Article abstract—Pseudotumor cerebri (PTC) is most commonly seen in obese women of reproductive age. We studied 109 women with PTC between ages 16 and 44 years. In 11, PTC started during pregnancy. Thirteen women with previous diagnosis of PTC, including two of the aforementioned 11, had an additional 17 documented pregnancies. Patients were matched by age and parity with controls. Obstetric complications occurred more frequently in the controls. Visual loss occurred with the same frequency in pregnant and nonpregnant patients. Treatment of PTC patients in pregnancy should be the same as for nonpregnant PTC patients, except that calorie restriction and diuretic use are contraindicated. Obstetric management is no different from that of normal pregnancy.

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Pseudotumor cerebri and pregnancy

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Pseudotumor cerebri (PTC) is defined by papilledema, headache, and increased intracranial pressure without focal neurologic abnormality. It once accounted for 10%[1] of patients with papilledema and no lateralizing neurologic signs, but that figure is now 70%.[2] Because the etiology is unknown, the reported associations of PTC with other illnesses vary.[3-5] It is universally accepted that PTC is much more prevalent in women (2:1 to 8:1),[6-14] especially in young obese women of childbearing age[15] with alleged menstrual irregularity.[16] One might expect PTC to be common among pregnant women. However, there has been only one study of PTC in pregnancy.[17] There have been single case reports,[18-24] and review articles have mentioned pregnancy as an etiologic factor.[7-10,14,25-30] There have been no published studies of PTC in pregnancy with age- and parity-matched controls, as we have done.

Patients and methods. *Identification of study patients.* The charts of all women of reproductive age who were inpatients at University of Iowa Hospitals, admitted between 1966 and 1982 with the diagnosis of "pseudotumor cerebri" or "benign intracranial hypertension," were identified by a computer-assisted search of hospital discharge diagnoses. Because this search did not include outpatients in any year or inpatients admitted before 1966, additional cases dating from 1939 were identified from the files of one of the authors (JJC). Diagnostic criteria included (1) papilledema, (2) increased CSF pressure, (3) absence of any other cerebral disease, and (4) normal CSF content except for low protein levels. The searches yielded 109 patients who met these criteria.

Each record was reviewed for history of associated pregnancy. Twenty-one of the 109 patients were found to have had at least one pregnancy temporally associated with PTC.

All charts were reviewed for age, parity, menstrual history, evidence of endocrine disease, pregnancies, and pregnancy complications. Pregnancies during the PTC episode were studied for last menstrual period (LMP), week of gestation when the patient was first seen, duration of symptoms, complications of pregnancy, and outcome of pregnancy; initial and maximum weights in pregnancy were noted. Obesity was assumed when body weight exceeded maximum weight-for-height according to the 1959 Metropolitan Life Insurance tables.[31] Fertility problems, other medical problems, and drug or medication use were noted.

We directed attention to (1) time of symptomatic onset of PTC; (2) results of initial treatment, usually 1 to 2 months after onset; (3) condition of PTC after pregnancy ended; and (4) condition at last visit.

We sought evidence of papilledema, visual acuity, visual fields (VF), other neurologic signs (eg, abducens nerve pareses), blood pressure, CT, skull roentgenogram, angiography, EEG, pneumoencephalogram (PEG), CSF analysis, and routine blood studies. We recorded the type of treatment and its presumed effects.

We defined resolution of PTC as occurring when

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Table 1. Diagnosis of pseudotumor established during pregnancy

Pt	Age at Dx	Wk of gest Dxed	Duration of Sx	Obesity	HTN	Tx	PGY Outcome	Pseudotumor outcome	Visual outcome	Subsq* PGY	Was there later recurr?
#1 - VB G2 P1	18	5	2 wks	+	No	Chlorthalidone, prednisone after PGY terminated	Elective Ab 8 wks	Slow resolution 6 mos after Ab	Normal	0	No
#2 - BCa G1 P0	20	29	4 wks	+	Yes	R decompressive craniotomy	Normal	Resolved within 1 mo of Tx	Mod visual loss	0	No
#3 - DD G3 P2	23	1	6 wks	+	No	Acetazolamide stopped after PGY discovered; Wt reduction	Normal	Resolved by 4 mos after Tx	Normal	0	No
#4 - OH G7 P3 Ab1	25	12	8 wks	+	No	Aldactone; hydrochloro-thiazide; urea (po) LP × 2	Normal	Resolved by 5 mos after Tx	Normal	3	No
#5 - MH G4 P3	26	4	4 wks	+	No	Salt restriction, dexamethasone, glycerol	Normal	Resolved by 1-2 mos	Normal	0	Yes see pt # 3 table 2
#6 - SM G2 P0 Ab1	32	14	8-10 wks	+	No	Optic nerve sheath decomp unilateral	Normal	Resolved within 9 mos after Tx	Mod visual loss	0	No
#7 - JM G1 P0	18	20	10 wks	+	No	LP × 1; Wt reduction	Normal	Resolved 1 mo postdelivery	Normal	0	No
#8 - CN G2 P1	23	16	4 wks	+	Yes	Dexamethasone; Wt loss	Normal	Resolved 3-4 mos after Tx	Normal	0	No
#9 - DR G3 P2	24	16	8 wks	+	No	Salt restriction; diet; LP × 2	Unknown	Resolved unknown time, follow-up normal	Normal	0	No
#10 - DL G1 P0	20	24	8 wks	+	No	Prednisone; acetazolamide	Normal	Resolved 3 mos after delivery	Mod visual loss	0	Yes see pt #4 table 2
#11 - DS G2 P1	24	prior	prior	+	No	Acetazolamide; chlorthalidone; furosemide, diet	SAB 12 wks	Resolved 1 yr after Ab	Normal	0	No

