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ANTICHOLINERGIC TOXIN INDUCED DELIRIUM AND RARE CLINICAL SIGN OF CARPHOLOGIA, OBSERVED IN CRITICAL CARE

Neurology

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ABSTRACT

Delirium is a common presentation in Intensive care units treating neurological emergencies.

Anticholinergic toxicity [anti -Ach] is one of the causes of Delirium. Atropine, Hyoscine, scopolamine, belladonna, Deadly night shade [Mandrake], [Stinking Night Shade] Hyocimus Niger and Jimson weed or Datura seeds are anticholinergic toxins. Acute onset Delirium without any prior history of psychiatric illness, is the most common symptom of anticholinergic toxin ingestion or exposure.[1] Accidental consumption via traditional medical practioners or quacks often use crude extracts of anticholinergic drugs that lead to delirium, hallucinations, stupor, irrelevant talk and raised body temperature. Atropine, an anticholinergic drug, is also given as an antidote to Organophosphate toxicity in humans. Delirium is also seen in Atropinized patients. Central Anticholinergic syndrome CAS, has been described in Anaesthesia.[19]. Carphologia means "to behave as though one were collecting straw" First mentioned in the writings of Aristaeus. Carphologia, Crocydismus or Floccillation is described as “picking at clothes or bed linen”. It is an important clinical sign seen in agitated Delirium due to anticholinergic poisoning. In this retrospective cohort study of various forms of delirium cases, the Authors studied only anticholinergic toxicity related Delirium in 21 patients. Pre-and post-treatment recovery scores of organic deliriums were compared using DSR-98-16 item scale as a tool at onset and recovery. All patients showed dramatic complete recovery within 24 hrs of withdrawal of the drug. The study was conducted in patients to observe features of Delirium in Anticholinergic toxicity, the differences between delirium seen in Synthetic Vs natural anticholinergic extracts and to Calculate the optimum dose of Anticholinergic atropine as therapeutic agent as per bodyweight for minimising delirium & toxicity in OPC patients under critical care.

KEYWORDS

Delirium, Carphologia, Atropine, OPC poisoning, Organic brain syndromes, Neuro-Critical care

INTRODUCTION & BACKGROUND

Acute confusional state or Delirium is the presenting symptom of Toxicity due to anticholinergic drugs like Atropine, Scopolamine, Diphenhydramine, Chlorpheniramine. Toxic Plants like Deadly Nightshade: Mandragora officinarum[laxmana], Atropa Belladona [luckmanee], Datura Strammonium [Dhotara, Dhatura], Brugmansia and Hyocimus Niger [Khursani Ajwain] used in traditional medicine, can result in toxicity if not consumed under proper guidance. A common mnemonic used to describe the physiologic manifestations of anticholinergic drug overdose is: "hot as a hare, blind as a bat, dry as a bone, red as a beet, and mad as a hatter".[4] .Other symptoms include warm, dry skin from decreased sweating, blurry vision, decreased sweating/lacrimation, vasodilation, and central nervous system effects on muscarinic receptors, type M_4 and M_5. These symptoms are exclusive to anticholinergic drug use or abuse and differ from other causes of Delirium. Delirium seen in psychiatric practice or in ICU settings, Delirium and acute mania may include perplexed, fearful and labile emotions, distractible attention, jumbled speech, disinhibited behaviour, and hallucinations and delusions [6]. Presence of the sign or Carphologia, is almost exclusive to organic brain condition caused due to anticholinergic oral ingestion. Oral consumption of anticholinergic drugs leads to a set of symptoms are known as anticholinergic toxidrome[18].Table no 2 enlists the causes of acute confusional states. Before considering the diagnosis of Toxins, usual common causes should be investigated first.

Table 1 DSM classification of delirium.5 Diagnosis of Delirium.

DSM-5
A. Disturbance in attention (i.e., reduced ability to direct, focus, sustain, and shift attention) and awareness (reduced orientation to the environment).
B. The disturbance develops over a short period of time (usually hours to a few days), represents an acute change from baseline attention and awareness, and tends to fluctuate in severity during the course of a day.
C. An additional disturbance in cognition (e.g. memory deficit, disorientation, language, visuospatial ability, or perception).
D. The disturbances in Criteria A and C are not better explained by a pre-existing, established or evolving neurocognitive disorder and do not occur in the context of a severely reduced level of arousal such as coma.
E. There is evidence from the history, physical examination or laboratory findings that the disturbance is a direct physiological consequence of another medical condition, substance intoxication or withdrawal (i.e. due to a drug of abuse or to a medication), or exposure to a toxin, or is due to multiple aetiologies'.

;DSM-5, Diagnostic and Statistical Manual of Mental Disorders, fifth edition.

