Stroke during Pregnancy and Puerperium in Young Females below the Age of 40 Years as a Result of Cerebral Venous/Venous Sinus Thrombosis

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SUMMARY

One hundred and thirty-eight cases of cerebral venous/venous sinus thrombosis during the puerperium have been studied. Maximum number of cases (112 cases) occurred in the age below 30 years. Signs and symptoms appeared in most of the cases (72 cases) during the first 7 days of post partum period. One hundred and eleven cases were multipara. Commonest signs and symptoms were fever (85 cases); headache (66 cases), convulsions (64 cases), altered consciousness (57 cases), hemiplegia (60 cases), papilloedema (48 cases), etc. Carotid angiography done in 55 cases revealed block in middle part of superior sagittal sinus in 24, and localised, dilated and tortuous cortical veins in 5. A statistically significant rise in serum triglycerides, phospholipids, free fatty acids, blood platelet count, platelet adhesive index, and fall in blood fibrinolytic activity were found as compared with normals. These factors may be playing a role in the etiology of cerebral venous/venous sinus thrombosis during puerperium.

Additional Indexing Words:

Cerebral angiography   Cerebrovascular accident   Stroke   Venous thrombosis   Puerperium   Lipids   Platelets   Fibrinolytic activity

CEREBROVASCULAR accidents have been recognized for many years as a hazard in young females during pregnancy and puerperium. Meniere¹⁾ was the first to demonstrate that cerebral venous catastrophes could occur during pregnancy and puerperium and Abercrombie²⁾ published the first case of puerperal venous sinus thrombosis with post mortem examination. The risk of stroke during pregnancy and puerperium, however, did not receive much attention until Symonds,³⁾ Martin⁴⁾, Martin and Sheehan,⁵⁾ and Stansfield⁶⁾ published their clinical and autopsy studies. In Western countries the disease is uncommon⁷⁾⁻⁹⁾ but it is the commonest cause of strokes in

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young females in India.¹⁰⁾⁻¹⁸⁾

In India the incidence of primary cerebral venous sinus thrombosis is around 4.5 per 1,000 obstetric admissions¹⁷⁾ as compared with Western incidence of 1 per 1,666 to 1 per 10,000 pregnancies.¹⁹⁾⁻²²⁾ In India about 25% of maternal deaths related to child bearing occur as a result of cerebrovascular accidents.¹⁷⁾ The etiology and pathogenesis of the increased tendency to cerebral venous sinus thrombosis during pregnancy and puerperium are still not clear. Various mechanisms have been postulated for its occurrence viz. retrograde spread from pelvic veins via vertebral veins during uterine contractions,²³⁾ damage to the superior sagittal sinus due to fluctuations in the intracranial pressure during delivery,²⁴⁾ and an increased thrombotic tendency because of changes in various coagulation factors.^{18), 24)-28)} All these theories, however, do not explain the occurrence of the disease in the 2nd and 3rd trimester of pregnancy on the one hand and in the second week of puerperium on the other.

MATERIAL AND METHODS

One hundred and thirty-eight cases who developed cerebral venous/venous sinus thrombosis during pregnancy and puerperium were studied. The diagnosis was mainly clinical, based on the established criteria.^{29), 30)} A detailed history, thorough physical and neurological examinations were recorded on a special proforma. In all cases eclampsia, pre-eclamptic toxemia, albuminuria, hypertension or oedema, metabolic causes of coma and convulsions, meningial infection, and intracranial neoplasms were excluded by appropriate investigations.

The following routine investigations were done in each patient (hemoglobin, total leucocyte count, differential leucocyte count, erythrocyte sedimentation rate); urine and stool examination; blood urea; blood sugar; blood for V.D.R.L.; electrocardiogram; X ray skull; cerebrospinal fluid (C.S.F.) examination for cytology, biochemistry, and V.D.R.L. The following parameters were specially studied, serum triglycerides³¹⁾; serum phospholipids³²⁾; serum free fatty acids³³⁾; serum lipoproteins³⁴⁾; blood fibrinolytic activity³⁵⁾ measuring combined plasminogen activator and plasmin activity on fibrin plates containing plasminogen with plasma of patients; platelet adhesive index and adhesive platelets.³⁶⁾ A contralateral percutaneous common carotid angiography was also done well below the bifurcation in the neck with serial pictures in anteroposterior & lateral views whenever possible. All these investigations with the exception of the radiological investigation were also done in the following groups to serve as controls for comparison:

Group A: 25 non-pregnant healthy women of child bearing age;

Group B: 25 healthy pregnant women nearing term;

Group C: 25 women in the first week of normal puerperium after 48 hours of delivery.