* Subsequent pregnancy with inadequate documentation.

Dx Diagnosis. PGY Pregnancy. Mod Moderate.
Gest Gestation. Subsq Subsequent. Wt Weight.
Sx Symptoms. Recurr Recurrence. LP Lumbar puncture.
HTN Hypertension. Ab Abortion. decomp Decompression
Tx Treatment. R Right. SAB Spontaneous abortion.

the patient had no symptoms, no evidence of papilledema, and normal neurologic examination. Visual status was recorded as outlined by Corbett.[32] Finally, pregnancies that followed the diagnosis of PTC were reviewed for recurrence of PTC. In some cases, later pregnancies were noted, but with little other information. Patients were divided into three groups: (1) PTC onset during pregnancy (group 1); (2) PTC onset earlier, but recurrence during pregnancy (group 2); and (3) PTC onset earlier without recurrence in pregnancy (group 3).

Identification of controls. The University of Iowa Labor and Delivery Area records were reviewed for women delivering on or after the same date as each of the subjects. These were matched for age ± 1 year, gravidity, and parity. In the cases of spontaneous abortion, the Department of Obstetrics and Gynecology's operative records were reviewed, and controls were matched by age and parity.

Each of the control records was studied for all of the obstetric information outlined above.

Review of literature. A review of the literature on PTC and pregnancy was undertaken by searching the *Index Medicus* for pseudotumor cerebri. All articles were perused for reports of pregnant patients, and only those cases with well-documented PTC were included. We included only those whose PTC was first diagnosed while the patient was pregnant.

Table 2. Diagnosis of pseudotumor established earlier with recurrence during pregnancy

Pt	Age at Dx	Age at PGY	Wk of gest	Obesity	HTN	Tx	PGY outcome	Pseudotumor outcome	Visual outcome	Subsq* PGY	Was there later recurr?
#1 - CB G2 P1	29	32	32	+	Yes	Previously placed lumboperitoneal shunt replaced	Normal	Resolved 2 mos after delivery	Mod visual loss	0	No
#2 - SG G1 P0	18	19	8	+	No	Dexamethasone	SAB 24 wks	Resolved with Ab	Mod visual loss	0	No
#3 - MH G5 P4	26	28	24	+	No	Dexamethasone, glycerol	Unknown	Unknown	Normal	0	No
#4 - DL G2 P1	20	20	10	+	No	Unknown	Unknown	Unknown	Unknown	0	Unknown
#5 - BB G2 P1	28	31	28	+	No	Wt reduction	Normal	Resolved by 1 yr	Normal	0	No

* Subsequent pregnancy with inadequate documentation.
Abbreviations as in table 1.

Reported cases of PTC diagnosed during pregnancy appear in table 6. Two cases[33,34] were not included because of uncertain diagnosis of PTC. In a Russian series[35] of PTC in pregnancy, 20 of 22 women had focal signs including motor disturbance in 14, cranial nerve lesions (type not specified) in 12, sensory disturbance in 6, and aphasia in 4. These cases were not included. All cases were reviewed for age, previous pregnancies, week of gestation at onset (if known), obesity, hypertension, treatment if specified, pregnancy outcome, and PTC outcome (ie, when resolution of symptoms and signs occurred). Recurrence of PTC with subsequent pregnancy was noted.

Statistical methods. Comparisons of groups for statistical difference was made by using the chi-square test.

Results. Of the 109 women patients with PTC diagnosed between 1939 and 1982, 21 women had 28 adequately documented pregnancies. Three women had eight additional pregnancies for which sufficient obstetric data was unavailable. Eleven patients were pregnant when symptoms of PTC began; 5 pregnancies occurred after the onset of PTC but with recurrent PTC; and 12 pregnancies followed the onset of PTC but without recurrence of PTC.

