teratoma	Yes	Yes	Yes	Yes
5. Mature solid teratoma	Yes	Yes	Yes	No
Control cases				
Mature teratomas n = 14	3/14 cases	No	No	No
Immature teratomas n = 8	1/8 cases	No	No	No

LA indicates lymphoid aggregate; LP, lymphoplasmacytic; NG, neuroglial; NMDAR, *N*-methyl D-aspartate receptor.

(Figs. 1, 2). The lymphoid aggregates and germinal centers showed a distinct microanatomic localization with close apposition to the neuroglial tissue; germinal centers were not identified in other tumor areas. In 4 cases, there was also a more diffuse lymphoplasmacytic infiltrate that extended into the neuroglial matrix and focally surrounded neuronal and ganglion cells some of which showed degenerative appearances (Fig. 2). In the single immature teratoma in this series (Case 1), the lymphoid elements were associated only with the mature neuroglial tissue. The smallest tumor (0.7 cm diameter, Case 2) included a single lymphoid aggregate, but this was situated adjacent to the only focus of neuroglial tissue (Fig. 3). Immunohistochemistry for CD20 and glial fibrillary acidic protein highlighted the colocalization of the B-cell aggregates and the neural elements in all of the teratomas (Figs. 1–3), whereas CD3 staining showed a less conspicuous and more dispersed population of small T-lymphocytes within the adjacent stroma. All tumors also showed less prominent epithelial-associated lymphoid infiltrates similar to those observed within the control cases described below.

Review of the non-NMDAR-associated tumors showed lymphoid elements in 12/14 mature teratomas and in all 8 immature teratomas. The lymphoid tissue, including small lymphoid aggregates, was associated most commonly with cysts lined by squamous or respiratory-type epithelium, and there was often a focal lymphocytic infiltration around cutaneous-type adnexal glands (Fig. 4). Germinal centers were seen in only 2 tumors (1 mature teratoma and 1 immature teratoma) where they were related to respiratory and gastrointestinal type epithelium, and not to neuroglial tissue. Small stromal lymphoid aggregates were also present in 7 tumors, and in 4 cases, these focally abutted mature neuroglial elements; 1 of the latter tumors also included clusters of perivascular lymphoid cells within the glial matrix (Fig. 4). However, none of the "control" cases showed germinal centers or diffuse lymphoplasmacytic infiltrates within or adjacent to the neuroglial tissues nor were there obvious degenerative changes within the neuronal/ ganglion type cells.

DISCUSSION

Anti-NMDAR encephalitis is a recently characterized syndrome that is frequently associated with an underlying neoplasm, most commonly an ovarian teratoma. It differs from most other paraneoplastic neurological disorders in that the patients are typically young women and the clinical presentation is dominated by psychiatric-type symptoms, which may delay the correct diagnosis. This is illustrated by 1 of the patients presented herein who initially was thought to have a postpartum psychosis. Anti-NMDAR encephalitis also differs from other paraneoplastic disorders in that the associated tumors are usually benign, or in the case of immature teratomas are still likely to be curable with surgery and chemotherapy. Thus, early diagnosis and treatment of the underlying neoplasm is important, particularly because this appears to improve the neurological outcomes. In the patients with ovarian teratomas, the tumors may be discovered on clinical or radiological examination after the onset of encephalitis, but some patients present after tumor resection as in one of the cases in this report. Neurological symptoms may also persist despite removal of the tumor, and in 1 case in this series, recurrence of symptoms was associated with a diagnosis of teratoma in the contralateral ovary, a feature that has been reported only rarely in the literature (1,5); interestingly, neuroglial tissue was identified only in the

