isms remained bilaterally, however. An attempt was made to remove the remaining organisms with forceps, but their tenacious adherence to the conjunctivae and the patient's general discomfort made their removal impossible. Instead, Neosporin* ointment was generously applied to the conjunctival surfaces of both eyes in an effort to suffocate the remaining organisms. The eyes were firmly patched. When the patient returned for follow-up the next morning, the remaining organisms were dead and were readily removed. The patient was treated with topical antibiotic ointment for several more days, and the follicular conjunctival reaction subsided over the ensuing week. A careful fundus examination several days later showed no evidence of intraocular involvement.

Comment

The larvae of *Oestrus ovis* are approximately 1 mm in length. They have two large distinctive buccal hooks and numerous hooklets along the abdominal segments. The botfly larvae are obligate parasites of sheep, goats and occasionally horses. In late summer and fall, gravid female flies deposit as many as 50 larvae in the nares of the above species, either directly or by ejecting a milky stream containing the larvae while in free flight. The larvae mature in the nares or paranasal sinuses. They are then sneezed by the animal onto the soil, where further maturation occurs.[4]

*Each gram contains polymyxin B, 5,000 units; zinc bacitracin, 400 units, and neomycin sulfate, 5 mg, equivalent to neomycin, 3.5 mg (Burroughs Wellcome).

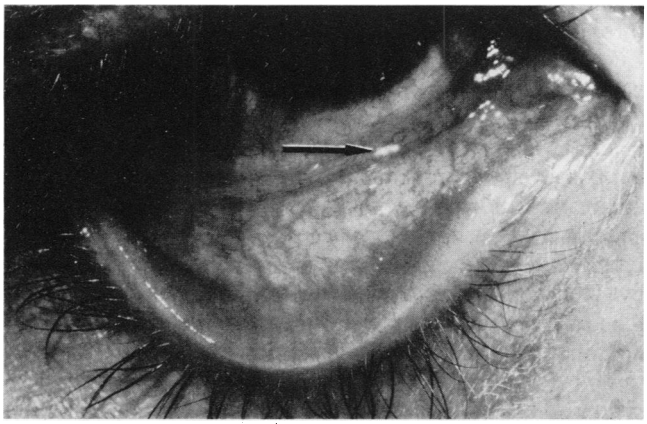

Figure 1.—Mild follicular conjunctivitis, with *Oestrus ovis* larvae in the inferior fornix **(arrow)**.

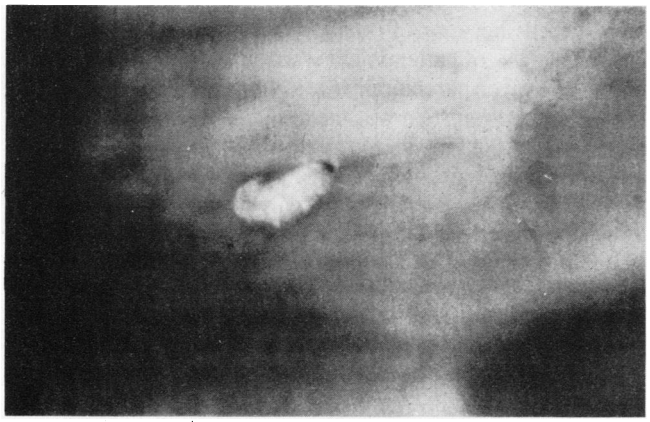

Figure 2.—A slit-lamp photograph of an *Oestrus ovis* larva on the conjunctiva.

In the event that a human is accidentally infected, it is usually via the free-flight spray method. But infection via contaminated water has been reported.[2] When the larvae are deposited on human conjunctivae, they will not mature and can survive for only about ten days, unless the globe is penetrated.

Ophthalmomyiasis externa is primarily a self-limited disease. It has been postulated, however, that these organisms have the ability to penetrate the globe and cause the more serious ophthalmomyiasis interna.[2] Additionally, the irritation caused by external organisms can be severe, as in this case. Thus, prompt diagnosis and treatment are imperative.

Several methods of treatment have been recommended. First, topical anesthetic and anticholinesterase solutions should be administered to immobilize the organisms. Irrigation may be used to eliminate some of the larvae. It has been our experience, however, that even the immobilized organisms tend to cling tenaciously to the conjunctivae. In such cases, the organisms must be removed individually with forceps, which may be difficult in an emergency room. If the individual larvae cannot be removed immediately, we recommend instilling an antiobiotic or anticholinesterase ointment in the affected eyes and patching overnight. This will either kill or immobilize the remaining organisms, allowing easier removal later. When infestations are handled expeditiously in this manner, full recovery is likely.

