Pseudodementia And Dissociative Phenomena

Псевдо-деменция и диссоциативные явления.

Summary. Psychiatric disorders, characterized by non organic deterioration of cognitive functions and reversibility or symptoms remission, have been called pseudo-dementia. In this report the Author, also from a historical perspective, presents many diseases clinically similar to dementia, showing the clinical features helpful in distinguishing pseudo-dementia from dementia, and analyzes with particular attention Ganser's syndrome and simulated dementia. Psychiatric disorders can mimetized dementia, and relationship between depression and dementia is complex: while depression may mimic dementia, it is more common for depression to be superimposed on dementia, and difficulties in differentiating the two conditions arise because of overlapping symptoms. The extensive discussion about pseudodementia lead to numerous studies, and the Author believes that pseudodementia, due to its eterogeneity, is better conceptualized as a broad category with many subtypes.

Резюме. Психиатрические расстройства, проявляющиеся неорганическими расстройствами познавательных функций и обратимостью или ремиссией симптомов, были названы псевдо-деменцией. В этой статье автор, также с исторической точки зрения, рассматривает много болезней, клинически подобных деменции, показывая клинические особенности, полезные в дифференциации псевдо-деменции от деменции, с особым вниманием анализирует синдром Гансера и симулируемую деменцию. Психиатрические расстройства могут имитировать деменцию, и с оотношение между депрессией и деменцией является сложным: в то время как депрессия может походить на деменцию, она все же более часто накладывается на дем енцию, и возникают трудности в дифференциации двух состояний из-за перекрещивающихся симптомов. Обширное обсуждение относительно псевдо-деменции обусловили многочисленные изучения этого явления, и автор считает, что псевдо-деменция, учитывая ее гетерогенность, лучше концептуализируется как ши рокая категория с многими подтипами.

Many different diseases, some ones with non organic pathology, are clinically similar to dementia, namely depression. So it is quite comprehensible the need for a concept that embrace illnesses which appeared dementia-like but considered to have nonorganic and reversible cognitive deterioration. This reversibility is obtained by treatment or the passage of time. This kind of disorders has been called Pseudodementia.

Historically, it was used, in the 1880s, Vesanic Dementia to designate reversibles demential syndromes. By the end of the century this expression was replaced by Pseudodementia (Wernicke). Ganser (1898) describes the syndrome named after him (Bulbena and Berrios, 1986). Ganser Syndrome and Pseudodementia became, wrongly, equivalents (Kiloh, 1981; Emery, 1988).

In a historical point of view, Pseudodementia was characterized by non organic deterioration of the cognitive function and reversibility or symptoms remission. This reversibility is, of course, not compatible with the dementia concept.

These characteristics lead to three dichotomies, concerning dementia and pseudodementia:

Organic - Non organic Structural Functional Irreversibility Reversibility.

Pseudodementia, therefore, seems as a disorder which appears to be degenerative but reverts with treatment or with time as we said above. Kiloh (1961) defines Pseudodementia, a purely descriptive term, as a clinical picture that mimics dementia but which course

proves this diagnosis wrong. It is thus viewed as a dementia copy or caricature and not as a dementia category or an autonomous nosologic entity. Caines (1981), in a very complex and tautologous way, defines pseudodementia as an "intellectual impairment in patients with a primary psychiatric disorder in which the features of intellectual abnormality resemble, at least in part, those of a neuropathologically induced cognitive deficit. This neuropsychological impairment is reversible, and there is no apparent primary neuropathological process that leads to the genesis of this disturbance". Lishman (1987) describes pseudodementia as a "number of conditions in which a clinical picture resembling organic dementia presents for attention yet physical disease proves to be little if at all responsible". Pseudosenility (Kiloh, 1961; Libow, 1972) or Reversible Dementia were used as synonymous of Pseudodementia. Bulbena and Berrios (1986) remark that there is no consensus on the use and application of the diagnosis of pseudodementia. Some authors (eg. Eisdorfer and Cohen, 1978; Libow, 1977) think that pseudodementia may be the result of a wide range of physical, psychological or environmental conditions. These conditions would be causes of secondary dementias, potentially reversibles and which prevalence is calculated as 10/15% of the whole range of dementias.

Actually, all that people with apparent dementia are not necessarily suffering from chronic organic brain syndrome and it is indispensable to ensure if we are not dealing with a case of reversible cognitive disorder and thus fail to do an adequate treatment.