Three cases of arterial occlusion seen during this study were analysed separately.

OBSERVATIONS

Age: One hundred and twelve cases were below the age of 30 years (15–20 years 22 cases; 21–30 years 90 cases). No case was seen above the age of 40 years (31–40 years 26 cases).

Parity: Cerebral venous/venous sinus thrombosis was commonly seen in multipara (111 cases). Only 27 cases were primipara. Delivery was of full term and normal in all cases. There was neither any evidence of pre-mature or post mature delivery nor excessive bleeding. In all these cases delivery was conducted at home by an untrained nurse under inadequate

Table I. Clinical Features in 138 Cases

Name of symptoms and sign	No. of cases
Fever	85
Headache	66
Convulsion-generalised	40
-focal	24
Unconsciousness	57
Vomiting	49
Giddiness	6
Paresthesia	5
Irrelevant talk	5
Vertigo	2
Diplopia	2
Severe pain in paralysed limb	2
Blurred vision	1
Excessive salivation	1
<i>Focal neurological deficit</i>	
Complete hemiplegia	60
Papilloedema	48
Motor aphasia	35
Paraplegia with incontinence of urine and faeces	29
Quadriplegia	12
Isolated supranuclear 7th nerve palsy	12
Monoplegia	7
Hemiplegia with visceral involvement	3
Sixth nerve palsy	2
Hemianesthesia, cortical type	2
<i>Others</i>	
Neck rigidity	4
Oedema eyes	2
Thrombophlebitis legs	1

asepsis.

Time of onset of symptoms: More than half of the cases (72 cases) were within first 7 days, 44 cases between 8–14 days, and 21 cases after 14 days of the delivery. Only 1 patient developed cerebral venous/venous sinus thrombosis in the third trimester of pregnancy.

Clinical features: Clinical features are summarised in Table I. Headache was mostly unilateral limited either to the forehead or temple and was severe and persistent in 16 cases. Jacksonian seizure with a spreading tendency occurred in 5 cases. One case developed status epilepticus. Four patients presented with symptoms of raised intracranial pressure consisting of headache, vomiting, coma, papilloedema, and neck stiffness. Puerperal sepsis was seen in 4 cases only. Three patients developed terminal uraemia.

Cerebrospinal fluid (C.S.F.): Lumbar puncture was done in 97 cases. Detailed findings are shown in Table II. A raised C.S.F. pressure was the most frequent finding (80 cases). Red blood cells were seen in 10 cases. Proteins were raised in 34 cases (34.9%) of which 8 cases had protein of more than 250 mg% and all depicted xanthochromia. V.D.R.L. was negative in all the cases. No change in sugar or chloride was seen.

Cerebral angiography: Carotid angiography was possible in 55 cases only.

Table II. Cases C.S.F. Findings in 97

	No. of cases
Raised pressure	80
Xanthochromia	8
M/E R.B.C.	10
W.B.C.	10
<i>Protein</i>	
20–40 mgm%	63 (65.1%)
41–100 mgm%	22 (22.6%)
>100 mgm%	12 (12.3%)

Table III. Angiographic Findings in 55 Cases

Type of lesion	No. of cases
Normal arterial phase	55
Middle part of superior sagittal sinus not seen	24
Localized dilated tortuous cortical veins	5
Club shaped sinus deformity	1

Table VI. Serum Lipids in 50 Cases of Cerebral Venous Sinus Thrombosis and Controls

Group	S. trigly- cerides. (mg%)	S. Phospho- lipids (mgm%)	Serum free fatty acids (mg%)	Lipoproteins (%)		
				Beta	Pre-beta	Alpha
A. Non-pregnant	103±20.95	231±64.72	547±176.07	54±6.01	30±3.26	17±5.44
B. Healthy pregnant	105±19.47	283±69.29	600±309.22	61±9.41	26±3.09	14±4.23
C. Normal puerperium	124±33.62	299±89.88	912±375.99	60±8.49	27±4.95	13±4.73
D. Cerebral venous sinus thrombosis	153±51.6	292±58.4	763±329.09	58±7.75	27±4.43	15±5.40

Table V. Blood Fibrinolytic Activity in 19 Cases of Cerebral Venous Sinus Thrombosis and Controls

Group	Blood fibrinolytic activity lysis zone (mm ²)
A. Non pregnant	391±S.D. 158.69
B. Healthy pregnant	467±S.D. 170.55
C. Normal puerperium	347±S.D. 125.77
D. Cerebral venous sinus thrombosis	253±S.D. 70.78

The arterial phase was normal in all the cases. Detailed findings are shown in Table III.