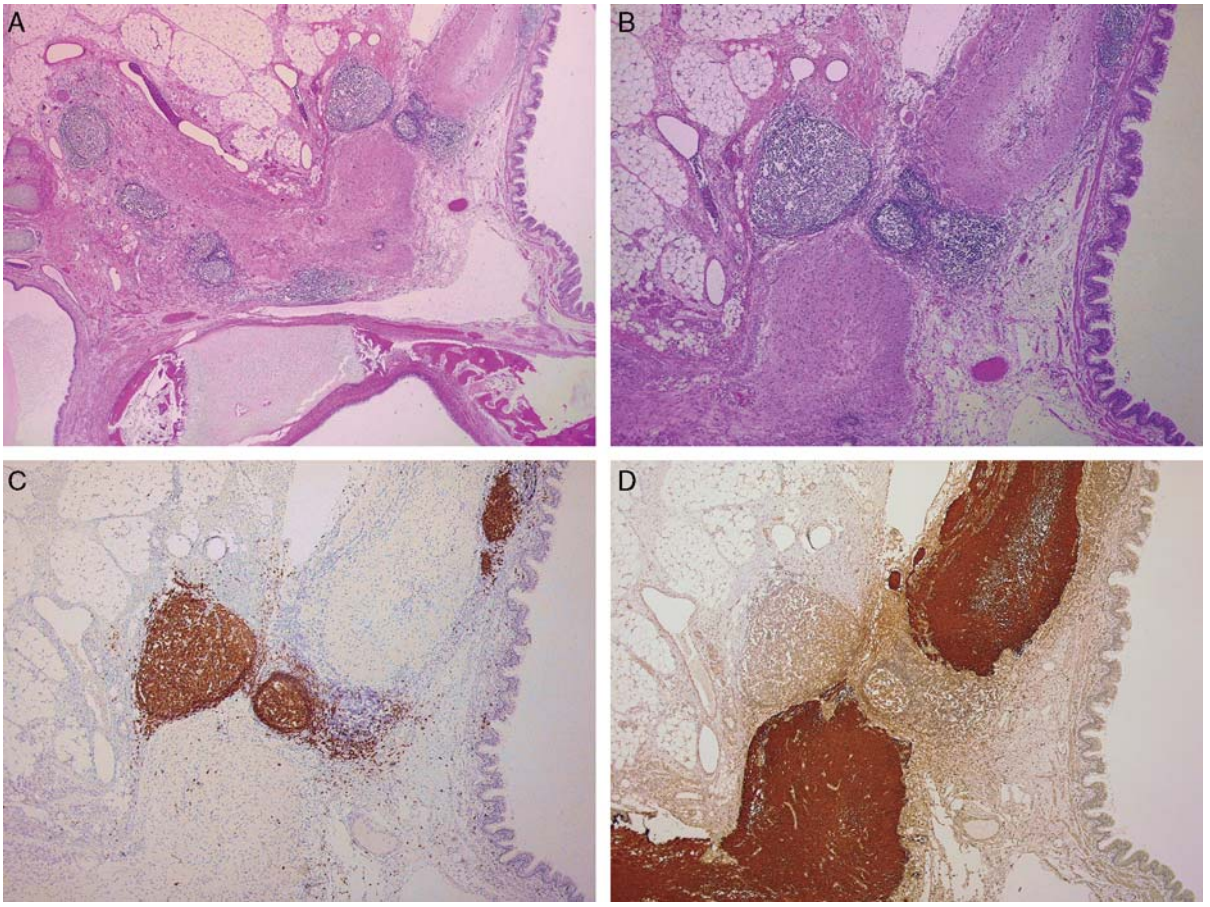

FIG. 1. Case 4. Scanning magnification (A) shows mature cystic teratoma with prominent lymphoid aggregates associated with neuroglial tissue. At a higher magnification, (B) reactive germinal centers are apparent, while immunohistochemistry for glial fibrillary acidic protein (C) and CD20 (D) highlights colocalization of the neuroglial tissue and B cells, respectively.

second tumor in our patient. Ovarian teratomas associated with anti-NMDAR encephalitis are usually unilateral, but approximately 15% of the cases are bilateral (1,7–10). From a pathologic perspective, it is also worth noting that the ovarian tumors may be small and radiologically occult, thus necessitating extensive or complete sampling of ovarian tissues where no macroscopic lesion is evident. In earlier reports, the ovarian teratomas associated with anti-NMDAR encephalitis have ranged from 1 to 22 cm in size with a mean diameter of 6 cm (1), but 1 tumor in the present series was only 0.7 cm diameter, similar to the smaller of bilateral teratomas reported recently by Frawley et al. (8); to our knowledge, these 2 cases are the smallest teratomas associated with anti-NMDAR encephalitis yet recorded.