In Tab. 1 we can see the most important causes of secondary dementias, some ones leading to an acute syndrome and others to a chronic syndrome, many of them treatable. Let us emphasize that many of these conditions are overlapping with others; for instance, many diseases lead to metabolic changes and the inverse is also true. Otherwise the elderly is vulnerable to illness

Endocrine	Hypo or hyperthyroidism		
	Hypo or hypercortisolism		
	Hyperparathyroidis		
	Hypo and hyperglicemia		
	Hypo and hypercalcemia		
	Hypopituitarism		
	(eg. chronic cardiovascular and		
	pulmonary diseases)		
Inflammatory	Connectivopathies with		
	intracerebral vasculitis		
Infectious	Acute and subacute (eg.		
	Pneumonia)		
	Chronic (eg. Abscess)		
	HIV / AIDS		
	Whipple's disease		
	Prion diseases		
	Human		
	Creutzfeld – Jakob's disease		
	Fatal familial insomnia		
	Kuru		
	Gerstmann - Straussler -		
	Scheinker Syndrome		
	Animal		
	Scrapie		
	BSE		
Metabolic	Electrolyte disorders Azotemia		
	Vit. B6 / B12 deficiency Folate		
	deficiency Pellagra		
	Malabsortion syndrome		
	Sprue		
	Alcoholism		
	Nutritional deficiency		
	Renal / hepatic failure		

Tab. 1	- Causes of	sencondary	dementias ((10/15%)) - Potentially 1	reversibles

Neurological	Epilepsy		
	Chronic subdural hematoma		
	Normal – pressure		
	hydrocephalus		
	Meningitis / encephalitis		
	Encephalopathies		
	Neurosyphilis		
	Brain tumors		
	Head injury		
	Dementia of boxers		
	Sensory deprivation		
Medicine	Drug intoxications		
related	Adverse effects		
	Self-prescriptions Interactions		
	Misuse / errors Polypharmacy		
Psychological /	Medical procedures / doctor		
environmental	Fear of losing home		
	Clinical discussion with		
	young relatives		
	Hospital fear		
	Recent affective loss		

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and prone to malnutrition states by deficiency of care or absent dentures. Commonly old people suffer from misuse and errors of self-administration of medication. The alcoholism is usual in the elderly. The aged is also subject to psychological and environmental pejorative factors. We can conclude that the differential diagnosis of true dementia and pseudodementia is essential. According to Wells (1979), pseudodementia is a syndrome in which dementia is mimetized by a functional psychiatric disorder, usually depression.

This author built a clinical picture differentiating pseudodementia from dementia, separating in this way those patients which difficulties are primarily organic from those which difficulties are primarily functional.

Clinical course and history	Pseudodementia	Dementia
Family aware of dysfunction	Common	Unusual
Onset dated with precision	Common	Unusual
Symptoms duration before medical help	Short	Long
Symptoms progression	Rapid	Slow
Previous psychiatric disorder	Common	Unusual
Cognitive function	Pseudodementia	Dementia
Attention	Preserved	Faulty
Concentration	Preserved	Faulty
Types of answer	"Don't know"	"Don't remember"
Equally deficient recentand remote memory	Common	Unusual
Memory gaps	Common	Unusual
Performance in tasks of similar difficulty	Variable	Consistently poor
Complaints and clinical behavior	Pseudodementia	Dementia
Nature of complaints	Cognitive loss	General
Form of complaints	Detailed	Vague
Disability	Exaggerate	Concealed
Capacities	Minimized	Maximized
Motivation	Poor	Excellent
Coping	Minimal	Adequate
Emotional reaction	Anxiety	Indifference
Depressive affect	Pervasive	Labile or shallow
Loss of social skills	Precocious	Late
Consistency between cognitive and behavioral dysfunctions	Inconsistent	Consistent
Nocturnal accentuation of dysfunction	Absent	Common

Tab. 2 - Pseudodementia and Dementia

Tab. 3 - Psychiatric disorders which provoke pseudodementia

Disorders simulating dementia	Conscious and intentional	Malingering / Factitious disorder	
		Hysteria or Conversion / Dissociation	
	Inconscious		
Psychiatric disorders which provoke severe cognitive mpairment not necessarily progressive or irreversible or degenerative	 Depression – depressive dementia (McHugh and Folstein, 1978) Mania Schizophrenia – late, "Bouffonerie (Bleuler, 1924)" OCD Depersonalization Mental retardation GAD Delirium 		
Ganser syndrome			

He recognizes that the symptoms and signs presented by the elderly are often due to both organic and functional factors.