Prognosis: Out of a total of 138 cases only 38 cases (27.5%) died while 100 (72.5%) cases recovered completely.

Serum lipids: There was a rise in almost all the lipid fractions in cases of cerebral venous/venous sinus thrombosis (Group D) as compared with non-pregnant women (Group A) and women who had a normal puerperium (Group C). This rise was statistically significant ($p<0.001$) in respect to serum triglycerides, phospholipids and free fatty acids (Table IV).

Blood fibrinolytic activity: There was a fall of blood fibrinolytic activity in cases of Group C and in cases of cerebral venous sinus thrombosis (Group D). The fall in cases of cerebral venous sinus thrombosis has been found to be statistically significant ($p<0.001$) in comparison with Group A and Group C patients (Table V).

Platelet adhesiveness: Platelet adhesive index and absolute number of adhesive platelets were higher in Group B patients, Group C patients and in cases of cerebral venous/venous sinus thrombosis (Group D) as compared with Group A patients, but the results were much higher in cases of cerebral

Table VI. Platelet Function Tests in 25 Cases of Cerebral Venous Sinus Thrombosis and Controls

Group	Platelet adhesive index	Platelet adhesive number (per cmm)
A. Non pregnant	1.05±S.D. 0.05	502.40±S.D. 5195
B. Healthy pregnant	1.10±S.D. 0.06	540.00±S.D. 2093
C. Normal puerperium	1.20±S.D. 0.089	645.00±S.D. 8230
D. Cerebral venous sinus thrombosis	1.35±S.D. 0.11	718.00±S.D. 3830

Table VII. Statistical Analysis of Cases of Cerebral Venous Sinus Thrombosis as Compared with Normal Delivery Cases

Group	Platelet adhesive index	Adhesive platelets (1 cmm)
C. Normal puerperium	1.20	64.600
D. Cerebral venous/venous sinus thrombosis	1.35	71.500
Difference	0.15	7.200
't' value*	5.4	36.4

* 't' value from the table for 48° freedom at 5% level of significance is 2.01. So values higher than 2.01 are statistically significant.

venous/venous sinus thrombosis than in any other group (Table VI and VII).

Leucocytosis was seen in 10 cases. It probably does not indicate infection but reflects the thrombotic process itself. One hundred and one (89.9%) cases had a hemoglobin of less than 10 Gm%. Only 12 (11.1%) cases had hemoglobin between 10–13 Gm%.

Arterial thrombosis: All the 3 cases of arterial thrombosis were conscious with complete hemiplegia, without seizures and papilloedema, normal C.S.F. and showed no improvement of the neurological deficit. Two cases showed extracranial internal carotid artery block in the neck and 1 case had middle cerebral artery block.

DISCUSSION

In India majority of patients with cerebral strokes associated with pregnancy and the puerperium have cerebral venous/venous sinus thrombosis.^{(11)–(13), (17), (18), (21), (30), (37)–(41)} On the other hand Cross et al⁽⁸⁾ have reported over 70% of these strokes as being due to occlusive cerebral arterial disease or ischaemic lesions unrelated to thrombosis of the intracranial venous system. Others have also considered cerebral venous thrombosis as a rare complica-

tion.^{7),9),29),42),43)} In India cerebral arterial occlusion is far less common and was proven in 47 (15.6%) out of a total of 301 patients reported.^{13),14),17),38),44)} In this study only 3 (2%) of our patients had arterial occlusion. Dashpande³⁷⁾ described the post mortem findings in 7 cases of cerebral venous sinus thrombosis and Banerjee et al⁴¹⁾ in 8 fatal cases seen over a period of 2 years of cerebral venous sinus thrombosis out of a total of 24 patients.