Although the association between anti-NMDAR encephalitis and neoplasia is now well established, most reports in the literature have not provided detailed descriptions of the tumor pathology. In the patients with ovarian teratoma, it has been noted that mature or immature neuroglial tissues are almost always present as found in the current study (1,5,6,11). In contrast, neuroglial tissue has been identified in 30% to 50% of otherwise typical mature cystic teratomas (12). Tuzun et al. (6) examined 5 ovarian teratomas from patients with anti-NMDAR encephalitis and tumors from 3 patients without encephalitis. The tumors from patients with encephalitis were reported to show extensive inflammatory infiltrates that were composed mainly of T cells and macrophages. B-cell elements were described as less abundant but were relatively more conspicuous than in the nonencephalitis cases. There was no mention of germinal centers, and the microanatomic relationship between the lymphoid and the neuroglial elements was not specified. However, in the nonencephalitis tumors, it was noted that the inflammatory infiltrates were not

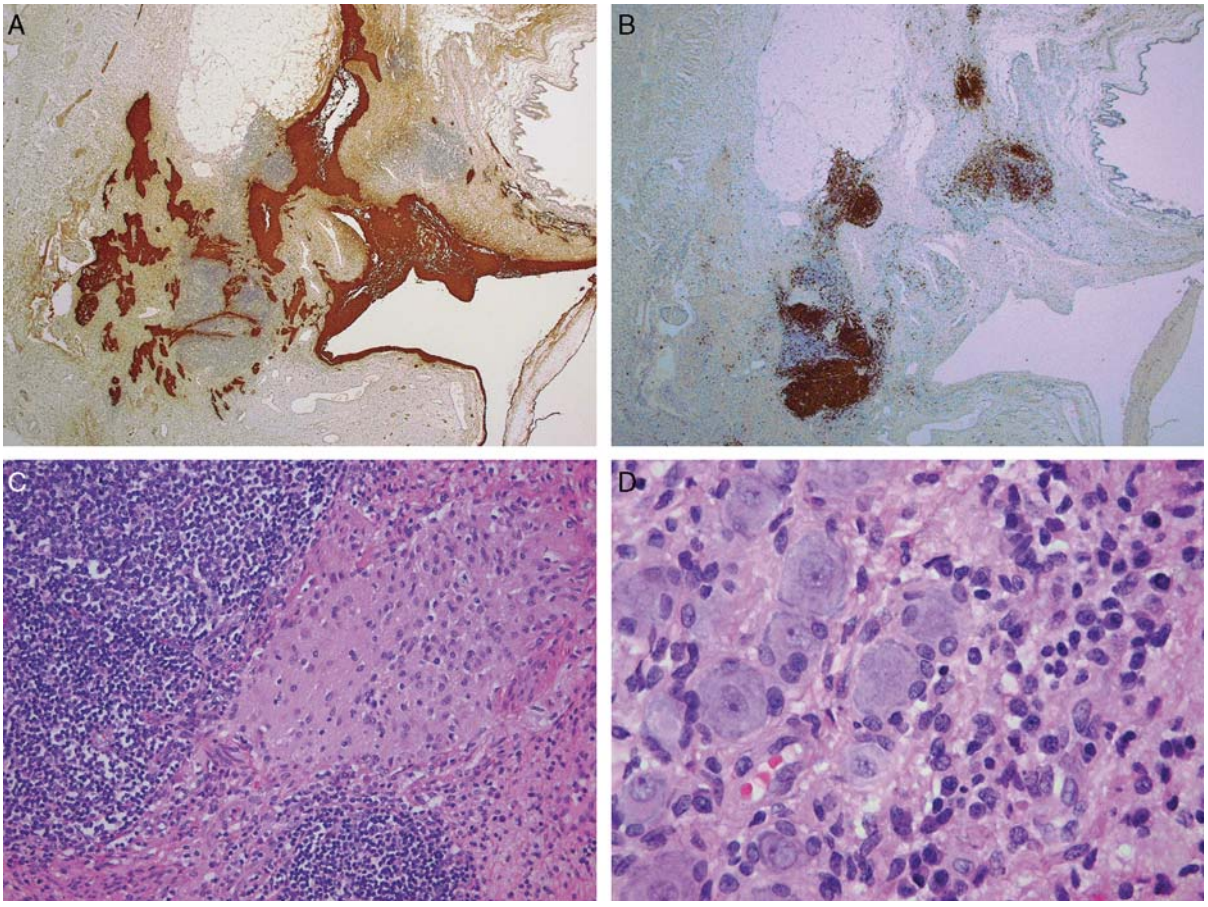

FIG. 2. Case 1. Prominent lymphoid aggregates and mature neuroglial elements are evident at low magnification and are highlighted by immunohistochemistry for glial fibrillary acidic protein (A) and CD20 (B). Higher magnification (C) shows close apposition of the 2 cellular components and the presence of focal lymphoplasmacytic infiltrates around ganglion cells (D).