The clinical features helpful in distinguishing pseudodementia from true dementia are shown in Tab. 2. According to Kiloh (1981) as Pseudodementia may be provoked by a number of psychiatric disorders it is better to use the plural term Pseudodementias. In this more restrictive point of view we can see in Tab. 3 the psychiatric disorders which provoke pseudodementia.

Concerning the disorders simulating dementia the Tab. 4, 5 and 6 show, in a synthetic way, their

characteristics and in Tab. 7 (Sachdev and Kiloh, 1994) their differential diagnosis.

Concerning hysterical pseudodementia the presumption of unconscious simulation is not certain as many follow-up studies have shown (Tissenbaum, Harter and Friedman, 1951; Slater, 1965; Merskey and Buchrich, 1975).

Current trend to drop the term hysteria in favor of somatoform and dissociative disorders (APA, 1987) has proved useful. Nevertheless, these terms don't seem the more accurate to describe those syndromes.

Ganser syndrome was considered for many years

Tab. 4 - Malingering characteristics

•Uncommon

•Difficult to carried out with conviction and consistency (Anderson, Trethoman and Kenna, 1959; Lishman, 1987)

- •More usual on forensic and medicolegal conditions. (Gilandas and Touzy, 1983)
- •Conscious and intentional
- •Gross exaggeration of neuropsychologic deficits

Motivations

•Conscious

•Obtain drugs

- •Avoiding military duty / work / criminal prosecution
- •Better living conditions
- •Financial compensation
- •Adaptive behavior- simulate illness while prisoner

Tab. 5 - Factitious disorder characteristics

•More common in men

•Episodic or chronic

- •Pansymptomatic similar to other simulated dementias and with Ganser Syndrome
- •Severe personality disorder

•Motivation – psychological need to assume the role of the patient: past hospitalizations, medical procedures, surgical interventions (Munchausen Syndrome)

Tab. 6 - Hysteric disorder characteristics

Inconscious

•Intrapsychic emotional conflicts

•Personality disorder: histrionic traits, dependency and suggestibility (Wells, 1979)

•Emotional precipitants: interpersonal conflicts, p sychosexual difficulties

•Amnesia or multiple cognitive deficits

•Episodic or chronic and resistant to treatment

•Symptoms can improve with hypnosis or amobarbital interview

In rare cases, regression to a state of infantilism (hysterical puerilism or infantilism - Bleuler, 1924; Daumezon's acute regressive state: lying in bed, mutism, refuse food, incontinent, antagonic, confused)
May occur in the context of other psychiatric disorders: depression, Ganser Syndrome, dementia

Characteristics	Malingering	Factitious	Hysteric
Process	Conscious	Conscious	Inconscious
Emotional conflicts	Absent	Present	Present
Motivation	Material gain	Hospitalization	Primary gain
Past history	Anti-sociality orlitigation	Multiple hospitalizations and medical interventions	Pseudoneurological symptoms (paralysis, blindness, sensory loss) or dissociative symptoms (amnesia, fugue states)
Onset	After minor cerebral injury, material desire or frustrating circumstance	After emotional conflict	After emotional trauma, fugue or minor cerebral injury
Attitude towards symptoms	Insistent and concerned	Appear concerned until hospitalized	" La belle indifférence"
Attitude towards examiner	Evasive and on guard	May be suggestible and uncooperative	Cooperative and suggestible
Presentation	Usually not dramatic	May be dramatic	Often dramatic and histrionic
Response to confrontation	Evasive or indignant	Disbelief or anger and escape from hospital	Unconcerned, uses denial

Tab. 7 - Differential diagnosis of simulated dementias

and by many authors as the protype of pseudodementia. It is a very special and polemic entity. Their more important characteristics are reported in tab. 8.

Among psychiatric disorders which provoke severe cognitive impairment not necessarily progressive or irreversible or degenerative, depression deserves a prominent place. The term pseudodementia has been traditionally given to the cognitive dysfunction in depression.

In current clinical practice the term is many times used as a central part of depression in elderly.

In geriatric depression the cognitive symptoms are often observed: difficulties on memory, attention, concentration and mental efficiency.