Group 1. Onset during pregnancy. Among patients with onset of PTC during pregnancy (group 1, table 1), the mean age was 23.0 years. The mean duration of gestation was 14.1 weeks when PTC was diagnosed. The average duration of symptomatic PTC was 6.2 months. Three of 11 patients had moderate visual loss. Nine of 11 (81.8%) had onset in the first trimester (0 to 13 weeks), 2 of 11 (18.2%) in the second trimester (14 to 26 weeks), and none during the third trimester (26 to 40 weeks). In two patients (DS and DD), PTC symptoms began before conception. CSF pressure measured between 240 and 550 mm H_2O, with a mean pressure of 360 mm H_2O. CSF protein ranged from 20 to 47 mg/dl, with a mean of 27 mg/dl.

Group 2. Reactivation during pregnancy. Five patients became pregnant after the onset of PTC and had recurrent PTC in pregnancy (group 2, table 2). The mean age of these patients was 24 years. The mean week of gestation in which PTC reappeared was 19.8 weeks. The mean number of years between diagnosis and pregnancy was 1.8 years. The average duration of PTC was 8 months after onset during pregnancy.

Group 3. Remission during pregnancy. In 12 patients, pregnancy occurred after onset of PTC that was in remission throughout the pregnancy (group 3, table 3). The average age at time of pregnancy was 22.3 years. One patient (CH) had two normal deliveries before the diagnosis of PTC. She became legally blind within 1 year of onset of PTC, had a lumboperitoneal shunt placed, and had a normal pregnancy 3 years later. The average duration between diagnosis of PTC and pregnancy was 2.7 years.

When groups 1 and 2 were reviewed (ie, those with active PTC during pregnancy), 10 of 16 patients had used oral contraceptives. However, when PTC patients were compared with matched controls, there was no statistically significant difference between the two groups (tables 4 and 5). One of the 16 was sterilized after pregnancy because she wanted no further pregnancies. There were no associated signs of hormonal imbalance (galactorrhea or hirsutism) in any of the patients. There was a history of menstrual irregularity in 3 of 16 cases, but this was not significantly different from that of controls. When all three groups of patients were reviewed, only one patient had a history of vitamin A use. Three

Table 3. Diagnosis of pseudotumor established earlier without recurrence during pregnancy

Pt	Age at Dx	Age at PGY	Obesity	HTN	Tx	PGY outcome	Pseudotumor outcome	Visual outcome	Subsq* PGY	Was there later recurr?
#1 - BC G2 P1	18	20	+	No		Normal	Recurr 5 mos after delivery	Normal	0	Yes
#2 - CH G3 P2	24	27	+	Yes	S/P lumbo-peritoneal shunt	Normal		Blind OU before PGY	0	No
#3 - RL G2 P1	20	22	+	No		Elective Ab 12 wks		Normal	0	No
#4 - LP G1 P0	21	28	+	Mild yes		Normal		Normal	0	No
#5 - PL G1 P0	20	22	+	No		Elective Ab 10 weeks		Normal	4	No
#6 - MA G1 P0	18	19	+	No	S/P temp decomp	SAB 10 wks		Mod visual Loss	0	No
#7 - ML G1 P0	22	26	+	No		Normal		Normal	1	No
#8 - RL G3 P1 Ab1	20	23	+	No		SAB ? wks		Normal	0	No
#9 - VB G3 P1 Ab1	18	19	+	No		? normal		Normal	0	No
#10 - VB G4 P1 Ab1	18	21	+	No		Normal		Normal	0	No
#11 - SG G2 P0 Ab1	18	19	+	No		Normal		Normal	0	No
#12 - SG G3 P1 Ab1	18	21	+	No		Normal		Normal	0	No

* Subsequent pregnancy with inadequate documentation.
S/P Status post.
Other abbreviations as in table 1.

patients had used tetracycline at least once in the past. None had systemic lupus erythematosus. One patient had a history of thyroiditis (BC).

We compared PTC study patients with age- and parity-matched controls for factors alleged to be associated with PTC (tables 4 and 5). All women in groups 1 and 2 were obese.

