Several drugs can induce meningitis, resulting in a diagnostic and therapeutic challenge. The situation becomes more complex if the offending drug is an antibiotic, where the decision of withdrawing the drug needs to be weighed against the risk of missing the treatment of an underlying infectious disorder. Furthermore, these drugs are often used to treat disorders that in turn may cause meningitis. Therefore, physicians should be aware of this possibility and a careful drug history should be obtained in every case of meningitis that could obviate inappropriate therapies, such as prolonged antimicrobial therapy or high-dose steroids.

The incidence of drug-induced meningitis is unknown. Recent series have allowed for an approximation to the extent of the problem. Four groups of drugs have been associated with drug-induced aseptic meningitis (DIAM) (Table 1): nonsteroidal anti-inflammatory drugs (NSAIDs),1-92 antibiotics,93-73 intravenous immunoglobulins (IVIGs),74-99 and OKT3 monoclonal antibodies (directed against the T3 receptor and, therefore, pan T-cell antibodies).100-119 These drugs are frequently prescribed by different specialists (rheumatologists, neurologists, internists, etc), who should be aware of this problem. The internal medicine literature, however, lacks a review on this topic. Thus, our purpose was to review critically those published cases of meningitis associated with the use of drugs. We have compared the characteristics associated with the different drugs to see if differences exist among them so as to suggest a specific drug as the culprit of the disorder and to distinguish it from other types of meningitides.

Using the MEDLINE database, we searched the literature up to June 1998 and have included only those cases that speci-
fied the cerebrospinal fluid (CSF) findings and provided sufficient information to exclude other causes. We only included cases that met the following criteria: a temporal relationship with the drug intake, CSF pleocytosis (>5 cells/mm³) and negative cultures, absence of any other explanation for the meningitis, and the resolution of the syndrome on drug withdrawal. We discarded cases with normal or absent CSF parameters (2 cases each by Sekul and colleagues⁸²) as well as a case of pachymeningitis associated with penicillin therapy,³² and 1 case of myelopathy and brainstem dysfunction with CSF pleocytosis after trimethoprim-sulfamethoxazole administration suggestive of systemic lupus erythematosus (SLE) exacerbation (case 2).⁵⁵

### CLINICAL CHARACTERISTICS AND CSF PROFILES OF PATIENTS WITH DIAM

The vast majority of patients with DIAM, irrespective of the offending drug, presented with headache, fever, meningismus, and changes in mental status (Table 2). This is also characteristic of infectious meningitis,¹²¹ and, therefore, the clinical presentation does not help in differentiating DIAM from infectious meningitis. Other findings less frequently reported included rash, arthralgias, myalgias, facial edema, and lymph node or liver test abnormalities, which can also occur in infectious meningitis, mainly of viral origin, with variable frequency.

There are statistically significant differences in the presentation of the meningitides induced by the different drugs, but these involve nonspecific symptoms that may be accounted for by differences in data collection. For instance, patients in the OKT3 monoclonal antibody group had less vomiting and meningismus and those in the antibiotic group had more abnormalities of consciousness, but we do not believe that these data differentiate or suggest a specific drug as the culprit. Overall, seizures were recorded in 7% to 16% of the patients. Although it is well known that some antibiotics can cause seizures,¹²² in this case they cannot be attributed to the drugs, since they also appear in up to 20% of patients with bacterial meningitis irrespective of therapy.¹²³

The interval between drug intake and the development of meningitis varies between several minutes and 4 months for all patients in the drug groups and prior exposure to the drug is present in 45% of patients taking NSAIDs; antibiotics and IVIGs, 35%; and OKT3, 3% (Table 1). This rate of prior exposure can be anticipated considering the high frequency with which NSAIDs and antibiotics are prescribed and that IVIGs are usually used periodically to treat recurrent disorders such as idiopathic thrombocytopenic purpura. On the contrary, OKT3 antibodies are used for cases of transplant rejection, which gives the patient little opportunity for prior exposure. There was no relation between the development of DIAM and the dose of the drug used, which was always within the recommended therapeutic range.

