

POST-CATARACTOMY DELIRIUM : A TWO YEAR PROSPECTIVE STUDY

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A two year prospective study of 221 consecutive inpatients undergoing cataractomy revealed the incidence of post-cataractomy delirium to be 1.8%. While in one case the delirium was due to anticholinergic toxicity, in the remaining three cases no organic cause was evident. Sensory deprivation was present in two patients. Preventive measures for the condition are discussed.

Certain patients who enter the strange environment of a hospital to undergo cataractomy, which is an anxiety-producing operation and is followed by temporary loss of vision, may develop post-cataractomy delirium (PCD). A recent review of the western literature found the incidence rising to 60-70% with pre-existing brain syndromes (Sebert, 1986). History of psychiatric disorders and alcohol increases the risk of developing this disorder. A number of theories have been postulated about the relation between eye surgery and delirium. Few have proposed the cause to be sensory deprivation, although the absence of patching is not necessarily preventive. Others support a combination of susceptibility on the basis of age and pre-existing brain dysfunction, in conjunction with drug effects, particularly the anticholinergics.

The absence of Indian studies on this subject prompted us to undertake a prospective study to determine the incidence, investigate the causes and suggest preventive measures for this little understood condition.

MATERIAL AND METHODS

Two hundred and twenty one consecutive inpatients (128 male & 93 female) undergoing cataractomy at a military hospital from 1 Jan 88 to 31 Dec 89 were included in the study with their consent. Mean age of the patients was 60.14 (range 8 to 88) years.

Each patient was interviewed once pre-operatively and at least thrice post-operatively. The pre-operative interview included a medical-surgical history, psychiatric history and mental status examination, including memory testing. Specific inquiry was made about past and family history of psychiatric disorders, past history of delirium, concurrent drug administration and alcohol and drug addiction. All patients underwent the following investigations : blood ESR, Hb%, TLC, DLC, BT, CT, Urinalysis, LFT, blood sugar (F & PP), blood urea and ECG (in patients over 45 years of age) Extracapsular extraction of lens or intracapsular cryoextraction of the lens was performed under local anaesthesia. After the operation all patients were detained in the post-operative ward for five days. A mental status examination including memory testing was repeated on the evening of the operation, 48-72 hours after surgery and 6-7 days after surgery. PCD was diagnosed following the criteria of Summers & Reich (1979) which requires the presence of disorientation and disturbance of recent or remote memory as well as 2 or more of the following 6 symptoms : delusions, hallucinations, illusions, depersonalisation, change in affect (anxiety, fear, depression or euphoria) and abnormal psychomotor activity.

After the onset of PCD the patients were repeatedly examined to rule out any organic cause for delirium and the following investigations were carried out : Blood ESR, Hb%, TLC, DLC, urinalysis, LFT SGOT, SGPT, alkaline phosphatase, blood sugar (random), blood

urea, serum creatinine, serum electrolytes, ECG, fundoscopy & X-ray skull.

In the first year of our study 4 patients developed PCD. While anticholinergic drug toxicity was the precipitating factor in 1, sensory deprivation was incriminated in two cases. In view of the above, a strategy for the prevention of PCD was evolved which included greater caution in administering drugs to these patients, minimising sensory deprivation and anxiety by detailed explanation of the operation by the ophthalmologist, admitting the patients to the post-operative ward the day prior to surgery so that he became familiar and oriented to the new environment, repeated reassurance by the nursing staff both pre and post-operatively and by avoiding pad & bandage in patients more than 70 years, those with extreme anxiety, patients with aphakia/loss of vision of the unoperated eye and those with impaired hearing.

RESULTS

Four (3 male & 1 female) of the 93 patients (59 male & 34 female ; mean age 59.5 years ; range 23-83 years) undergoing cataractomy in the first year of our study developed PCD. The incident of PCD in the first year was 4.3%. None of the 128 cataractomy patients (69 male 59 female ; mean age 60.6 years ; range 8-88 years) in the second year of our study suffered from PCD giving a zero incidence of PCD. The overall incidence of PCD in our study was 1.4%.

The mean age of the four patients who developed PCD was 66.4% years (range 56-75 years). PCD occurred on the 2nd to 4th (average 3) post-operative days (Day of surgery counted as day one) and lasted 1 to 5 (average 3) days. None of these patient had a past or family history of psychiatric disorders. None of them suffered from chronic organic brain syndrome, renal disease or other medical illness. One patient suffered from uveitis post-opera-

tively. Apart from anticholinergic toxicity in this patient no organic cause was evident. One patient had aphakia of the unoperated eye along with impaired hearing while another had loss of vision of the unoperated eye due to leucoma adherent. These two patients obviously suffered from significant sensory deprivation after the operation.

DISCUSSION

Though the number of patients with PCD in the present study is too small for reliable statistical comparisons, several points of interest emerge.