The diagnosis of cerebral venous sinus thrombosis is mainly clinical. The clinical picture of venous occlusion is often diagnostic. In arterial occlusion patients are usually conscious, seizures are rare, there is dense hemiparesis, papilloedema is absent, the C.S.F. is normal, neurological deficit persists, positive angiographic findings are more common and the prognosis for life is good, but on the other hand in cerebral venous sinus thrombosis, the mortality is high, deterioration in the level of consciousness is high, convulsions are common and those who survive have very little disability. Cerebral venous/venous sinus thrombosis should always be suspected in a previously healthy woman developing a triad, of varying degrees of paresis, headache, and convulsions after delivery.^{24),29),30)} The percentage of various clinical features associated with cerebral venous/venous sinus thrombosis during pregnancy and the puerperium vary because the development of these symptoms depends on the site and extent of the thrombosis of the sinuses, cortical veins or deep veins and the potentialities of the collateral circulation.

Headache occurred in 66 of our cases and may precede the other symptoms by several hours or days.^{30),43),45),46)} The intensity of the headache parallels the severity of the cerebral lesions and is mostly unilateral, limited either to the forehead, temple or occiput or may be diffuse⁴⁶⁾ but is severe and persistent. Headache is not necessarily due to raised intracranial pressure but is probably primarily caused by direct involvement of pain sensitive dural sinuses by thrombophlebitis.⁴⁷⁾ Convulsions may be generalized or focal²⁹⁾ but are most often the former.^{17),30),44)} They were seen in 64 cases in this series, being generalised in 40 and focal in 24 cases. Jacksonian seizures were seen in 5 cases and had a spreading tendency. Similar observations have also been made by other workers.⁴⁸⁾ Adversive seizures or status epilepticus have been recorded in the literature,¹⁷⁾ but were seen only in 1 case in this study. These are due to local cortical irritation caused by cerebral oedema, hemorrhage or venous engorgement and may be followed by paralysis within a few days.

All degrees of mono or hemiparesis, crossed hemiparalysis of face and limbs with or without sensory disturbances, speech defects and visual disturbances can occur.⁴⁵⁾ Hemiplegia which may be accompanied with varying degree of sensory and motor aphasia.^{11),17),21),30),38),44),47)} is the commonest

feature and was seen in 60 cases. Paraplegia with normal upper limbs and incontinence of urine and stools which occurs due to involvement of both para sagittal regions by the thrombosis⁴⁹⁾ was seen in 29 cases. Cortical anaesthesia seen in 2 cases without hemiplegia and aphasia has also been reported.⁵⁰⁾

Less commonly a syndrome of raised intracranial pressure consisting of headache, nausea, vomiting, photophobia, coma, papilloedema, neck stiffness, and raised C.S.F. pressure can occur due to sagittal sinus thrombosis.^{17),26),47),51)} Four patients were seen with such a syndrome. Papilloedema and signs of raised tension can occur in cerebral venous sinus thrombosis^{29),43),44),52)} and were seen in 48 of our patients. The meningitic picture seen in these cases is due to hemorrhagic infarction and subarachnoid hemorrhage¹⁷⁾ and was seen in 4 of our cases. Pyrexia found in 85 cases despite lack of evidence of active infection probably reflects the thrombotic process. It was also seen by other workers.^{17),21),29),44),47)} Sixth nerve palsy, seen in 2 cases in this series of cerebral venous sinus thrombosis, occurs due to thrombosis of the inferior petrosal sinus or severe venous stasis or upwards or downward displacement of the nerve due to swelling of the cerebral hemispheres.⁵³⁾ Paralysis of the 3rd, 5th or 6th nerve can also occur due to thrombosis of the cavernous sinus⁵⁴⁾ and extension of the thrombosis from the cavernous to the superior petrosal sinus can cause paralysis of the 5th nerve followed by 9th, 10th, and 11th cranial nerves.⁵³⁾ Thrombosis of the superior sagittal sinuses is sometimes associated with proptosis of the eyes without involvement of cavernous sinus^{55),56)} and occurred in 2 of our patients. Other less common clinical features include cortical blindness, persistent hypotension, postural hypotension, hypothermia, hypoglycemia with sweating associated with clinical akinetic mutism (alert but inert) probably due to ischaemia of hypothalamic area secondary to puerperal cerebral deep vein thrombosis.¹⁷⁾ Fluctuating or sustained hypertension can also occur^{21),38),47)} due to reflex response designed to overcome increased cerebrovascular resistance as a result of increased intracranial pressure.⁵⁷⁾