The CSF of patients with DIAM typically shows pleocytosis of sev-
eral hundred to several thousand cells per cubic millimeter, normal-to-low glucose values, and increased protein values (Table 3). The number of cells was significantly lower in the OKT3 group ($P = .01$, Kruskal-Wallis analysis of variance [ANOVA]) than in the other 3 groups. The protein values were significantly lower in the IVIG and OKT3 groups compared with the other 2 groups ($P = .01$, Kruskal-Wallis ANOVA). Characteristically, neutrophils predominate, with a percentage more than the total cell count that varies between an average of 57% in OKT3 and 78% in IVIG. Eosinophils were noted in 9 patients: 2 patients from the NSAIDs group (between 13% and 60% of the total cell count),8,41 3 from the antibiotic group (2% and 24%, respectively; no counts were given in the third case),51,61,69 and 4 in IVIG (1%, 3%, 3%, and 100%, respectively). The degree of pleocytosis correlated directly with the severity of fever and inversely with the interval from drug exposure in a series of OKT3-associated DIAM, but neither association reached statistical significance.110

When performed, neuroimaging was normal in all patients except for 2 with NSAID-induced DIAM where diffuse contrast hemispheric enhancement was evident (by magnetic reso-
nance imaging [MRI] in one case and by computed tomographic [CT] scan in the other), probably reflecting a blood-brain barrier breakdown. In 2 additional patients with antibiotic-related meningitis, MRI showed bilateral supratentorial white matter T2-signal abnormalities without gadolinium enhancement with complete resolution in several months.

Neuroimaging was performed in 8 patients with OKT3-associated DIAM and was abnormal in 3: a patient showed cerebral edema on brain CT scan and MRI disclosed high intensities on T2-weighted sequences in 2 additional patients that disappeared in 10 days. In 10 patients, meningitis was associated with OKT3 in 11 (2 episodes), and in the other, probably reflecting a blood-brain barrier breakdown. In 2 additional patients with antibiotic-related meningitis, MRI showed bilateral supratentorial white matter T2-signal abnormalities without gadolinium enhancement with complete resolution in several months.

The outcome was always excellent provided that the offending drug was withdrawn, with complete recovery in all cases. In the case of OKT3, it resolved even without withdrawal in 45% of the patients.

**UNDERLYING CONDITIONS IN PATIENTS WITH DIAM**

Systemic lupus erythematosus stands as the single most frequent underlying condition associated with DIAM (Table 4). We pooled and analyzed the clinical and CSF profiles of this group of patients to see if they shared any special or distinctive characteristic. The predominance of females was marked (90%), as expected for SLE, but there were no other obvious indicators (Tables 1, 2, and 4).

Although migraine has been suggested as a predisposing condition to DIAM and reported in several patients, the retrospective analysis of these heterogeneous case reports does not allow for the determination of the exact prevalence of prior, potentially predisposing conditions such as migraine. To further complicate matters, the high prevalence of migraine in healthy populations (6%-12%) and its even higher prevalence in populations also prone to DIAM, such as patients with SLE, should be considered.

**RECURRENT DIAM**

We found 29 patients with recurrent DIAM, totaling 71 episodes, and analyzed their clinical and CSF characteristics (Table 5). In 8 patients, meningitis was associated with NSAIDs in 15 (1 episode), antibiotics in 18 (2 episodes), and IVIGs in 2 (4 episodes). The highest number of episodes reported in a single patient was 5, but we have only analyzed the first 3 episodes due to the lack of information on fourth and fifth episodes. Overall, there was a female predominance (81%) with a mean age of 40 ± 22 years (range, 2-82 years).

### Table 4. Underlying Disorder in Patients With Drug-Induced Aseptic Meningitis*

<table>
<thead>
<tr>
<th>Drug Group, No. (%)</th>
<th>Common Underlying Condition, No. (%)</th>
<th>Uncommon Underlying Condition, No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mixed connective tissue disease, 4:7:25:34:37</td>
<td>6 (14)</td>
</tr>
<tr>
<td>Antibiotics, 14 (37)</td>
<td>SLE, 46:2:5:8:14:10 (10)</td>
<td>10% of cases</td>
</tr>
<tr>
<td></td>
<td>HIV infection, 17:21:85:10 (10)</td>
<td>10% of cases</td>
</tr>
</tbody>
</table>

* The different underlying conditions were arbitrarily split into common (>10% of cases) and uncommon (<10%). NSAIDs indicates nonsteroidal anti-inflammatory drugs; SLE, systemic lupus erythematosus; HIV, human immunodeficiency virus; IVIGs, immunoglobulins; CIDP, chronic inflammatory demyelinating polyneuropathy; and OKT3, monoclonal antibodies against the T3 receptor. Ellipses indicate data not applicable.

† The patient had SLE.