The mean age (60.02 years) and age range (8-88 years) of our patients who did not develop PCD was not remarkably different from that of our patients with PCD (mean age 66.5 years : range 56-57 years). Thus, in agreement with the conclusions of Summers & Reich (1979) our data disproves the theory that delirium is associated with older age. Our data did not also reveal any association of PCD with medical illness or previous psychiatric illness (Peru & Guida, 1937) as none of our PCD patients gave such a history. Since all the patients with PCD in our study had normal renal function, the hypothesis of renal autotoxicity having a etiological role in PCD (Summers and Reich, 1979) was also not supported. However, an association with blindness or reduced vision in the unoperated eye (Ziskind *et al.*, 1960) and impaired hearing (Singh *et al.*, 1984) was strongly supported. Two of our patients with PCD definitely had sensory deprivation and improved after removal of the patch. Thus our study strongly supports an aetiological role for sensory deprivation in PCD. From table-1 it is obvious that in the majority of PCD patients reported in the literature there was no obvious cause for the delirium. It seems likely that sensory deprivation may have played an aetiological role in at least some of these patients.

Table-1 : Probable predisposing factors for PCD

Authors	Number of patients studied	Patients with PCD N (%)	Probable predisposing factors					
			Psychiatric disorders			Post surgical complications (n = 18)	Medical disease	
			Alcohol abuse (n = 15)	Chronic organic brain syndrome (n = 21)	Others (n = 3)		Renal (n = 8)	Others (n = 6)
Dupytren (1833)	21	2 (9.5)	-	1	-	1	-	-
Posey (1900)	-	19	-	2	-	1	-	-
Finlay (1904)	194	1 (0.3)	-	-	-	-	1	-
Parker (1913)	376	11 (2.9)	-	-	-	1	1	-
Brownell (1917)	962	30 (3.1)	11	-	-	1	-	-
Fisher (1920)	200	4 (2.0)	-	-	-	2	-	-
Lowe (1922)	-	1	-	-	-	-	1	-
Thomas (1926)	-	2	-	1	1	1	2	-
Preu & Guide (1937)	-	4	-	-	1	-	1	1
Boyd & Norris (1941)	-	1	-	-	1	-	-	1
Linn <i>et al</i> (1954)	21	13(61.9)	-	13	-	-	-	-
Weeisman & Hackett (1958)	-	2	-	1	-	-	-	-
Ziskind <i>et al</i> (1960)	88	14(15.9)	4	2	-	5	-	-
Steanly (1976)	-	1	-	-	-	-	-	1
Summers & Reich (1979)	27	2 (07.4)	-	1	-	1	2	1
Singh <i>et al</i> (1984)	-	1	-	-	-	1	-	1
Dash & Saxena (1989)	-	5	-	-	-	3	-	1
Chaudhury <i>et al</i>	221	4 (1.8)	-	-	-	1	-	-

Finally, in agreement with few earlier reports (Summers & reich, 1979; Dash & Saxena, 1989) our study supported the fact that in some cases PCD is an acute anticholinergic syndrome.

The 1.8% incidence of PCD in the present study is some what less than the incidence of 3.9% obtained from pooled data from 12 studies (Dupuytren, 1833; Schnabel, 1880; Finaly, 1904; David, 1910; Parker, 1913; Burns, 1916; Brownell, 1917; Fisher, 1920; Linn *et al.*, 1954; Ziskind *et al.*, 1960; Jackson, 1969; Summers & reich, 1979). However, the incidence of PCD in the present study is remarkably less than the reported incidence of 16% found in a recent review of the western literature (Serbert, 1986).

This is surprising considering the fact that with the advanced medical care available in these countries the incidence of PCD should have been much less. One possible reason could be that with the increasing sophistication and mechanisation in medical care less importance is placed on the establishment of a good doctor-patient relationship which had been claimed to decrease the incidence of PCD (Weisman & Hackett, 1958).

One important reason for the low incidence of PCD in the present study was that none of the patients in the second year of our study developed PCD. The zero incidence of PCD in the second year of our study was surprising especially because the number of cataractomy patients were infact more in the second year as compared to the first year (128 vs 93). This also could not be explained on the basis of differences in the demographic characteristics of the patients. In view of the small numbers this could have occurred purely by chance and the contribution of the preventive measures taken by us remain speculative. However we strongly feel that this merits further study with larger samples.

In conclusion we can state that the aetiology of PCD is multifactorial. In addition to

alcohol abuse, chronic organic brain syndrome, post-surgical complications, renal disease and drug toxicity, sensory deprivation also plays an important role in its causation. Therefore, in addition to specific and symptomatic therapy, steps to orientate the patient and increase sensory stimuli would hasten recovery of the patient. An awareness of the condition, cautious use of anticholinergic drugs along with steps to avoid sensory deprivation plays important roles in preventing this condition.

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