

Confusional state and cerebral infarcts

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Summary: Thirteen patients with confusional state and cerebral infarction were studied. Seven patients had optic pathway alterations. On computed tomographic scan, 2 patients had multiple infarctions and 10 had single infarctions, predominantly located in the temporo-occipital associative cortex. One patient had a normal scan. Reduction of 'selective attention', 'release' hallucinations, amnesic syndrome and secondary individual adjustment could explain the confusional state.

Introduction

Confusional state (CS) has long been considered the consequence of acute diffuse cerebral dysfunction or injury to specific cerebral areas, generally medial in the Wernicke-Korsakoff syndrome. CS due to cerebral infarction, not attributable to metabolic encephalopathy, acute intoxication, drug withdrawal or infarction, is relatively uncommon.

Agitated delirium,¹ acute confusional state,² vascular psychotic organic brain syndrome,³ delayed psychosis,⁴ transient behavioural syndrome⁵ or agitated confusion⁶ are all terms used to designate CS in patients with ischaemic cerebral lesions, sometimes with scanty localizing signs.

Our paper describes 13 patients with CS and ischaemic lesions of different size and location, and underlines the difficulty in assigning this complex syndrome to a specific area.

Methods

We reviewed the records of patients admitted to the Neurology Service of the 'Primer de Octubre' Hospital (Madrid, Spain) from February 1981 to December 1985. CS was defined by the presence of disorientation, inattentiveness, poor concentration, agitation and disordered perception (illusions, delusions and hallucinations). Patients with metabolic encephalopathy, acute intoxication, drug withdrawal, systemic infection or pre-existent dementia were excluded. Alcoholism, malnutrition or chronic ingestion of drugs with psychotropic effects were specifically

excluded in the cases selected. Thirteen patients (9 males and 4 females, 47–80 years) met these criteria. Computed tomographic (CT) scan was performed in all cases.

Results

Results are summarized in Table I. Onset of CS was simultaneous with development of neurological deficits in 7 cases. In 5 patients, CS onset was delayed 24 hours (2 cases), 1 week, 1 month and 2 months (1 case each) with respect to the onset of other deficits. The course of CS was transitory in most patients, but in 4 the confusional syndrome persisted after one year (1 case) and two years (3 cases) of follow-up. Nine patients had arterial hypertension and 7 had suffered prior ischaemic episodes. There were optic pathway alterations in 7 patients: 4 homonymous hemianopsia, 1 homonymous quadrantanopia and 2 amaurosis. Six patients had motor or sensorial deficits, never severe. One patient suffered aphasia. No patient evidenced dementia in the strict sense.⁷ The manifestations of CS were similar in these patients. In 8, vivid complex hallucinations of persons or animals in motion induced intense motor activity evoking 'delirium tremens'. Hallucinations were occasionally terrifying, producing panic in the patient. The hallucinatory imagery constantly coexisted with perceptive illusions and appeared or became more accentuated at night. Eleven patients were agitated at some time, requiring sedation or mechanical restraint. The aggression of case 4 led to transfer to a specialized psychiatric centre. Four patients had theft and persecution delusions. In no case was leukocytosis found in peripheral blood and a clear chest X-ray was present in all patients at the time of appearance of the confusional syndrome.