REFERENCES

1. Healey MC, Collins RK, Hawkins JA: Ophthalmooestriasis externa. South Med J 1980 Oct; 73:1387-1389
2. Hennessy DJ, Sherrill JW, Binder PS: External ophthalmomyiasis caused by *Estrus ovis*. Am J Ophthalmol 1977 Dec; 84:802-805
3. Eifrig DE: Ocular myiasis. Arch Ophthalmol 1969 Jul; 82:137
4. Reingold WJ, Robin JB, Leipa E, et al: *Oestrus ovis* ophthalmomyiasis externa. Am J Ophthalmol 1984 Jan; 97:7-10
5. Brown HS Jr, Hitchcock JC Jr, Foos RY: Larval conjunctivitis in California caused by *Oestrus ovis*. Calif Med 1969 Oct; 111:272-274
6. Gass JDM, Lewis RA: Subretinal tracts in ophthalmomyiasis. Trans Am Acad Ophthalmol Otolaryngol 1976 May-Jun; 81:483-490
7. Vine AK, Schatz H: Bilateral posterior internal ophthalmomyiasis. Ann Ophthalmol 1981 Sep; 13:1041-1043

Hemolysis, Elevated Liver Enzymes and Low Platelet Count

The HELLP Syndrome

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PREECLAMPSIA IS A DISEASE that occurs predominantly in primigravid women and is characterized by the development of hypertension, proteinuria and extracellular fluid retention. Although preeclampsia is a multisystem disease without a single cause, it seems certain that two pathologic processes underlie the clinical course: generalized small artery spasm and increased capillary permeability. A unique group of pa-

(Bertakis KD, Hufford DB: Hemolysis, elevated liver enzymes and low platelet count—The HELLP syndrome. West J Med 1986 Jan; 144:81-83)

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Submitted, revised, February 13, 1985.

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ABBREVIATIONS USED IN TEXT

AST = aspartate aminotransferase
HELLP = hemolysis, elevated liver enzymes and low platelet count
LDH = lactic dehydrogenase

tients with preeclampsia and eclampsia who have hemolysis (H), elevated liver enzymes (EL) and low platelet count (LP) has been reported. Weinstein[1] recently termed this set of symptoms the HELLP syndrome. Since then, there has been debate as to the appropriate use of this acronym. The following is a case report of a patient showing the classic HELLP syndrome. It serves as evidence that this syndrome is, in fact, a clinical disorder that requires early recognition and therapy.

Report of a Case

The patient, a 23-year-old gravida 5 para 0 woman, first came to the Family Practice Clinic, University of California Davis Medical Center, at about eight weeks' gestation. Her pregnancy progressed without event until 36 weeks, when she had edema of her hands and legs, a 1.8-kg (4-lb) weight gain in one week, 1+ proteinuria and a mild elevation in blood pressure from a baseline of 110/70 to 124/80 mm of mercury. She also had had a severe bilateral, frontal headache and frequent Braxton Hicks contractions for several days previously. Laboratory tests done at that time gave the following values: a hematocrit of 41.8%; 179,000 platelets per μl; a serum aspartate aminotransferase (AST; formerly serum glutamic-oxaloacetic transaminase) level, 28 units per liter, and uric acid, 5.1 mg per dl. After a week of bed rest, the patient's edema decreased, her urine had only a slight excess of protein, her blood pressure returned to baseline and results of laboratory tests were normal. A breast stimulation stress test done at 40 weeks showed fetal well-being.

At 40.5 weeks' gestation, the patient began having regular uterine contractions every 12 to 15 minutes. Over the next 24 hours her contractions continued to be infrequent, but she complained of pain localized to her right upper quadrant. Examination at that time confirmed right upper quadrant tenderness, 2+ edema of the lower extremities and 3+ deep tendon reflexes, without clonus. Her blood pressure was 130/108 mm of mercury. Her cervix had dilated to 2 cm with 80% effacement. The hematocrit was 37.5%; platelet count, 61,000 per μl; AST, 257 units per liter; fibrinogen, 491 mg per dl; fibrin monomers, weakly positive; fibrinogen split products, positive at 10 to 40 μg per ml, and prothrombin time and partial thromboplastin time within the normal range. A diagnosis of preeclampsia and the HELLP syndrome was made and an intravenous infusion of magnesium sulfate and oxytocin was started. A cesarean section was eventually done because of failure of labor to progress and worsening right upper quadrant pain. The patient was delivered of a 2,980-gram male infant, Apgar scores 7 and 9, who did very well.

