# Psychiatric intensive care of Dementia praecox

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33 year old British male's first presentation to mental health services was prompted by florid paranoid psychosis and volatile aggression. The patient developed agitated catatonia which eventually improved after 12 courses of ECT. The ongoing psychopharmacological management includes a second generation antipsychotic, a mood stabilizer antiepileptic and an anxiolytic. All investigations including blood tests, CSF analysis, urine and hair drug screen, CT and MRI scans with multidisciplinary medical consultations excluded any underlying pathology. The working diagnosis is an enduring paranoid psychosis with prominent signs of cognitive decline, all of which conclude to Kraepelin's Dementia Praecox.

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**Keywords:** Paranoid psychosis, aggression, agitated catatonia, cognitive decline, dementia praecox

## INTRODUCTION

### *History of presenting complaint and circumstances of admission*

The case of a 33 year old British Caucasian man from South East England has evolved to a complex clinical picture with diagnostic dilemma and a comprehensive multidisciplinary management.

He has been working as a paramedic for 8 years, with no records of a previous mental illness, criminal records, or illicit substance misuse. Positive family history of bipolar affective disorder (paternal grandmother) noted. He has been married for 4 years and lives with his wife and two children.

There were strains in the relationship and this has been a major source of stress in his life recently. The prodromal history of this man developing suspicions that "mould" in the family home affected him leading to physical illness. The family reported that his problems began about 6 months ago when he started to get obsessed of the belief that one of his sons also had cancer and required medical investigation. He has undertaken extensive tests via his GP, none of which indicated that there were any physical health problems. Three month prior to his first presentation to mental health services he injured his knee during martial arts training, which he was undertaking for some time, and he has been off work since then. He stayed with his parents for a few days during Christmas time, without them noticing anything. They recalled that during the last 2 months he was accusing his father of mistreating his children. During the New Year, following an argument with his wife, he went to stay with his parents. Matters escalated as he had consulted with his GP and it had been questioned whether there might be a mental health problem, possibly linked to stress.

He had left the GP surgery, and later that day had presented as aggressive towards his family. His wife felt scared about the way he presented, asked him to wait outside while she was contacting the police. He broke the front window and entered the house through the window and caused some damage to property inside. He did not assault anyone and jumped out through the window and went off down the street. Whilst outside, he knocked on a few neighbour's doors and allegedly told them that he was a "vampire". He had stripped off and was running on to the street. It is also alleged that he stopped a car on the road and threw concrete blocks and drain covers onto them. He pulled the drivers of the cars outside. When the police arrived he was seen walking into the sea. Initially he was more lucid and polite and they persuaded him to come back to the shore, then he attacked the police. As a result an officer sustained a leg fracture after the patient pushed him over when they tried to stop him walking into the sea. He was charged with grievous bodily harm and criminal damage. He was arrested and brought to the Mental Health Unit.

## DISCUSSION

### *Progress and management*

At the Mental Health Unit he became more lucid, remorseful and tearful about the incident, he said that he could not understand why he was doing it. He sat through the assessment and agreed to be admitted to hospital voluntarily. About 3 hours later, he experienced another aggressive outburst, which led to him being restrained and transferred to the Psychiatric Intensive Care Unit. Considering that he was very physically fit and healthy and was doing martial arts training, this was difficult and he was transferred to the Intensive Care Area. He continued with assaults on individuals which were unprovoked. This led to him being secluded, which continued for 72 hours. He was given medications, haloperidol and lorazepam intramuscularly. In the seclusion room he was throwing himself all around to the wall, had taken all his clothes off and appeared sexually disinhibited. He appeared to be in a mixed state with elated mood and depression. He injured himself by running into walls and has exhibited unpredictable behaviour such as somersaulting.

His symptomatology, i.e. lucid periods in between, with the background of no past psychiatric problems at the age of 33, the possibility of an organic cause was considered. It was not possible to organise any investigation or to do a full physical examination on him. On the fourth day of his admission, the medical team anaesthetised and intubated him on the Psychiatric Intensive Care Unit and had an emergency CT scan done with plain and contrast. At the same time a lumbar puncture was also done, to rule out encephalitis. The results of the scans were normal as well as that from the CSF analysis. Raised creatinine kinase was noted around 4000 IU/L about 2 days after he was admitted, but there were no signs of any Neuroleptic Malignant Syndrome. His temperature and BP were normal, but the heart rate remained in the region of 140-150 BPM. The antipsychotics were continued following consultations with the physicians and the CK came down significantly. No abnormalities were found upon neurological examination (though a detailed examination was not possible as he was not cooperative). Normal metabolic and neurological findings with raised CK were attributed to external muscle damage. Despite the negative lumbar puncture and CT (head) scan, the fluctuating nature of his presentation since admission i.e. intermittent lucid periods, still pointed to an underlying organic condition.

Initially the patient was mute and unresponsive, adopting an odd posture (lying face down, rolling on the floor), and unresponsive to communication, inconsistently responding to simple physical commands, e.g. being requested to stick out his tongue. He deliberately punched doors and walls resulting in extensive superficial bruising over various parts of his body. He has also been observed openly masturbating and urinating inappropriately. He started to show symptoms of catatonia: waxy flexibility, echolalia and echopraxia were observed. His challenging behaviour was unpredictable of nature, become more withdrawn and less communicative. He shouted random words and sometimes with non-verbal vocalised sounds which he uttered repeatedly. The most likely diagnosis was a catatonic-like presentation of an acute psychotic syndrome. In view of all these ECT was indicated.