Discussion. Most authors believe that PTC is provoked or exacerbated by pregnancy,[5,7-12,14,18,25-30] but in one case PTC improved during pregnancy.[13] PTC was once "successfully treated" with extracts made from urine of pregnant women.[36] Sometimes PTC began or was discovered immediately after delivery.[8,25,37]

Quincke is said to have been the first to relate menstrual irregularity or pregnancy to PTC.[3,9,38] It was not until Foley[9] reported that 7 of 44 (15.9%) patients were pregnant at the time of PTC that the association received serious attention. In subsequent series, the rate of pregnancy in PTC patients ranged from 2[2,13] to 12%[7], with a mean of 5%. In our series we have found that 8.3% were pregnant at onset of PTC, and 14.7% had PTC with pregnancy. According to the US Bureau of Statistics, after excluding illegal abortions and miscarriages in women age 15 to 44, the total number of viable pregnancies in 1979 was 3,494,000. The total number of legal abortions was 1,409,600. The total population of women aged 15 to 44 was 41,079,000.[39] Therefore, the chance that any one woman in that age group would be pregnant in that year is at least 11.9%. The association of pregnancies with PTC is probably only a measure of the fact that PTC affects women of childbearing years.

Onset of PTC in pregnancy. Greer[17] and Foley[9] suggested that PTC tends to occur in the first trimester of pregnancy, but Donaldson[40] reported onset

Table 4. Onset of pseudotumor during pregnancy

	Study (N = 11)	Control (N = 11)	*p* value
Obesity	11	1	−0.0005
HTN	2	0	NS
PGY outcome	8 - Normal 1 - Elective Ab 1 - Unknown 1 - SAB	9 - Normal 1 - SB 1 - SAB	
History of oral contraceptive use	6	5	NS
Obstetric complications	1 - C-section 1 - Poor wt gain	1 - Twin PGY 1 - Premature labor 1 - Hepatitis 1 - Postpartum hemorrhage 1 - Syncope	0.0057
Neonatal complications	0	0	NS
Postpartum sterilization	0	2	NS

SB Stillborn.
Other abbreviations as in table 1.

Table 5. Diagnosis of PTC established earlier with recurrence of active PTC during pregnancy

	Study (N = 5)	Control (N = 5)	*p* value
Obesity	5	0	0.005
HTN	1	0	NS
PGY outcome	2 - Normal 1 - SAB 2 - Unknown	4 - Normal 1 - SAB	
History of oral contraceptive use	4	3	NS
Obstetric complications	1 - Meconium	1 - Cesarean	NS
Neonatal complications	0	0	NS
Postpartum sterilization	1	2	NS

Abbreviations as in table 1.

in the third, fourth, or fifth month. In the literature, the range of gestational age at onset of PTC was from conception to 33 weeks.[9,14,17-24,27-29,41-43] The average duration of gestation of these reported cases at diagnosis was 14.4 weeks; 61.2% occurred in the first trimester, 19.4% in the second trimester, and 19.4% in the third trimester. In our patients, the average gestation was 9.3 weeks in those pregnant at onset of PTC; 81.8% are associated with the first trimester, 18.2% during the second trimester, and none with the third trimester. Symptomatic onset was most often (89%) seen in the first half of pregnancy. However, some patients with PTC are asymptomatic,[13,32] and the diagnosis is not established until papilledema is discovered accidentally.[44] Since ophthalmoscopic examination should be part of the initial evaluation of obstetric patients, and since most pregnant patients register for obstetric care in the first half of pregnancy, the discovery of PTC in the first half of pregnancy could be, at least in part, a selection artifact.

Pregnancy outcome and fetal wastage. It has been suggested that women with PTC have increased rates of spontaneous abortion.[9,45] In general, the rate of spontaneous abortion is 14 to 20%.[46] In previously reported cases of PTC (table 6), the rate during PTC was 15.4%. In our series, there is one spontaneous abortion in the 11 patients with onset of PTC during pregnancy. However, in reviewing all our cases of PTC during pregnancy (groups 1 and 2), the spontaneous abortion rate was 12.5%. These figures were not significantly different from those expected.

Twenty-one of 26, or 81%, of pregnancies (table 6) had normal outcomes. In our series, 83% of pregnancies complicated by active PTC had normal outcomes.

When we compared PTC patients and controls, the controls had more obstetric complications (tables 4 and 5). Although this difference was statistically significant, at least in group 1, it was probably a measure of the referral pattern to the university obstetric service.

Visual outcome. Aside from pregnancy outcome, the other major issue in the management of the pregnant patient with PTC is vision. In PTC without associated pregnancy, 4 of 11 (37%) women reported by Davidoff and Dyke[26] had some loss of vision. Later series[6,7,9,12,25-27] suffered from lack of follow-up or lack of detail of the visual examination. Although most agreed that visual acuity and visual fields may be abnormal at the time of PTC diagnosis, there was no adequate follow-up. Rush[13] reported long-term visual impairment in 11% of his population. Corbett et al[32] found that 14 of 57 patients followed for 5 to 41 years had permanent severe visual loss in one or both eyes; some visual loss affected almost 55%.[32]

Among reported pregnant patients with PTC (table 6), vision was normal in 18 cases and abnormal in two (11.1%). In our series (group 1 and 2), no patient was blind, but of the 16 pregnancies with active PTC, 5 pregnancies in 5 patients (31.3%) were associated with some loss of visual acuity or constriction of visual field. In none did the visual impairment interfere with the patient's life. PTC during pregnancy did not increase the risk for visual loss.