‡ One of the patients had lupus nephropathy.
Differential Diagnosis

The differential diagnosis of DIAM is broad and includes infectious causes. A meningeal syndrome accompanied by CSF neutrophilic pleocytosis suggests acute bacterial meningitis, which needs to be ruled out by CSF culture. More than 85% of the patients with bacterial meningitis present with fever, headache, meningismus, and signs of cerebral dysfunction (ie, confusion, delirium, or a declining level of consciousness ranging from lethargy to coma).126,127

Therefore, distinction on clinical grounds alone is not possible. Even more problematic is the case of a patient treated with an antibiotic who develops meningitis and in whom the possibility of a partially treated meningitis should be considered first but needs to be separated from an antibiotic-related DIAM. Again, these entities cannot be distinguished on clinical and CSF grounds alone. Of help may be to consider the type of antibiotic, since DIAM is mainly caused by certain antibiotics (Table 1). In cases where bacterial meningitis is a possibility, we suggest the patient be treated with third-generation cephalosporins, which are known to cause DIAM only exceptionally72 and that would be active against the most frequent organisms in a healthy individual until the appropriate CSF studies are available. Although corticosteroids have been used in several patients, their effectiveness in DIAM has not been proven and therefore cannot be recommended routinely.

Since recovery on drug discontinuation is the rule, chronic infectious meningitis (tuberculous, fungal, etc) would only rarely pose a diagnostic problem. If such is the case, appropriate CSF studies (culture in appropriate media and adequate stains) will be necessary. Meningitis due to parasites may need to be ruled out in those cases with CSF eosinophilia that occur in the appropriate epidemiological context.

Viral aseptic meningitis is another important consideration in terms of frequency, although less critical in terms of prognosis and management. Clinically, it is marked by fever (76%-100%), nuchal rigidity, and headache that may be accompanied by vomiting, rash, diarrhea, pharyngitis, arthralgias, and myalgias.126 Neutrophils may occasionally dominate the CSF profile early in the infection, although there is usually a shift to lymphocytic predominance during the first 48 hours. Cerebrospinal fluid glucose levels are usually normal. Again, clinical and CSF overlapping with DIAM may occur. It could also be argued whether certain cases of DIAM would in fact correspond to viral meningitis considering the difficulty of making a definitive diagnosis in viral infections. Polymerase chain reaction could have been of help in elucidating a possible viral origin,129 but this information was not available in any of the reported cases, since most of them predate the use of polymerase chain reaction. In any case, the time to recovery after drug withdrawal may be of help, since it is rapid in DIAM (1-5 days) but usually takes 10 to 14 days in viral meningitis.

Many other noninfectious causes of aseptic meningitis exist, an important fact considering that many patients with DIAM harbor underlying systemic disorders that may cause meningitis and that can also predispose them to neurologic infections by diverse organisms. In the setting of SLE, DIAM needs to be specifically distinguished from lupus aseptic meningitis. Although signs of meningeal inflammation are present at autopsy in 18% of patients with SLE,130 aseptic meningitis is infrequently diagnosed, since the clinical presentation can be overwhelmed by other neuropsychiatric manifestations. Unlike DIAM, the cellular infiltrate of the CSF in lupus meningitis is usually lymphocytic (<50 cells) and is accompanied by other data consistent with a lupus flare-up.131,132 In turn, DIAM could mimic a drug-induced exacerbation in SLE if accompanied by systemic manifestations. The rapid onset and resolution of the signs and symptoms as well as the lack of data of SLE activity, especially a fall in serum complement levels, argue against an exacerbation of SLE.1

Antibiotics are probably under-recognized as etiologic agents of

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*CSF indicates cerebrospinal fluid.

†To convert glucose values from milligrams per deciliter to millimoles per liter, multiply milligrams per deciliter by 0.055 51.

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Table 5. CSF Characteristics in Recurrent Episodes of Drug-Induced Aseptic Meningitis*

<table>
<thead>
<tr>
<th>Episode</th>
<th>Latency Range (Median)</th>
<th>Cells/mm³, Predominant Cells, %</th>
<th>Predominant Cells, %</th>
<th>Median Glucose Value (Range), mmol/L</th>
<th>Median Protein Value (Range), g/L</th>
<th>Median Time to Recovery (Range), h</th>
</tr>
</thead>
<tbody>
<tr>
<td>First†</td>
<td>20 min to 4 mo (18 h)</td>
<td>Lymphocytes, 15 Neutrophils, 85</td>
<td></td>
<td>3.16 (1.50-4.05)</td>
<td>0.88 (0.15-2.19)</td>
<td>72 (24-336)</td>
</tr>
<tr>
<td>Second</td>
<td>20 min to 14 d (2.5 h)</td>
<td>Lymphocytes, 24 Neutrophils, 76</td>
<td></td>
<td>3.28 (0.56-6.27)</td>
<td>1.22 (0.21-2.84)</td>
<td>48 (12-168)</td>
</tr>
<tr>
<td>Third</td>
<td>2-14 d (15 h)</td>
<td>Lymphocytes, 14 Neutrophils, 86</td>
<td></td>
<td>3.44 (1.78-11.93)</td>
<td>1.45 (0.97-3.08)</td>
<td>48 (40-120)</td>
</tr>
</tbody>
</table>

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*CSF indicates cerebrospinal fluid.