confined to the neuroglial tissue. The authors also studied autopsy brain tissue from two patients with anti-NMDAR encephalitis. The major findings were microgliosis, neuronal degeneration, and rare inflammatory infiltrates in the hippocampus, forebrain, basal ganglia, and spinal cord. Additional autopsy studies have also shown perivascular lymphocytic infiltrates in the parahippocampal regions, thalami, insula, and medulla (5,6). The lymphocytic infiltrates were predominantly B cells, with $<1\%$ of cells staining as cytotoxic T cells. This differs from most autoimmune paraneoplastic encephalitides, which are predominantly T-cell mediated. Intense deposits of immunoglobulin were also identified in the hippocampus, and at a much lesser degree in other brain regions (5,6)

The histologic findings in the present series of anti-NMDAR-associated ovarian teratomas agree to some extent with those reported by Tuzun et al. (6) in that all tumors showed lymphoid (inflammatory) infiltrates. However, there were some differences and additional features worthy of comment. First, the degree of lymphoid reaction in the tumors was unusual and often striking at low-power magnification, especially in the 3 cases in which reactive germinal centers were present. Second, the lymphoid tissue was not randomly distributed but rather was concentrated within and around the neuroglial elements. This was evident in the smallest tumor in which a single lymphoid aggregate immediately abutted the only focus of neuroglial tissue that was present in the tumor. Third, 4 tumors showed a more diffuse lymphoplasmacytic infiltrate within the neuroglial tissue and in 2 cases this was associated with degenerative changes in neuronal-type cells. Finally, immunohistochemistry demonstrated that the lymphoid aggregates (including those without germinal centers) mainly comprised CD20-positive B cells, whereas CD3-positive T cells were less numerous. In contrast, in ovarian teratomas