He was transferred to the general hospital twice, once for dehydration and the second time for aspiration pneumonia. The patient presented prior to admission with neutropenia and leukocytopenia which worsened following the administration of semisodium valproate and olanzapine and further improved up to the discontinuation of the psychotropics. Haematology review concluded recurrent neutropenia possible drug induced and febrile neutropenia. He was treated with antibiotics and antivirals, although HIV screening was negative and a number of tests came out to be normal including culture of both the blood and sputum aspirate which did not reveal any growth.

He was subsequently transferred back to the psychiatric unit and ECT was proceeded. By this time his behaviour was not aggressive or violent but continued to behave in a bizarre manner. There were no periods of lucidity but he was able to recognise his family and staff members.

MRI brain scan under general anaesthesia revealed white matter hyper-intensity lesion in right frontal lobe of doubtful significance. EEG was considered of value to exclude encephalopathic process but due to non-cooperativeness and the influence of psychotropics it has been postponed. Other Causal factors excluded via CSF analysis, drug hair analysis and extensive medical investigations.

The current psychopharmacological regime includes the following per oral regular doses of an antipsychotic (risperidone 6mg once daily); benzodiazepine (Diazepam 5mg three times daily); mood stabilizer antiepileptic (Semi-Sodium Valproate 1 gram twice daily); small regular dose of an antimuscarinic (procyclidine 5mg once daily).

## CONCLUSION

### *Diagnostic considerations*

The patient tested positive for phenylcyclidine (PCP) via urine drug screen. PCP is a psychoactive drug with central nervous system depressant, stimulant, analgesic, and hallucinogenic effects (1). It's a dissociative anaesthetic (similar to ketamine). It is thought to be associated with the clinical picture of acute psychosis, confusion, agitation or even delirious states (2). Well known false-positive PCP results include dextromethorphan, diphenhydramine, doxylamine, ibuprofen, imipramine, ketamine, meperidine, mesoridazine, thioridazine, tramadol and venlafaxine (3). Drug hair analysis did not reveal evidence of cannabis, cocaine, methadone, metamphetamine, amphetamine, ketamine, LSD, mephedrone or PCP for the last 4 months.

Subacute panencephalitis has a typical clinical picture of personality and behavioural changes, followed by myoclonic seizures, paresis, dyspraxias, memory impairment, language difficulties, stupor and coma. Characteristic EEG changes and elevated CSF globulin levels as well as raised titers of measles antibodies in blood and CSF would support the clinical picture. But the most definite diagnosis is post-mortem via typical histopathological finding in brain biopsy or autopsy (4).

Frontotemporal degeneration (FTD) is a disease process that results in progressive damage to the anterior temporal and/or frontal lobes of the brain. The hallmark of FTD is a gradual, progressive decline in behaviour and/or language that often has a relatively young age at onset (mid-50s to 60s), but has been seen as early as 21. Primarily presents with personality and cognitive changes, emotional blunting or loss of empathy, disinhibition, reduced initiative, lack of motivation.

Catatonia as the initial presenting feature of panencephalitis with changing gait, abnormal movements, speech impairment, inability to walk or stand, seizures, dementia, visual disturbances, pyramidal and extrapyramidal signs (4). Behavioural disturbances have also been reported as common initial presentation. Atypical presentations have been described including isolated psychiatric manifestations.

Periods of confusion and disorientation, extreme variations of behaviour from aggression to disinhibition suggesting organic brain syndrome of frontal lobe. Fluctuations are rapid and fleeting but not clouding consciousness seen in delirium and encephalopathy. Acute florid psychosis which followed a prodrome of a few months during he developed a delusion contributing to a extreme violence; the time of course of illness has been too long for an encephalitic process, fits a psychotic disorder with strong affective component without emergence of neurological signs i.e. seizures or focal weakness. His incontinence is to be regarded as wilful/ deliberate urination.

The patient received 12 sessions of ECT under anaesthesia and muscle relaxation. He is able to answer simple questions with persistent difficulty of communicating. His behaviour is less unpredictable, but requires close supervision in a psychiatric intensive care unit. ECT and antipsychotic treatment did not achieve dramatic improvement in mental state, but stabilised orientation and confusion which might not have been expected in encephalopathy. The patient's delusional ideations are emotionally charged. Inappropriate sexual behaviour and aggression are in line with frontal lobe dysfunction, but may be driven by elevated affect.

Kraepelin described cognitive disintegration and disruption in mental functioning such as in attention, memory, and goal-directed behaviour as Dementia Praecox. He thought it's a progressively deteriorating disease with residual cognitive defect in most cases, from which no one recovered. In this case good prognostic factors are late onset, above average IQ, mood congruent psychotic symptoms, stable work record with normal premorbide personality. Clozapine with Lithium augmentation would be beneficial indicated by the patient's most likely condition: treatment resistant schizoaffective disorder.

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# A dementia praecox pszichiátriai intenzív terápiás vonzata

A 33 éves angol férfi első pszichiátriai felvételére paranoid pszichózis és következményes violens agresszió miatt került sor. A beteg agitált excitatórikus katatónia tüneti képét mutatta, mely 12 elektrokonvulzív kezelést követően megszűnt. A fenntartó pszichofarmakológiai terápia második generációs antipszichotikumból, hangulatstabilizaló antiepileptikumból és anxiolitikumból áll. Az elvégzett viszgálatok (vérkép, CSF analízis, vizelet és haj drogteszt, CT és MR koponya képalkotók, valamint széleskörű konzíliumi kivizsgálás) kizárták az organikus hátteret, így a jelenlegi diagnózis elhúzódó paranoid pszichózis markáns kognitív hanyatlással, mely legközelebb a kraepelini dementia praecoxhoz áll.

**Kulcsszavak:** paranoid pszichózis, agresszió, agitált katatónia, kognitív hanyatlás, dementia praecox