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Table I Clinical details

Case	Age	Sex	Cerebrovascular antecedents	Confusional state	Onset	Duration	Associated signs	Computed tomography
1	77	M	—	Delusion. Persecution delirium. Insomnia. Agitation	Immediate	3 months	Left homonymous hemianopsia	Right temporo-occipital infarction. Generalized wide sulci
2	72	F	—	Terrifying illusions and hallucinations. Agitation	24 h delay	15 days	Cataracts	Left temporo-occipital infarction
3	76	M	—	Disorientation. Poor memory. Anosognosia. Nocturnal agitation. Delusions	Immediate	20 days	AHT. Aphasia. Right homonymous hemianopsia	Left temporo-occipital infarction. Generalized wide sulci
4	52	M	Vertebrobasilar TIAs	Hallucinations. Agitation. Aggression. Admitted to psychiatric centre	24 h delay	6 months	AHT. Left hemiparesis. Left homonymous hemianopsia	Right parieto-temporal infarction. Left frontal infarction
5	70	F	—	Delusions. Hallucinations. Agitation	Immediate	Unknown (>1 mo)	Right homonymous superior quadrantanopia. Right hyperpathia	Left occipital infarction
6	68	M	Transitory global amnesia episodes. Stroke with right hemiparesia	Disorientation. Nocturnal panic. Fear and terrifying illusions. Agitation	Immediate	Persists after 2 years	AHT. Nystagmus. Dystonia	Left temporo-occipital infarction
7	68	M	Stroke with left hemiparesis	Vivid dreams. 'War' delusions. Occasional agitation	Immediate	6 months	AHT. Aphasia. Double hemianopsia	Left temporo-occipital infarction. Generalized intense wide sulci
8	64	F	Stroke with right hemiplegia	Disorientation. Illusions and hallucination. Nocturnal predominance	1 month delay	1 month	AHT. Right hemiparesis	Normal
9	52	M	3 TIAs: 2 left hemiparesis, 1 right hemiparesis	Terrifying hallucinations. Panic episodes. Emotional incontinence	Not known	Persists after 2 years	AHT	Right parieto-temporal infarction. Generalized wide sulci
10	71	M	—	Disorientation. Confabulation. Agitation	2 months delay	6 months	AHT	Left temporal infarction. Generalized wide sulci
11	53	M	—	Delusions: 'poisoning' delirium. Nocturnal illusions	Immediate	Persists after 1 year	AHT. Left hemiparesis	Right parieto-temporo-occipital infarction. Generalized wide sulci
12	47	M	2 strokes with right hemiparesis	Delusions: 'theft' delirium. Hallucinations. Agitation	7 day delay	1 month	AHT. Right homonymous hemianopsia. Right hemiparesis	Left occipital infarction. Bilateral basal ganglia infarction
13	80	F	Sudden blurring of vision (10 years earlier)	Complex hallucinations. Confabulation	Immediate	Persists after 2 years	Bilateral amaurosis. 'Central' right facial palsy	Left occipital infarction

AHT: arterial hypertension; TIA: transient ischaemic attack.

On CT scan, the size and location of the infarctions varied widely (Figure 1). Case 8, with a history of stroke 5 years earlier and the present sudden agitated delirium and right hemiparesia, had a normal CT. Two cases presented multiple cerebral infarctions on CT: case 4 with right temporal and left frontal infarction and case 12 with left occipital infarction and small bilateral infarctions in basal ganglia. Six patients had single infarctions with diffuse widening of sulci, although this was always moderate. Three of these cases had lesions in the right hemisphere (parieto-temporal, parieto-temporo-occipital and temporo-occipital) and 3 in the left hemisphere (2 temporo-occipital and 1 temporal infarct). In 4 patients with single lesions and normal sulci, the infarction was located in the left hemisphere (3 temporo-occipital, 1 occipital lobe). The size of the lesion varied but was never large. The CS morphology was similar in every case.

Discussion

CS caused by ischaemic cerebral lesions is not as rare as the scarcity of reports suggests.² It is probable that a paucity of focal neurological findings leads to some cases being missed. Of the 13 patients reported here, only 6 presented sensorimotor deficits and these were never severe. In most of our patients, the diagnoses of metabolic encephalopathy, acute intoxication, infection or alcohol withdrawal were initially considered. In spite of the complexity and relative lack of definition of the syndrome, the morphology of CS was similar in all cases. Subgroups with clinicopathological significance could not be isolated. The most prominent elements were disorientation, distraction by irrelevant stimuli, perceptual errors, complex and terrifying hallucinations, vivid dreams, wild fantasies and sometimes paranoid delusions. Inability to sleep, worsening at night, accompanied by agitation was almost constant. Nocturnal disorientation has



Figure 1 Diagrammatic representation of location of infarcts.

been considered by some authors⁸ as characteristic of vascular dementia. Our patients did not suffer dementia in the strict sense and the lesions were frequently single. The interval between stroke and onset of CS ranged from 1 to 60 days. This delay has been noted by Levine and Finklestein.⁴ Recovery of our patients was variable. In 4 'chronic' patients, CS persisted for one year or more after the initial episode. Two showed atrophy on CT and one had multiple lesions. Horenstein *et al.*¹ and Medina *et al.*⁹ reported 9 patients with CS in whom symptoms persisted for weeks or months with death resulting from intercurrent illness. Infarcts were bilateral in 3 of these cases. Medina *et al.*¹⁰ reported 4 similar cases, one with confusion and delirium that persisted until death 17 months later.