Examination of the C.S.F. is of little advantage for the diagnosis of cerebral venous/venous sinus thrombosis. Characteristic features of xanthochromia, markedly raised protein and varying degrees of R.B.C. and W.B.C. were seen in 10 cases, probably due to spread of thrombosis to the cortical veins.^{29),52),58)}

Earlier attempts to demonstrate occlusion of the major dural sinuses were made by sinography.^{59)–62)} The importance of cerebral angiography was stressed by Krayenbuhl⁶³⁾ and Weber⁵²⁾ and they considered that a deficiency in the venous phase of angiogram could be diagnostic of primary

cortical vein occlusion. Occasionally the arterial phase may show displacement as a consequence of cerebral edema or infarction in the involved hemisphere, which may become normal when repeated after 3–4 weeks.¹⁷⁾ Certain abnormalities of the venous phase suggest a sagittal sinus thrombosis.²⁰⁾ The middle part of the superior sagittal sinus was not seen in 14 cases and localized tortuosity of the cortical veins and a club shaped deformity of the sinus was seen in 5 and in 1 case, respectively. Failure to obtain filling of the basal sinus is a bad prognostic sign. Other abnormalities of venous phase suggestive of cerebral venous sinus thrombosis are:

- i) Slowing of the circulation which is almost a constant feature;
- ii) Cork screw vessels which do not reach the cortical surface^{63), 64)};
- iii) Failure to fill draining superficial cerebral veins in a circumscribed area of the brain in the relevant hemisphere and related to the patients' physical signs. This can be confirmed by comparison with the healthy side;
- iv) Failure to demonstrate all or part of a sinus or localised irregularity of the lumen⁴³⁾;
- v) Wide filling of the inferior sagittal sinus and internal cerebral vein along with failure in filling of the superior sagittal sinus.²⁹⁾

Of the various theories postulated for the occurrence of cerebral venous/venous sinus thrombosis one of the principal ones has been a hypercoagulate state.²⁵⁾ This hypercoagulate state is due to increase of factors VII and X²⁷⁾, hypophospholipemia,⁶⁵⁾ hyperlipemia, increased platelet adhesiveness,²⁸⁾ hyperfibrinogenemia,⁶⁶⁾ and decreased fibrinolysis.^{66), 67)} A 30% rise in platelets has been demonstrated during puerperium⁶⁸⁾ and this rise is even higher if a severe post partum hemorrhage occurs.⁶⁹⁾ Peak increase in platelets occurs about the 10th post partum day,²⁵⁾ the time when venous thrombosis usually occurs.⁶⁸⁾ In addition, there occurs increased platelet stickiness during pregnancy and the puerperium.^{25), 28)} In this series levels of platelet adhesive index and absolute number of adhesive platelets were higher during pregnancy and the puerperium as compared with non pregnant controls. A rise in serum lipids occurs during pregnancy and this rise continues even during the puerperium^{28), 70)–74)} irrespective of the socio-economic status and diet taken.⁷⁴⁾ A statistically significant rise in serum triglycerides, phospholipids, free fatty acids and increase in platelet adhesiveness as well as absolute number of adhesive platelets found in this study have also been reported by other workers.^{17), 28)} Increased platelet aggregation can occur not only with high serum levels of fatty acids,⁷⁵⁾ but can also be induced by long chain fatty acids in experimental models.^{76), 77)} A rise in beta and prebeta lipoproteins occurs in cerebral venous sinus thrombosis^{44), 78)} and the affinity between platelets and certain lipoproteins has also been stressed.⁷⁹⁾

Plasma fibrinogen levels rise during pregnancy^{25),70),75),80)} which may increase by 50% in the last trimester⁸¹⁾ and remain elevated during the puerperium.^{13),66)} Increased values of fibrinogen occur in cerebral venous sinus thrombosis⁴⁴⁾ and the progressive rise in serial estimations is a useful warning sign of its progression.¹⁷⁾

In addition, there is evidence that fibrinolysins are reduced during pregnancy and the puerperium.^{66),67)} A statistically significant fall in fibrinolytic activity was seen in cases of cerebral venous sinus thrombosis as compared with normal puerperium.

This increased coagulability may be playing a role in the etiology of cerebral venous/venous sinus thrombosis occurring during the puerperium in young females below the age of 40 years.

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