Related factors. Pseudotumor cerebri has been linked to obesity[2,11,13,15,32] in 11.1[7] to 90%.[2,32] All our patients were obese, and there was a significant difference in obesity among patients with PTC and age-parity-matched controls ($p < 0.0005$). PTC is asso-

Table 6. Summary of previously reported cases of pseudotumor cerebri with onset during pregnancy

Author/yr	PGY N	Female pts with PTC	Age (range/mean)	Gravidity (range/mean)	# Ab/Prev PGYs	Wks gest at onset (range/mean)	% Obese	#HTN	Tx
Foley, 1955[9]	5	44	—	—	—	4-20 wks/10 wks	2/5	—	—
Zuidema and Cohen, 1954[46]	1	39	—	—	—	—	—	—	—
Paterson et al, 1961[29]	2	17	21-25/$\bar{x}$=23	1-5/3	0/4	16-20 wks/17 wks	—	0	Steroids
Greer, 1963[15]	8	—	14-32/$\bar{x}$=22	1-4/2.5	2/12	4-16 wks/10.6 wks	2/8	0	None (4) R subtemp decomp (3) LP (1)
Nickerson and Kirk, 1965[11]	2	—	19-22/$\bar{x}$=20.5	3/3	1/4	?-14 wks/12 wks	—	—	Temporal decomp (1) diuretics (1)
Elian et al, 1968[40]	1	—	19/19	1/1	0/0	12 wks	—	—	Elective Ab
Guidetti et al, 1968[10]	4	74	—	—	—	—	—	—	—
Powell, 1972[20]	1	—	24/24	3/3	0/2	4 wks	1	—	Decadron
Benini, 1973[18]	1	—	26/26	2/2	—	5 wks	—	—	Acetazolamide + LP (1)
Meythaler and Meythaler-Radek, 1973[22]	1	—	37/37	1/1	0/0	28 wks	—	—	Delivery at 36 wks
Johnston and Paterson, 1974[11]	3	50	—	—	—	—	—	—	—
Weisberg, 1975[14]	2	100	—	—	—	1st trimester (2/2)	—	—	—
Jefferson and Clark, 1976[25]	2	28	19-38/$\bar{x}$=285	—	—	4-13 wks/8 wks	1/2	—	Decomp (1) diuretics (1)
Traviesa et al, 1976[43]	1	—	22/22	—	—	12 wks	1	0	Acetazolamide (1)
Caroscio and Pellmar, 1978[39]	1	—	20/20	2/2	0/1	28 wks	1	0	Bedrest + LP ×3 (1)
Bulens et al, 1979[5]	3	25	—	—	—	—	—	—	—
Henry and Jacques, 1979[3]	1	—	24/24	6/6	1/5	16 wks	—	0	Steroids (1)
Keltner et al, 1979[28]	1	—	20/20	—	—	10 wks	1	—	Lumboperitoneal shunt (1)
Palop et al, 1979[12]	1	—	20/20	2/2	0/1	28 wks	0	—	LP + diuretics (1)
Gupta et al, 1980[21]	2	—	25-25/$\bar{x}$=25.5	2-3/2.5	—	27-32 wks/29.5 wks	0	0	Cerebral decongestives + acetazolamide (1)
Rush, 1980[13]	1	63	23/23	—	—	—	—	—	—
Shekleton et al, 1980[41]	1	—	30/30	2/2	0/1	33 wks	—	0	Decadron, salt & fluid restriction, decomp, C-section (1)
Digre et al, 1983	11	109	18-32/$\bar{x}$=23	1-7/2.6	2/13	0-25 wks/9.3 wks	11/11	2	Steroids (4), acetazolamide (3), diuretic (3), wt reduction (4), surgery (2)

Prev Previous.
Subtemp Subtemporal.
Other abbreviations as in table 1.