Tab. 8 - Ganser syndrome characteristics

•Heterogenous disorder overlaping with the simulated dementias, schizophrenia, delirium, "true" dementia

•Symptoms: "approximate answers" (Moeli, 1888; Ganser, 1898), visual and auditory hallucinations, illusions, clouding of consciousness, "hysterical" stigmata of various kinds, transient excitement, anxiety, perplexity, amnesia for the duration of disorder

•Answers may be close to the correct ones or totally absurd, given with seriousness and at times with obsessive quality; may be interspersed with the right ones

•Duration of few days

•Acute clinical picture. Psychotic, confusional or dissociative?

•Follows head injury, infections or imprisonment. Also in depression, schizophrenia, emotional trauma, stroke, dementia, acute confusional states caused by intoxications.

•Ganser believed that the disorder was caused in part by hysteric mechanisms (dissociative state) but the organic component must be emphasized (typical cases show alteration of consciousness and amnesia)

•Organic dysfunction and psychosis may be released by hysteric mechanisms according to Ganser

•Distinct from hysteric pseudodementia because of its heterogeneity and common basis in psychosis and organicity

If these or others cognitive problems remain after complete treatment, the patients need to be carefully considered to mislead dementia.

Depressive pseudodementia or the dementia of depression (Rabins, 1983) or depressive dementia (Mchugh and Folstein, 1978; Wells, 1979; Emery, 1988) are considered by many clinicians the most prevalent disorder that can be cause of confusion with dementia.

The relationship between depression and dementia is complex. While depression may mimic dementia, it is more common for depression to be superimposed on dementias, particularly if early in their course. Difficulties in differentiating the two conditions arise because of overlapping symptoms: mild cognitive impairment, withdrawal and personality changes may occur in each.

Probably the likeliest misdiagnosis of depression as dementia is by the patient. Depressed people expect the worst. They are unforgiving about memory lapses and taken them as evidence of their senility (Kahn et al., 1975).

The depressive dementia is concerned with a major depression with cognitive deficits that reach clinical proportions. In this case cognitive deficits are secondary to depression but the differential diagnosis is so hard that it is only possible to identify depression retrospectively after treatment. Of course it is important established the age of onset of depression, family history, depressive delusions, treatment response and cognitive decline.

As we know, the depression even with early onset, is accompanied with cognitive deficiencies and it is controversial if the depressions with late onset are associated with greater cognitive impairment or equal to that early onset (Greenwald and Kramer-Ginsberg, 1988; Conwell et al., 1989; Pearlson et al., 1989). It remains that late-onset depression in older subjects is us ually as so ci at ed with mo re me di cal and neuroanatomic changes and therefore greater risk of cognitive impairment or dementia (Jacob and Levy, 1980; Alexopoulos, 1990; Rabins et al., 1991).

Some authors as Emery and Oxman (1994) believe that the classic dichotomies of irreversibility versus reversibility and structural versus functional or organic versus non organic connected with pseudodementia are questionable because some form of degenerative brain disorder can bepresent in depressive pseudodementia. Other psychiatric disorders can mimetize dementia.

Mania and hypomania, mainly in elderly causing garrulity hyperactivity simulating agitation, circumstantiality, lability of mood, self-neglected eccentricity, distractibility, disinhibition can be mistaken as dementia and delirium (Kiloh, 1961; Shiles and Cohen, 1979; Smith and Kiloh, 1981; Summers, 1983; Koenigsberg, 1984; Thase and Reynolds, 1984; Casey and Fitzgerald, 1988; Wright and Silov, 1988).

The severe mania results in disorganization of thinking and memory processes (Kiloh, 1961; Smith and Kiloh, 1981). The clinical picture appears as delirium which may be mistakenly equated with dementia. Actually, the incoherence, the poor concentration, the poor memory and physical deterioration may suggest an organic process.

We must take into consideration the family and personal histories, the symptoms development chronology, the response to treatment and definitive absence of organic factors to help the diagnosis.

The difficulty to make a correct diagnosis is aggravated by the existence of organic manic disorders secondary to central nervous system and systemic disorders.

Moreover schizophrenia may present a dementia-like pictures.

In acute episodes, the considerable concentration deficits, the disorganized behaviour and thinking, the poor judgement, the impaired memory and, in some cases, the clouding of consciousness can confuse the diagnostic.

In chronic schizophrenia, the self-neglected, the peculiar talked, the poverty of thought, the poor abstraction, the memory deficit, the erratic behaviour, and the poor insight may became difficult to distinguish from a primary degenerative dementia.

Another example of schizophrenia as pseudodementia is the buffoonery syndrome (Bleuler, 1924) in which the patient presents with a bizarre and dramatic behavior and is unable to perform very simple cognitive tasks.