Immediately postpartum, the patient had gross hematuria and prolonged seepage of serosanguineous fluid from the incisional site. Her condition continued to deteriorate, with the following laboratory results being elicited 24 hours later: hematocrit, 22%; platelet count, 17,000 per μl; a peripheral blood smear showing schistocytosis and polychromasia; AST, 756 units per liter; lactic dehydrogenase (LDH), 2,844 units per liter; fibrinogen, 368 mg per dl; fibrin monomers, strongly positive; fibrinogen split products, positive at greater than 80 μg per ml; prothrombin and partial thromboplastin times, normal range. She was given a transfusion of two units of packed erythrocytes at that time. Diuresis started and her elevated liver function tests, anemia and thrombocytopenia began to correct. The magnesium sulfate administration was discontinued 48 hours after the cesarean section.

Her hospital course was complicated by endometritis, which responded to intravenous therapy with gentamicin, ampicillin and clindamycin. The patient was discharged home on the eighth postpartum day with a normal blood pressure and platelet count and improving liver function test values and hematocrit. She has been followed routinely in the Family Practice Clinic without problems except an episode of right upper quadrant pain at three months' postpartum. This was evaluated with an abdominal ultrasonogram of the liver, gallbladder and pancreas in addition to liver function tests, a complete blood count and platelet count. The results of all tests were normal and the pain resolved without further problem.

Discussion

This patient exhibited findings pathognomonic for the HELLP syndrome. The schistocytes and polychromasia seen on peripheral blood smears and the falling hematocrit were compatible with microangiopathic hemolytic anemia. Prothrombin and partial thromboplastin times were normal. Liver function test results, exemplified by AST and LDH values, were greatly elevated. There was a significant thrombocytopenia present. Clinical symptoms included right upper quadrant tenderness and edema. Hypertension was present but not severe. Postoperatively, generalized oozing from the cesarean section incision site was noted and endometritis complicated her recovery. This directly parallels the results reported in the 29 patients with the HELLP syndrome studied by Weinstein.[1] He found abnormalities on the peripheral blood smears, including burr cells or schistocytes, or both, in addition to polychromasia in 28 out of 29 patients. Each had thrombocytopenia and 72% had a fall in hematocrit out of proportion to the estimated blood loss. All 29 of his patients had elevated AST values and all had right upper quadrant tenderness to palpation. The overall cesarean section rate was 76% and often the wound was left open from the fascia (and later closed) because of oozing. Complications of infection ensued in 31% of patients, but were successfully treated with antibiotics given parenterally.

Although Weinstein was the first to use the term "HELLP syndrome" to characterize the thrombocytopenia, microangiopathic hemolytic anemia and abnormal liver function findings seen in some patients with eclampsia or preeclampsia, these complications have been noted separately in the past. Platelet depletion is a significant early feature in preeclampsia and can lead to thrombocytopenia. This drop in platelet count does not occur in uncomplicated pregnancies. Microangiopathic hemolytic anemia has been reported in some patients with preeclampsia, but the frequency is unknown. Finally, abnormalities in liver function and on microscopic examination of the blood are commonly found in patients with preeclampsia. The demonstration, however, of

all three complications together in preeclampsia and eclampsia provides evidence for the existence of the HELLP syndrome. In 1954, Pritchard and associates reported three cases of eclampsia in women with hemoglobinemia, hemoglobinuria, thrombocytopenia and clotting defects. Two of the women died. In both of those cases, severe epigastric pain and generalized abdominal discomfort were noted on admission to hospital. At autopsy, microscopic examination of the liver showed focal periportal necrosis in one case and diffuse degenerative changes within the parenchymal cells, with subacute inflammation of portal areas in the other.[2]