†To convert glucose values from milligrams per deciliter to millimoles per liter, multiply milligrams per deciliter by 0.055 51.
meningitis and must be considered also in the differential diagnosis of recurrent meningitis, particularly in relation to anatomical skull defects, parameningeal infectious foci, immunodeficiencies, and Mollaret meningitis.  

The abrupt onset in some patients with DIAM may suggest intracranial bleeding, especially in patients with idiopathic thrombocytopenic purpura and low platelet counts. Computed tomographic scans can be used to rule out hemorrhage.  

Cerebrospinal fluid pleocytosis may occasionally accompany migraine, but the cell count is almost always lymphocytic and rarely exceeds 100 cells/mm³. Since NSAIDs are commonly used to treat migraines and can lead to DIAM, these drugs could play a role in producing pleocytosis, but this aspect has never been assessed systematically. Some patients develop the “pseudo-migraine with pleocytosis” syndrome, which is characterized by variable neurologic deficits, headache, fever, and lymphocytic pleocytosis. The predominance of lymphocytic pleocytosis in the CSF and the concomitant focal neurologic deficit (present in 86% of patients with pseudo-migraine but only in 10%-16% of DIAM) might be of help to differentiate this entity from DIAM. As for migraine, the hypothetical role of drugs, such as NSAIDs, should be assessed in pseudo-migraine with pleocytosis. The only confirmatory test of DIAM would be a rechallenge with the drug, which has been previously reported in the literature but does not seem ethically justified.  

PATHOGENESIS

The pathogenic mechanisms of DIAM are not fully understood, but there is evidence to suggest that they may be diverse, perhaps different for the various types of drugs. Most of the authors invoke a hypersensitivity mechanism (especially type 1 and 3) for NSAID-, IVIG- and antibiotic-related cases. OKT3 cases are likely mediated, at least in part, by cytokine release. However, it is striking that such reactions are mainly or exclusively confined to the CSF compartment.

NSAID-INDUCED MENINGITIS

In the case of NSAIDs, it seems clear that inhibition of the cyclooxygenase pathway is not involved. It has been reported that patients can tolerate other NSAIDs both before and after the meningitis episode and that not all the drugs in this group lead to meningitis. Cerebrospinal fluid penetration does not seem relevant since it is similar for all the NSAIDs. Some data point to a hypersensitivity reaction: the temporal relationship between drug intake and the development of meningitis; prior exposure to the offensive drug and disappearance of symptoms after drug discontinuation; the presence of accompanying “allergic” signs, such as facial edema and conjunctivitis (NSAIDs 25%, antibiotics 21%) and rash (NSAIDs 22%, antibiotics 10%); and more severe symptoms on drug re-exposure. Latency after drug intake, however, was not shorter with re-exposure. In those patients who developed meningitis on their first exposure to the NSAIDs, it has been suggested that a prior contact with a chemical cross-reactive with the offending drug could have mediated the sensitization. A few patients have developed meningitis after the intake of several unrelated drugs, which could suggest an individual predisposition. Although CSF data that confirm the meningitis are lacking or incomplete.

In SLE-susceptible mice (NZB and NZW), meningitis developed and was more intense in older animals that had been exposed to ibuprofen longer. The mice developed meningitis (100% of the animals), only if exposed to ibuprofen and not to ketoprofen (another propionic acid derivative), suggesting that only certain drugs lead to meningitis. Furthermore, a specific cell-mediated immunity to ibuprofen has been described in patients with SLE who had not been previously exposed to this drug, perhaps due to cross-reactivity with some natural constituents to which the autoimmune reaction is directed. Also, presumably a lack of suppressor cells as noted in SLE, could allow for a greater magnitude of responsiveness.

Some authors have proposed a role for immune complexes, but controversial findings have been reported. In this context, cytokine levels were found to be normal in another study and high in another. Based on these findings, some authors have concluded that ibuprofen-induced meningitis is an antigen-specific humoral immune process confined to the central nervous system, where the drug, and not a metabolite, would potentiate the activity of a preexisting autoantibody, resulting in complement fixation and development of an acute meningitis.

Bernstein has proposed that the drug combines with a CSF or meningeal protein that acts as a hapten, leading to an inflammatory response in the meninges. However, ibuprofen does not reach high concentrations in the CSF, even with high serum concentrations, which indicates that DIAM is not due to accumulation of antigenic determinants of the drug in the CSF.