The late-onset schizophrenia or paraphrenia is another aspect of confusion between schizophrenia and ps eu do de mentia. We should notice that this heterogeneous disorder includes patients who progress to dementia and inclusive brain-imaging studies (Jacoby, Levy and Bird, 1981; Flint, Rifat and Eastwood, 1991; Miller et al., 1991) suggested the presence of structural lesions consistent with an etiologic organic brain disorder in many patients with late-onset schizophrenia or paraphrenia.

The new views about schizophrenia, emphasizing the cognitive impairment supported by the evidence of organic brain abnormalities (Jacoby, Levy and Bird, 1981; Holden, 1987; Flint, Rifat and Eastwood, 1991; Miller et al., 1991) take again the Kraepelin's concept of *dementia praecox*. Can we say that there is a schizophrenia dementia type subcortical dementia?

In severe OCD, the patients may be so debilitated by the symptoms that self-neglect, deteriorate state and poor performance on cognitive tests can be the source of a mistaken diagnosis with dementia.

Similarly, a severe anxiety state can presents with memory complaints, testing poorly on examination can create the impression of dementia particularly if depersonalization occurs (Kiloh, 1961; Wells, 1979).

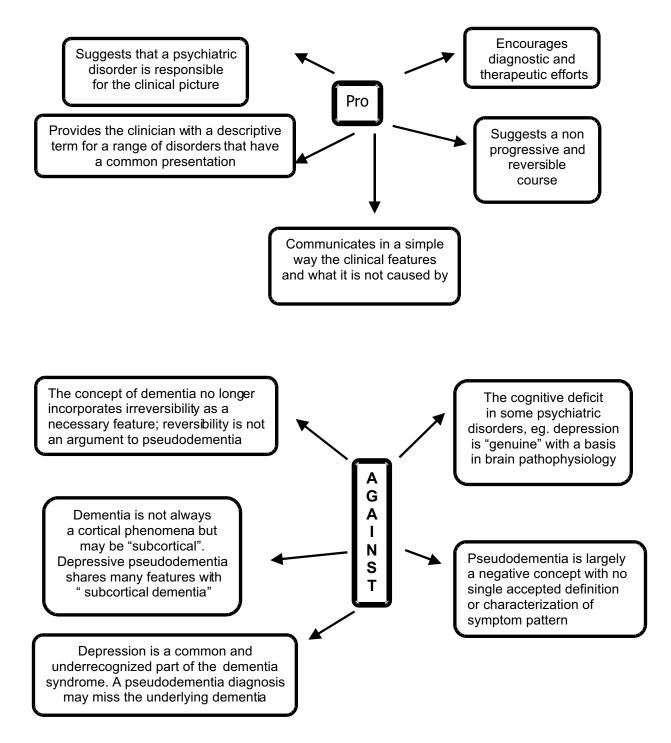
The delirium and mental retardation are examples of the major difficulty to distinguish pseudodementia from dementia.

The Ganser syndrome is a syndrome distinct from

hysteric pseudodementia because of its heterogeneity and its not uncommon basis in psychosis and organicity. The definition of Pseudodementia as well as the clinical and research usefulness of this concept have been extensively debated over the last decades beginning with the controversy overusage of the term (Reifler, 1982; Mahendra, 1984; Folstein and Rabins, 1991).

Sachdev and Kiloh (1994) have even exposed the arguments pro and against the use of the term pseudodementia (TabIX).

Tab. 9 - Pseudodementia: arguments for and against the use of the term



The extensive discussion about pseudodementia lead to numerous studies (Mairet, 1883; Newton, 1948; Roth, Tomlinson and Blessed, 1967; Wells, 1979; Mc Allister and Price, 1982; Murphy, 1983; Rabins, Merchant and Nestadt, 1984; Reding, Haycox and Blass, 1985; Bulbena and Berrios, 1986; Reifler et al., 1986; Alexopoulos et al., 1987; Kral and Emery, 1989) have shown that in many pseudodementia conditions, namely depressive, there are organic brain changes (eg. senile plaques and neurofibrillary tangles) and/or progression towards dementia. Emery and Oxman (1992) believe that pseudodementia, due to its heterogeneity, is better conceptualized as a broad category with many subtypes. These authors see pseudodementia as part of the spectrum of dementia. Because early stages are often reversible and organic degeneration appears later, they prefer avoid the term pseudodementia and use the term Transitory Dementias as it incorporates a diachronic dimension.

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