Seven of our patients had visual impairment due to interruption of the optic pathway. This observation has been reported by various authors^{1,6,10,11} who consider that this situation of deafferentation contributes to the genesis of CS in cerebral infarction.

The plurality of infarction sites in our cases was striking. Two of our patients had multiple infarcts, as did one case of Hyland,¹² 3 of Horenstein *et al.*¹ and 4 of Reuck *et al.*¹³ The presence on CT of wide sulci associated with single ischaemic lesions in 6 of our patients did not indicate prior dementia and its clinical significance is dubious. Disregarding the presence of wide sulci, 3 patients had a lesion in the right hemisphere and 7 patients in the left hemisphere. This predominance of lesions in the left hemisphere runs counter to the experience of most authors, who report a greater incidence in the right hemisphere.^{2,4,6,14} Alone or in combination with other sites, the temporal lobe (11 patients) and occipital lobe (10 patients) were the areas most commonly affected. Temporo-occipital infarction was present in 7 patients. Medina *et al.*¹⁰ consider the 'agitated delirium and visual impairment' syndrome a manifestation of medial temporo-occipital infarction. Although parietal lobe affection has been often reported,^{2,4,6,14,15} only 3 of our patients had lesions in this area, in conjunction with temporal (2 cases) and temporo-occipital infarcts (1 case). Our case 4 suffered right temporo-occipital infarction associated with left frontal infarction. Mesulam *et al.*² described a CS patient with fronto-parietal infarction as the anatomic substrate. Finally, our case 12 had a left occipital ischaemic lesion and bilateral infarctions in basal ganglia. Confusion was prominent in one of Fisher's¹⁶ patients, who had infarction involving mainly the internal capsule.

According to Mesulam *et al.*² CS consists of a complex pattern of deficits in mental status. Disorders in at least four fundamental functions can be identified, the combination of which can partly explain

CS. These are reduction in 'selective attention', 'release hallucinations', amnesic syndrome and secondary adjustment mechanism. Inattentiveness is a basic component of CS. The term 'attention' refers to at least two processes, one tonic ('arousal') and the other phasic ('selective attention'). The latter requires constant and rapid exercise of perception of external and internal stimuli. Integration of this information with data from experience to obtain a correct identification of reality, selection of its priority elements and elaboration of an appropriate conduct² corresponds to the association areas of highest order. Our patients' infarctions mainly correspond to those areas in which injury interferes with the integrative process of selective attention. Cogan¹⁷ proposes the term 'release' hallucinations to refer to non-epileptic visual hallucinations that occur in areas of visual loss; such hallucinations appear to be more common than is generally appreciated. They are continuous, simple or more complex, and coexist with illusions. They have been described in lesions of the eye, optic nerve, chiasm and optic tract. Occipital and temporo-occipital infarctions with 'release' hallucinations have been reported.¹¹ Amnesia, in the sense of reduction in the ability to learn new information, attributed to infarction of medial temporal structures, has been well-documented in posterior cerebral artery occlusion.¹⁸ These authors describe 10 patients with amnesia associated with either unilateral or bilateral visual defects. Four of their cases appeared to have left-sided unilateral occlusion. The clinical status of our patients made thorough analysis of possible memory disorders by psychological testing difficult, except in case 3. However, we can speculate by anatomo-clinical analogy that amnesic syndrome may have participated in the origin of CS.

According to Engel,¹⁹ clinical manifestations of brain syndromes derive from a combination of two components: (a) the direct effect of brain impairment resulting in cognitive disturbances and (b) the individual's efforts to cope with limitations, producing exaggerated adjustment mechanisms and release of basic personality traits. Many aspects of the behaviour disorder, such as agitation, are probably 'secondary', that is, individual responses to the 'primary' ischaemic deficit. These neuropsychological speculations must remain tentative.

We conclude that CS in patients with cerebral infarction is more common than generally appreciated. The highest associative temporal areas are those most frequently affected. Cerebrovascular disease must be considered in the differential diagnosis of CS.

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