Killam and colleagues published a report of five cases of HELLP syndrome associated with preeclampsia that were very similar to the one reported here. Three of their patients showed substantially abnormal hematologic and liver function test values but did not have severe hypertension. Initially these patients were mistakenly diagnosed as having hepatitis, hiatal hernia and pyelonephritis, respectively. Despite the difference in presentation, all five patients had evidence of liver dysfunction, depressed platelet counts and intravascular coagulation and hemolysis. The authors concluded that the disorder must be strongly considered in all patients with severe preeclampsia or eclampsia with epigastric pain. They also advised that the poor fetal and maternal prognoses necessitate expeditious delivery.[3]

In conclusion, our report lends support to the existence of a subset of women with preeclampsia and eclampsia who manifest hemolysis, elevated liver enzyme levels and low platelet counts. It is therefore important that physicians caring for obstetric patients be cognizant of the clinical and laboratory findings in the HELLP syndrome in order to give early supportive therapy and delivery.

REFERENCES

1. Weinstein L: Syndrome of hemolysis, elevated liver enzymes, and low platelet count: A severe consequence of hypertension in pregnancy. Am J Obstet Gynecol 1982 Jan 15; 142:159-167

2. Pritchard JA, Weisman R, Ratnoff OD, et al: Intravascular hemolysis, thrombocytopenia and other hematologic abnormalities associated with severe toxemia of pregnancy. N Engl J Med 1954 Jan 21; 250:89-98

3. Killam AP, Dillard SH, Patton RC, et al: Pregnancy-induced hypertension complicated by acute liver disease and disseminated intravascular coagulation. Am J Obstet Gynecol 1975 Dec 15; 123:823-828

Endoscopic Observations of the Pharyngeal Airway During Treatment of Obstructive Sleep Apnea With Nasal Continuous Positive Airway Pressure—A Pneumatic Splint

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SINCE THE FIRST DESCRIPTION OF SLEEP APNEA in Pickwickian patients in 1965,[1] the sleep apnea syndrome has been recognized with increasing frequency in a variety of clinical situations.[2] The cardiopulmonary complications associated with this disorder often lead to consideration of a tracheostomy. The psychological and physical trauma experienced by patients with tracheostomies,[3,4] however, has prompted the development of alternative modes of therapy.

The pathophysiology of the obstructive variety of sleep apnea has been proposed to be a combination of the negative oropharyngeal pressure generated during inspiration and reduced neurogenic tone in the upper airway muscles associated with sleep, especially the rapid-eye-movement period of sleep. The decreased neural activity leads to a relaxation of the upper respiratory muscles, which causes posterior displacement of the tongue and inward movement of the pharyngeal walls. This muscular relaxation, especially if combined with an upper airway anatomic abnormality, such as fatty infiltration,[5] macroglossia[6] or nasal polyps,[7] is often sufficient to cause a physical obstruction to airflow.

ABBREVIATIONS USED IN TEXT
CPAP = continuous positive airway pressure
EMG = electromyography

With this pathophysiology in mind, it can be appreciated that continuous positive airway pressure (CPAP) applied at the nares, as introduced by Sullivan,[8,9] may be useful in the treatment of obstructive sleep apnea. The mechanism of action of CPAP has been eloquently likened to that of a "pneumatic splint" of the upper airway. We offer our observations in one patient on the effects of nasal CPAP on the patency of the nasopharynx and hypopharynx that confirm this concept.

Report of a Case

The patient, a 60-year-old man, while in hospital for the evaluation of new onset of seizures, was referred for evaluation of apneic episodes observed during sleep. He admitted to snoring for many years, and his wife described nocturnal apneic episodes, often of long duration, for many years. The patient complained of frequent sleep interruptions for the past six years, associated with paroxysmal nocturnal dyspnea. These were often so severe as to require him to sleep sitting up in a chair.

The patient was formerly employed as a bus driver, but he often experienced daytime hypersomnolence of such severity that he would fall asleep while parking his bus. He would also fall asleep at the dinner table or during other activities that did not require his attention.

He said he did not have insomnia, nightmares, enuresis, morning headache, depression or sexual dysfunction. There was no history of cardiopulmonary or nasopharyngeal disease. The patient had had hypertension for 18 years and intermittent pedal edema for 6 years and took a thiazide diuretic

(Popper RA, Leidinger MJ, Williams AJ: Endoscopic observations of the pharyngeal airway during treatment of obstructive sleep apnea with nasal continuous positive airway pressure—A pneumatic splint. West J Med 1986 Jan; 144:83-85)

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Submitted, revised, February 25, 1985.

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