	PGY outcome	PTC outcome	Visual outcome	Recurr	Subsq PGY
	1-SAB 4-Normal	1 pt had resolution 10 d postpartum	—	—	—
	—	—	—	—	—
oids	2-Normal	Resolution in 10-36 d	Normal 2/2	None	—
e (4) ibtemp mp (3) (1)	6-Normal 2 SAB	Resolution within 2 wks	Normal 8/8	None	1
poral mp (1) etics (1)	2-Normal	Resolution within 2 wks	—	2/2	4
tive	1-Elective Ab at 20 wks	Resolution in 2 d	—	1/2	2
	—	—	—	None	—
adron	1-SAB (20 wks)	?	—	Yes—30 wks	1
tazol- ide + (1)	1-Normal	Resolution 3 wks postpartum	Normal	None	—
ivery 36 wks	1-Normal	Resolution 3 wks postpartum	Normal	None	—
	—	—	—	—	—
	—	—	—	—	—
comp (1) retics	—	Resolution at 10 d and 14 wks postpartum	Normal 2/2	—	—
etazol- ide (1)	—	—	Normal	None	—
drest + × 3 (1)	1-Normal	Resolution 4 months postpartum	Normal	None	—
	—	—	—	—	—
eroids)	—	—	—	—	—
mboperi- neal unt (1)	—	—	Abnormal	—	—
P + diu- tics (1)	1-Normal	Resolution	Normal	—	—
erebral de- ngestives acetazol- nide (1)	2-Normal	Resolution at 1 wk & 2 wks postpartum	Normal	1/2	2
	—	—	—	—	—
ecadron, lt & fluid striction, ecomp, C- action (1)	1-Normal	Resolution 6 wks postpartum	Abnormal	—	—
teroids (4), cetazol- mide (3), iuretic (3), t reduction 4), urgery (2)	8-Normal 1-elective AB 1-SAB 1-unknown	Resolution at 1-9 mos postpartum	Normal (8) Abnormal (3)	2	7

ciated with obesity in women, whether or not they are pregnant.

Use of oral contraceptives has been implicated in PTC,[7,18,30,37,40,45,47] but there have been no studies of the use of oral contraceptives in patients with PTC. According to recent (1981) statistics, there are 6,096,030 "pill" users, which would account for at least 15% of the female population between ages 16 and 44.[39] We found no significant difference between oral contraceptive use in patients with PTC and controls. With such pervasive use of oral contraceptives, claims of correlation between PTC and oral contraceptive use must be made cautiously.

Diagnosis of PTC during pregnancy. The clinical diagnosis of PTC requires normal CT, high CSF pressure, normal or low CSF protein, normal glucose, and no CSF cells. No procedure currently required to make the diagnosis of PTC is contraindicated during pregnancy.

Treatment. Treatment of pregnant PTC patients has been as varied as treatment of PTC in the nonpregnant patient. Some cases resolved with no treatment at all.[17] Others were treated by bed rest, lumbar punctures, or weight loss.[18,19] Surgical procedures have included subtemporal decompression,[17,27,41] lumboperitoneal shunts,[28] or optic nerve sheath decompression.[13,48] Termination of pregnancy[20,21] or early delivery[24] has been recommended.

We believe pregnancy should not be aborted solely because of PTC, and we do not agree with those[21,23,24,41] who advocate therapeutic abortion for PTC.

Almost all the treatment regimens used with nonpregnant PTC patients can be used in the pregnant patient.[45] The major exception is caloric restriction, which is proscribed because of the adverse effects of ketosis on the fetus.[46] However, weight gain should be limited to 20 pounds.[46] There is no contraindication to repeated lumbar puncture. Although use of corticosteroids has been associated with birth defects in laboratory animals, this association has not been substantiated in the human.[49] Reports that steroids may cause low birth weight of infants may reflect the effects of the disease in the mother for which the steroids are taken.[50] Although steroids have been reported to even cause PTC,[51-53] these are mostly case reports and are related to chronic use. Acetazolamide may be used after 20 weeks' gestation; earlier use has been associated with a single report of sacrococcygeal teratoma, but acetazolamide is not a confirmed teratogenic agent.[49] Glycerol, an osmotic diuretic, probably should not be used in the second half of pregnancy because of the potential decrease in placental blood flow associated with diminished maternal blood volume. The use of thiazide diuretics or chlorthalidone is controversial for the same reasons. Fetal growth and maternal electrolytes should be followed closely in patients treated with these agents.[49] There is no obstetric contraindication to

any of the possible surgical decompression procedures.

In the literature (table 6), medical treatment was associated with abnormal outcome of pregnancy in 3 of 16 patients, none of whom had visual loss. Surgical treatment (lumboperitoneal shunt, etc.) was associated with normal pregnancy outcome in six, but two of seven had visual loss—probably a measure of the indications for surgery. We found similar results in our patients. Three patients who had surgical procedures had normal pregnancies, but had suffered visual loss before the procedure. Surgical procedures did not adversely affect the outcome of a normal pregnancy in our cases.

Visual acuity and visual fields must be monitored closely. When vision is threatened, some authors suggest that treatment with steroids,[2] optic nerve sheath decompression,[13] or lumboperitoneal shunting[45] can be used without any adverse effect on the pregnancy.

Future pregnancy. Regarding future pregnancies, some[41] have recommended sterilization of women with PTC. Others[21] suggested no further pregnancies until all residual signs and symptoms of PTC abated.