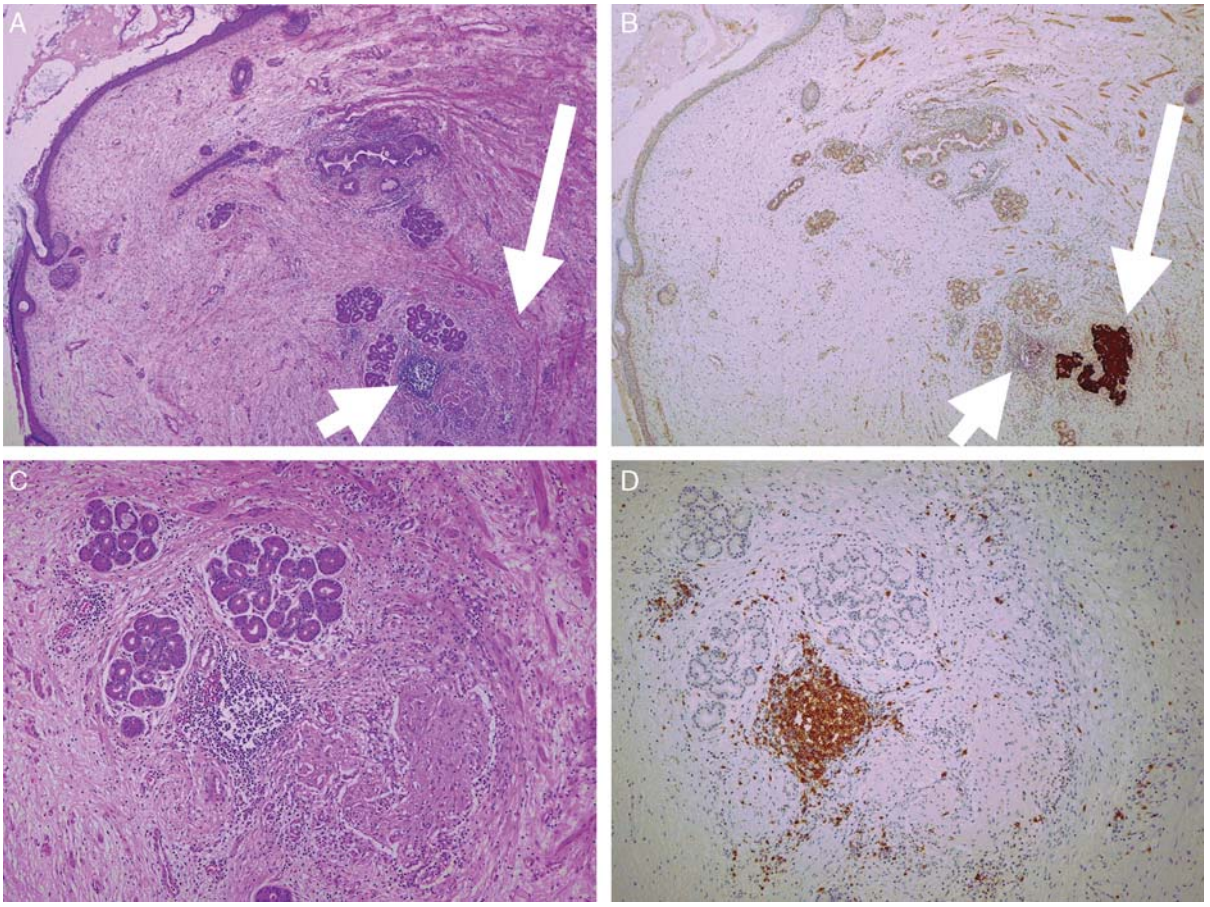

FIG. 3. Case 2. Scanning magnification (A) shows mature teratoma with a single lymphoid aggregate (short arrow). Neuroglial tissue (large arrow) is not obvious on the H&E-stained preparation, but is highlighted by glial fibrillary acidic protein immunostaining (B). Higher magnification (C) shows the close apposition of the 2 cellular components, whereas CD20 immunostaining (D) demonstrates B cells within the lymphoid aggregate and adjacent stroma.

from patients without encephalitis, the lymphoid aggregates were almost always related to epithelial elements, typically cysts lined by squamous or respiratory-type epithelium. None of these "control" tumors showed neuroglial-associated germinal centers or diffuse lymphoplasmacytic infiltrates, and degenerative neuronal changes were not identified. Thus, there appear to be quantitative and qualitative differences in the immune reaction within ovarian teratomas associated with anti-NMDAR encephalitis compared with otherwise sporadic tumors. These findings are similar to those described in neuroblastic tumors from children with opsoclonus-myoclonus syndrome, which is also thought to represent a paraneoplastic, immune-mediated neurological disorder. It has been shown that the tumors in patients with opsoclonus-myoclonus syndrome commonly show diffuse and extensive lymphoid infiltrates (13,14).

The pathogenesis of anti-NMDAR-associated encephalitis is not fully understood, but current evidence suggests that this is primarily a humoral-mediated autoimmune reaction in which autoantibodies to the NMDAR (specifically its NR1 and NR2 subunits) produce neuronal damage. The potential role of the ovarian tumors in inducing or propagating the immune reaction in these cases is of interest. It is possible that the ovarian lymphoid infiltrate represents a secondary and essentially passive immune reaction as the neoplastic neuroglial tissue expresses auto-antigens similar to those present in the brain (6). A similar mechanism could explain the lymphocytic infiltrates occasionally observed within struma ovarii in patients who have thyroid autoantibodies or clinical Hashimoto disease (15). Conversely, however, and as suggested by Dalmau et al. (16), the "ectopic" expression of NMDAR subunits within neoplastic

