DYSPHAGIA AND CHRONIC SCHIZOPHRENIA: A CASE REPORT

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ABSTRACT
It is known that dysphagia in schizophrenia can result from acute or tardive dystonic reactions or parkinsonism as a result of neuroleptics. This case illustrates that dysphagia may be inherent in schizophrenia itself and not necessarily be due to neuroleptics.

Keywords: deglutition disorders, dystonia

INTRODUCTION
Neuroleptics are known to cause acute laryngeal-pharyngeal dystonia resulting in dyspnea, acute dysphagia to solids and liquids and asphyxiation. I would like to report the following case to illustrate that dysphagia in schizophrenia is not always due to dystonic reactions but to swallowing abnormalities that may be inherent in schizophrenia.

CASE REPORT
Mr Y, a 52-year-old Chinese man, a long term inpatient, was first admitted to psychiatric hospital in 1973 after he tried to stab himself. At that time, he suddenly stopped working, became withdrawn, started to talk and laugh to himself and had paranoid delusions. He was diagnosed to have schizophrenia. He had attempted to drink caustic soda in 1971.

Between 1973 to 1981, he had at least five oesophagoscopy with dilation of lower third oesophageal stricture. In addition, he required removal of foreign bodies from the lower end of the oesophagus. He defaulted psychiatric follow-up and was not on any psychotropic medication. According to his family, he remained quite withdrawn at home.

In 1982, he was again admitted to the general hospital with the complaint that he choked on solid food. At that time, the doctors were not able to find any structural cause and thought he had "functional dysphagia". In 1983, he had a stroke that resulted in a mild left hemiplegia.

Between 1982 and 1987, he was admitted on several occasions to the same hospital for complaints of choking on solid food. It appears that the dysphagia was either of upper oesophageal or pharyngeal origin as he did not complain of vomiting or regurgitation after eating, as one would expect of a lower oesophageal or gastric abnormalities. Repeated barium studies and oesophagoscopy revealed that the stricture at the lower end of the oesophagus was much improved with only a mild narrowing. Barium studies in 1986 did not reveal any abnormalities. The Ear-Nose-Throat surgeon was consulted and also could not find any physical cause to explain his dysphagia. He was referred to the psychiatrist who thought his dysphagia was delusional. He was prescribed trifluoperazine with no improvement of his dysphagic symptoms.

In 1987, he was brought back to the psychiatric hospital because there was a deterioration in personal hygiene and behaviour. Since then he has continuously been in hospital. His medication was changed from trifluoperazine to thioridazine with no improvement of his dysphagia. He choked whenever he tried to take any solid food and was able to tolerate a soft diet only. In March 1989, he grabbed some solid food from a fellow patient and choked and was transferred to a general hospital for aspiration pneumonia. Barium studies were repeated in February 1991 and did not reveal any structural abnormalities.

Presently, he is on a soft diet and he still continues to choke whenever he tries to take any solid food. He is blunted in affect, withdrawn and avolitional. He has a mild left hemiplegia with a mild left seventh cranial nerve palsy. There is no dysarthria and tardive dyskinesia. The gag reflex is present. There is no parkinsonism either to explain his dysphagia. He is only taking thioridazine 25 mg at night.

DISCUSSION
Hassan and Bragg used cineradiographic techniques to investigate the swallowing functions of 34 chronic schizophrenic patients. They were able to demonstrate abnormalities in the cricopharynx and the proximal oesophagus in about half of the patients taking neuroleptics as well as those not on neuroleptics. This suggests other mechanisms besides neuroleptics mediating dysphagia. Keith noticed that chronic schizophrenics swallowed with a tongue-thrust pattern that is characteristic of young children and suggested that this immature style may result from abnormal neurobehavioural development. Additional evidence that chronic schizophrenia may result from structural brain abnormalities. Craig and Richardson report impairment of the gag reflex in schizophrenics with tardive dyskinesia and who are on anticholinergics. Swallowing may be partly mediated by dopamine. Moreover it is known that dysphagia often accompanies Parkinson's disease and dopamine blocking neuroleptics induces dystonic dysphagia. Therefore it would hardly be surprising that swallowing abnormalities be present in schizophrenia in which dopamine has been implicated. The dysphagia in the patient described, does not seem to be due to neuroleptic induced dystonia as it has been chronic, non-progressive and he is still able to swallow liquids effectively. Tardive dystonia resulting from neuroleptic use is also unlikely because his dysphagic symptoms preceded the use of trifluoperazine and it did not improve when it was switched to decreasing doses of lower potency thioridazine. Neither is it due to parkinsonism, nor is it due to a bulbar palsy as it preceded the stroke. He is also unlikely to have delusional dysphagia as he choking only on solid food. Unfortunately, he did not have an oesophageal motility study which may have confirmed that he does have a defective swallowing reflex.

This case may represent a more severe form of a spectrum
of deglutition disorders that may be inherent in schizophrenia. It is possible that schizophrenic patients may have some swallowing difficulties that may not be apparent clinically but may be revealed if more sophisticated tests like oesophageal motility or manometry tests are done. Complaints of dysphagia in schizophrenics are overlooked as there has been little emphasis on them in the psychiatric literature.

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REFERENCES

11TH REGIONAL CONFERENCE OF DERMATOLOGY
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357