Two authors reported eosinophilic pleocytosis in the CSF of patients with ibuprofen-induced meningitis without concomitant eosinophilia, which suggests a process confined to the meningeal compartment.

Taken together, the available data suggest that NSAID-related meningitis develops in individuals rendered susceptible by an underlying autoimmune disorder who were previously sensitized or had a natural immunity to the drug. Why the reaction is confined to the meninges is obscure but might involve cross-reactive mechanisms with antigenic determinants of the central nervous system.

ANTIBIOTIC-INDUCED MENINGITIS

The mechanism of action of antibiotic-induced meningitis is also supposed to be due to a hypersensitiv-
IMMUNOGLOBULIN-INDUCED MENINGITIS

The possibility that this type of DIAM is caused by sensitivity to the stabilizing agents of the commercial preparations, such as polyethylene glycol, maltose, sucrose, or glycyne, seems unlikely since this syndrome developed in the same patients who received other prior immunoglobulin preparations, and there are at least 6 different commercial products that induced aseptic meningitis.

Considering the presence of eosinophils in the CSF of some patients, Sekul et al have suggested a hypersensitivity reaction caused by the direct entry of the immunoglobulins into the cerebrospinal compartment. They also propose an alternative mechanism involving IgG. It was increased in all the patients in whom it was measured, although IgG indexes did not reveal intrathecal synthesis. Shorr and Kester also found increased levels of CSF IgG (1130 g/L, 28% of the total CSF protein). Serum immunoglobulins, especially IgG, can enter the CSF mainly if there is a blood-brain (or blood-nerve) barrier breakdown. Since the infused IgG, derived from a pool of more than 5000 donors, is allogenic, it could interact with antigenic determinants on the endothelial cells of the meningeal vasculature, resulting in a cytokerine-mediated inflammatory reaction.

OKT3 ANTIBODY-INDUCED MENINGITIS

Most authors believe that T cells are opsonized by OKT3 in the presence of complement and are phagocytized by the reticuloendothelial system, resulting in the release of circulating mediators, which induce inflammation in the meninges and fluid leakage leading to brain swelling. Shorr and Kester also found increased levels of CSF IgG (1130 g/L, 28% of the total CSF protein). Serum immunoglobulins, especially IgG, can enter the CSF mainly if there is a blood-brain (or blood-nerve) barrier breakdown. Since the infused IgG, derived from a pool of more than 5000 donors, is allogenic, it could interact with antigenic determinants on the endothelial cells of the meningeal vasculature, resulting in a cytokerine-mediated inflammatory reaction.

OTHER AGENTS ASSOCIATED WITH DIAM

Four patients have been described who developed meningitis after carbamazepine therapy, one of them with Sjögren syndrome and trigeminal neuralgia, 2 with manic depressive syndrome and another with isolated trigeminal neuralgia. The clinical case was indistinguishable from other DIAM, although 1 patient had myoclonus with normal carbamazepine levels. There was peripheral eosinophilia (30% of total white blood cell count) in 2 of them and 2% eosinophils in the CSF in 1. As noted by Quinn and colleagues, counting CSF eosinophils is challenging because of technical difficulties, inexperience of the examiner, and the fragility of this cell. It is thus likely that the presence of eosinophils in the CSF is underreported.

Merrin and Williams reported a case of aseptic meningitis in a 37-year-old woman with Sjögren syndrome and polyarthritis, 3 weeks after starting sulfasalazine and 8 hours after subsequent rechallenge with 0.5 g of the drug. Another case of aseptic meningitis induced by sulfasalazine has been reported in a 41-year-old man with rheumatoid arthritis.

We found 3 cases of isolated DIAM associated with phenazopyridine hydrochloride, zimeldine hydrochloride (a serotonin uptake inhibitor unavailable since 1983), and cytarabine hydrochloride. In the latter, meningitis was associated with cerebellar dysfunction.

Sergent and colleagues described 2 patients with SLE who developed a meningeal reaction after azathioprine therapy, but both patients had clinical and laboratory evidence of active SLE and they also had meningitis not associated with the administration of the offending drug.

Also, single case reports of DIAM in relation with salicylate overdose, hepatitis B vaccination, and ranitidine intake have been published.

In conclusion, the possibility of DIAM should be considered in every patient with neutrophilic meningitis and negative CSF culture, especially in the presence of an underlying autoimmune disorder. Most commonly, NSAIDs, antibiotics, IVIGs, and OKT3 monoclonal antibodies are the causative drugs, acting through different and par-

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