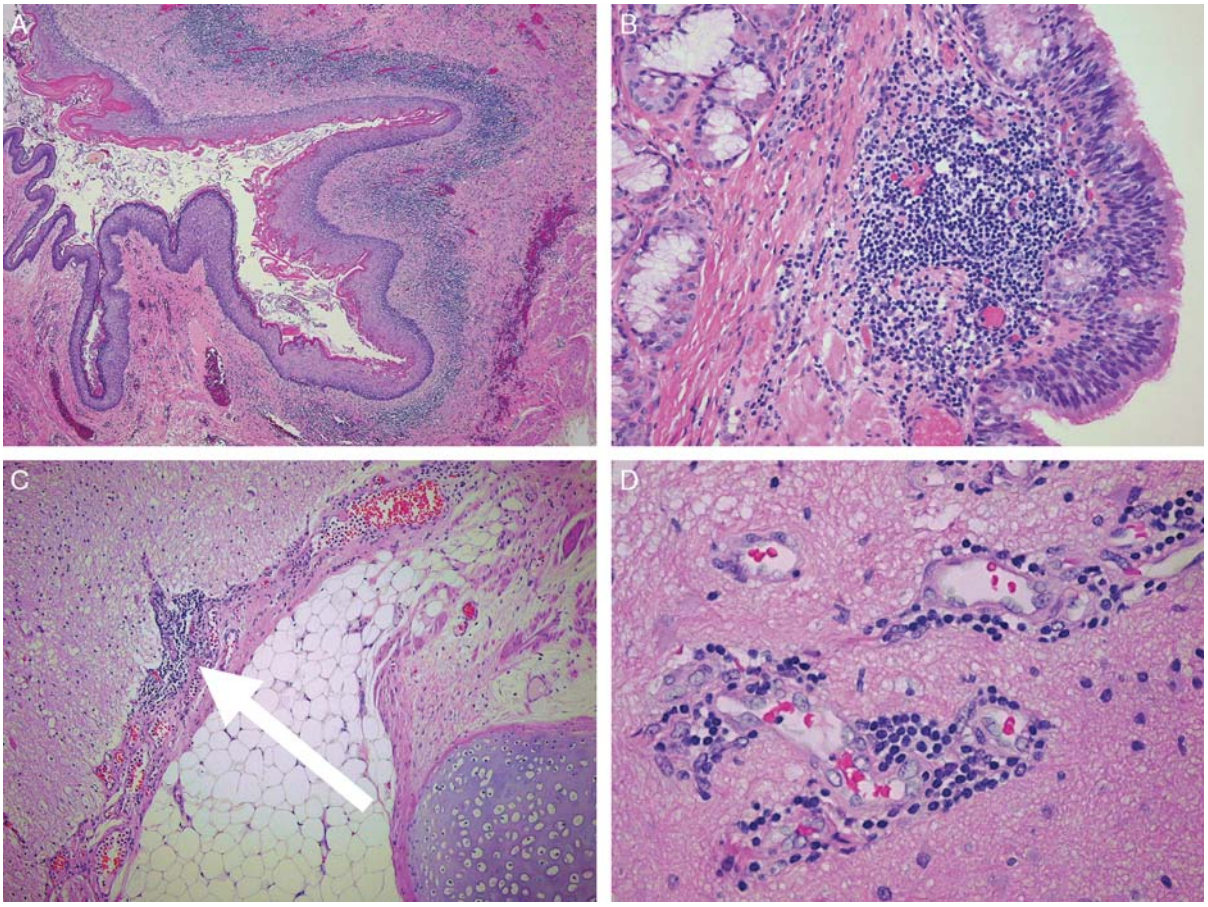

FIG. 4. Sporadic teratomas (nonencephalitis cases). Lymphoid cells are commonly identified around cysts lined by squamous (A) and respiratory-type epithelium (B). Small lymphoid aggregates (arrow) without germinal centers are seen occasionally at the margins of neuroglial tissue (C), whereas one case showed perivascular lymphoid cells within the neuroglial matrix (D).

neuroglial tissues might contribute toward a break in immune tolerance and thus play an active role in the development of the encephalitis. Interestingly, in the single immature teratoma encountered in this series, the lymphoid reaction was associated only with the mature neural elements, suggesting that the immature neuronal components did not express the auto-reactive NMDAR.

In summary, the histologic features of ovarian teratomas from 5 patients associated with anti-NMDAR encephalitis are presented. In comparison with sporadic teratomas, encephalitis-associated tumors show a more marked intratumoral lymphoid infiltrate that colocalized to histologically mature neuroglial elements. Reactive germinal centers, diffuse lymphoplasmacytic infiltrates, and degenerative neuronal changes were seen only in anti-NMDAR-positive cases. Although larger studies are required to confirm the specificity of these histologic findings, pathologists encountering ovarian teratomas with these distinctive features should consider the possibility of anti-NMDAR encephalitis, particularly because the neurological symptoms may develop after tumor resection.

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