Among reported cases of pregnancy following PTC,[17,20,21,23,41] 6 of 10 pregnancies were associated with recurrent PTC, but were only isolated individual case reports and may have inflated the prominence of recurrence in the PTC literature.

In 14 patients undergoing 17 subsequent pregnancies, we note 5 recurrences (29.4%) of PTC during pregnancy. However, three of these 14 women had an additional eight pregnancies for which adequate information was not available. All eight were delivered in smaller hospitals. If there was no PTC exacerbation in those eight, the recurrence rate was 5 of 25, or 20%.

Recurrence rates of PTC in nonpregnant patients have been recorded between 2[9] and 42.9%[30]; most were between 6 and 10%.[2,4,6,7,29] "Recurrence" of PTC may need to be defined as recurrence of symptoms and signs. CSF pressure may be increased in totally asymptomatic patients.[2,32,44]

These data suggest that future pregnancies in PTC patients need not be prohibited. Symptomatic recurrence of PTC is no more frequent in pregnant women than in other women with the disease who are not pregnant.

Conclusions. (1) Pregnancy occurs in PTC at about the same rate that pregnancy occurs in the general population.

(2) PTC can occur in any trimester, although it usually appears in the first half of pregnancy.

(3) Patients with PTC during pregnancy have the same spontaneous abortion rate as the general population.

(4) Visual outcome for pregnant women with PTC is the same as for women with PTC who are not pregnant.

(5) The pregnant patient with PTC should be treated the same as any other patient with PTC. The pregnancy should be managed with routine obstetric practice. Close communication between obstetrician, ophthalmologist, and neurologist should be encouraged.

(6) Therapeutic abortion to limit progression of disease is not indicated.

(7) Subsequent pregnancy does not increase the risk of recurrence of PTC above the risk of recurrence in any other woman with PTC, and is associated with normal outcome.

References

1. Berg L, Rosomoff HL, Aronson N, Silbermann M, Pool JL. The syndrome of increased intracranial pressure without localizing signs. Arch Neurol Psych 1955;75:498-505.
2. Weisberg LA. The syndrome of increased intracranial pressure without localizing signs: a reappraisal. Neurology (Minneap) 1975;25:85-8.
3. Quincke H. Uber meningitis serosa. Sammlung klinische vortr. Innere Medicin 1893;23:655-94.
4. Greer M. Management of benign intracranial hypertension (pseudotumor cerebri). Clin Neurosurg 1968;15:161-74.
5. Henry S, Jacques S. Benign intracranial hypertension in pregnancy. JACEP 1979;8:323-5.
6. Boddie HG, Banna M, Bradley WG. "Benign" intracranial hypertension. Brain 1974;97:313-26.
7. Bulens C, DeVries WAEJ, Van Crevel H. Benign intracranial hypertension. J Neurol Sci 1979;40:147-57.
8. Dandy WE. Intracranial pressure without brain tumor. Ann Surg 1937;106:492-513.
9. Foley J. Benign forms of intracranial hypertension: "toxic" and "otitic" hydrocephalus. Brain 1955;78:1-41.
10. Guidetti B, Guiffre R, Gambacorta D. Follow-up study of 100 cases of pseudotumor cerebri. Acta Neurochir (Wien) 1968;18:259-67.
11. Johnston I, Paterson A. Benign intracranial hypertension. Brain 1974;97:289-300.
12. Lysak WR, Svien HJ. Long-term follow-up on patients with diagnosis of pseudotumor cerebri. J Neurosurg 1966;25:284-7.
13. Rush J. Pseudotumor cerebri. Mayo Clin Proc 1980;55:541-6.
14. Weisberg LA. Benign intracranial hypertension. Medicine (Baltimore) 1975;54:197-207.
15. Wilson DH, Gardner WJ. Benign intracranial hypertension with particular reference to its occurrence in fat young women. Can Med Assoc J 1966;95:102-5.
16. Greer M. Benign intracranial hypertension. V. Menstrual dysfunction. Neurology (Minneap) 1964;14:668-73.
17. Greer M. Benign intracranial hypertension. III. Pregnancy. Neurology (Minneap) 1963;13:670-2.
18. Benini A. Pseudotumor cerebri. Schweiz Med Wochenschr 1973;103:921-6.
19. Caroscio JT, Pellmar M. Pseudotumor cerebri: occurrence during the third trimester of pregnancy. Mt Sinai J Med (NY) 1978;45:539-41.
20. Elian M, Ben-Tovim N, Bechar M, Bornstein B. Recurrent benign intracranial hypertension (pseudotumor cerebri) during pregnancy. Obstet Gynecol 1968;31:685-8.
21. Gupta H, Venkateswar N, Prakash SK. Benign intracranial hypertension in pregnancy. J Indian Med Assoc 1980;75:93-4.
22. Meythaler H, Meythaler-Radek B. Zur frage des pseudotumors cerebri. Klin Monatsbl Augenheilkd 1973;163:200-4.
23. Powell JL. Pseudotumor cerebri and pregnancy. Obstet Gynecol 1972;40:713-8.

24. Shekleton P, Fidler J, Grimwade J. A case of benign intracranial hypertension in pregnancy. Br J Obstet Gynaecol 1980;87:345-7.
25. Bradshaw P. Benign intracranial hypertension. J Neurol Neurosurg Psychiatry 1956;19:28-41.
26. Davidoff LM, Dyke CG. Hypertensive meningeal hydrops. Am J Ophthalmol 1937;20:908-27.
27. Jefferson A, Clark J. Treatment of benign intracranial hypertension by dehydrating agents with particular reference to the measurement of the blind spot area as a means of recording improvement. J Neurol Neurosurg Psychiatry 1976;39:627-39.
28. Keltner JL, Miller NR, Gittinger JW, Burde RM. Pseudotumor cerebri. Surv Ophthalmol 1979;23:315-22.
29. Paterson R, DePasquale N, Mann S. Pseudotumor cerebri. Medicine (Baltimore) 1961;140:85-99.
30. Zuidema GD, Cohen SJ. Pseudotumor cerebri. J Neurosurg 1954;11:433-41.
31. Metropolitan Life Insurance Company Statistical Bulletin 1960;41:9.
32. Corbett JJ, Savino PJ, Thompson HS, et al. Visual loss in pseudotumor cerebri. Arch Neurol 1982;39:461-74.
33. Ketz E. Pseudotumor cerebri. Deutsche Zeitsch f Nerven 1956; 174:331-56.
34. Kraus H, Fanta H. Uber Hirndrucksteigerung ohne Tumor. Zentralbl Neurochir 1953;13:339-47.
35. Gusev VA, Deev AS. Pseudotumor syndrome in venous pathology of the brain in pregnant women. Zh Nevropatol Psikhiatr 1982;82:533-6.
36. McCullagh EP. Menstrual edema with intracranial hypertension (pseudotumor cerebri). Cleve Clin Q 1941;8:202-12.
37. Rothner AD, Brust JCM. Pseudotumor cerebri. Arch Neurol 1974;30:110-1.
38. Quincke H. Ueber meningitis serosa und verwandte Zustande. Deutsche Z Nervenheilk 1897;9:149-68.
39. US Bureau of Census. Statistical Abstracts of the United States 1981. Washington, DC, 1981.
40. Donaldson JO. Neurology of pregnancy. Philadelphia: W.B. Saunders, 1978:171-5.
41. Nickerson CW, Kirk RF. Recurrent pseudotumor cerbri in pregnancy. Obstet Gynecol 1965;26:811-3.
42. Palop R, Choed-Amphai E, Miller R. Epidural anesthesia for delivery complicated by benign intracranial hypertension. Anesthesiology 1979;50:159-60.
43. Traviesa DC, Schwartzman RJ, Glaser JS, Savino P. Familial benign intracranial hypertension. J Neurol Neurosurg Psychiatry 1976;39:420-3.
44. Foley KM. Is benign intracranial hypertension a chronic disease? Neurology (Minneap) 1977;27:388.
45. Ahlskog JE, O'Neill BP. Pseudotumor cerebri. Ann Intern Med 1982;97:249-56.
46. Greenhill JP, Friedman EA. Biological principles and modern practice of obstetrics. Philadelphia: W.B. Saunders, 1974.
47. Coffey CE, Ross DR, Massey EW, Olanow CW. Familial benign intracranial hypertension and depression. Can J Neurol Sci 1982;9:45-7.
48. Galbraith JEK, Sullivan JH. Decompression of the perioptic meninges for relief of papilledema. Am J Ophthalmol 1973;76:687-92.
49. Berkowitz RL, Coustan DR, Mochizuki TK. Handbook for prescribing medications during pregnancy. Boston: Little, Brown, 1981.
50. Redmond GP. Effects of drugs on intrauterine growth. Clin Perinatol 1979;6:5-19.
51. Greer M. Benign intracranial hypertension. II. Following corticosteroid therapy. Neurology (Minneap) 1963;13:439-41.
52. Roussounis SH. Benign intracranial hypertension after withdrawal of topical steroids in an infant. Br Med J 1976;2:564.
53. Britton C, Boxhill C, Brust JCM, Healton EB, Braun CW, Killian P. Pseudotumor cerebri, empty sella syndrome, and adrenal adenoma. Neurology (NY) 1980;30:292-6.