Physiological mechanism of Delirium in anticholinergic overdose. [14]

Acetylcholine plays an important role in consciousness, perhaps by modulating the signal-noise ratio of sensory and cognitive input and focusing awareness by activating the reticular activating system. The major pathogenic mechanism in delirium is thus often presumed to be central cholinergic deficiency. Anticholinergic drugs act as antagonists at the receptor levels. The increase in anti-acetylcholine activity is also responsible to override the normal cholinergic inhibition in areas where Ach acts as inhibitory neurotransmitter.-. [18] Some evidence suggests that endogenous anticholinergic sub- stances may exist and be of clinical importance. Action of acetylcholine is mainly on 2 types of receptors, Muscarinic & Nicotinic. The muscarinic receptors are of 5 kinds M_1, M_2, M_3, M_4, M_5. The Homunculus of the cerebral cortex representing the locomotors area has mostly M_5 type receptors, Reticular activating system has M_3 receptors. Anticholinergics like atropine, scopolamine, Hyocimus, act as antagonists at these receptor

levels. The increased locomotor activity, loss of orientation, Motor agitation and increased muscle activity seen in delirium due to Anticholinergic overdose in our study, could be correlated with areas of the brain having the Muscarinic receptors namely hippocampus, Reticular activating system and Frontal lobe. Anti Ach drugs like atropine or substances may have a major role in impairment of recall and short-term memory.[22]

Neurological & non-neurological Causes of Delirium: table no 2

Neurological causes	Non-Neurological causes	Others Mimicking Delirious states.
Cerebrovascular Strokes, Infarction, Haemorrhage, transient ischemic attack, Subarachnoid haemorrhage	Endocrine diseases: Cushing's disease, Hyperthyroidism.	Drug/ toxins- Anticholinergics/antiemetic's, antihistamine /antihypertensive, antimicrobial/ antipsychotics, antispasmodics/benzodiazepines
Infection/inflammation: Meningitis, Encephalitis Demyelinating Encephalopathy, sepsis, Tumour-Carcinomatous, Metastasis, Trauma-Sub Dural hematoma, Epilepsy postictal Temporal lobe,	Hypoxia, Hypoglycaemia, Hypo/ hypernatremia Hyperthermia, Uremia, Post-traumatic stress disorder	Systemic Lupus Erythematosus, Psychoses, Mania, Dementia, Schizophrenia,

MATERIALS AND METHODS:

An observational Longitudinal cohort study of 54 patients with acute onset delirium, admitted in an ICU of a public hospital was conducted over a period of 2 years. Records were analysed retrospectively in 2 ways **Part 1**: was diagnosis of Medical causes of delirium and application of Validated 16 item DSR-98 scale to evaluate difference between psychiatric causes and medical causes of delirium. **Part 2**: The data was then divided into 2 groups. Group 1 who were diagnosed to have anticholinergic toxicity due to non-therapeutic oral drug ingestion Vs group 2, Delirium cases who were given therapeutic antidote of intravenous anticholinergic Atropine for OPC poisoning.

The objective was to 1] study different kinds of features seen in Delirium of organic origin among ICU patients. 2] to observe differences between delirium seen in Synthetic Vs natural anticholinergic extracts. 3] Calculate the optimum dose of Anticholinergic atropine as therapeutic agent as per bodyweight for minimising delirium & toxicity in OPC patients under critical care.

Inclusion criteria were select cases of anti-cholinergic drug ingestion presenting with delirium and cases of Organophosphorus poisonings in whom atropine drug induced Delirium was diagnosed. The diagnosis of Delirium in drug consumption was done by history, neurological examination and cognitive scale of DRs R 98-16 item Validated scale as per ICU protocol. The diagnosis of delirium in ICU patients who were given atropine for Organophosphorus poisonings and went into acute delirium, was done by same scale. The protocol involved analysis of the Demographic information, History, clinical presentations of symptoms and signs, Serum cholinesterase levels to confirm anticholinesterase activity. Scores were compared for severity of delirium on day 1 & day 3. Tools used were DRS R98 16 item scale including 13 severity items & 3 diagnostic criteria. Scores were given from 0-45, higher scores indicating delirium > 15.[8] as per Trzepacz et al. (2001). Albert et al.1992. Video recording of interviews were done with consent, pre-and post-therapy. Recovery from delirium was recorded by Comparison of test scores of Drs R98 16 items scale, pre-and post-therapy or withdrawal of drug. The dose range for atropine was tabulated by adding the total doses given for successfully treating carbamate poisoning cases per/kg body weight, on discharge. Any other Drug treatment given for controlling delirium was noted. Other investigations to rule out organic brain syndromes were done with standard laboratory tests and methods namely: Blood investigations of CBC, Blood sugar, Serum creatinine, Electrolytes, Serum ammonia, RI brain and EEG. The history and Low serum pseudo-cholinesterase activity measured by standard tests confirmed diagnosis of organophosphate toxicity in whom Atropine was given as antidote.

Exclusion criteria: Children, pregnant women, Comatose patients, patients having psychiatric diseases or psychosis, patients with established neurological diseases, Comatose patients, patients who left against medical advice, ICU psychosis, other systemic diseases or metabolic brain disorders leading to delirium, Blind patients or with physical disabilities, were excluded. Adequate sample size was taken into consideration by applying 95 % confidence interval.

RESULTS AND OUTCOMES.

Primary observations were, Delirium due to anticholinergic toxicity has definitive distinct symptoms than due to delirium due to other organic brain syndromes. The prominent features are 1] Extreme motor aggression requiring restraint 2] Hallucinations of paranoia 3] Difficult recall and memory impairment after withdrawal of drug 4] Rage phenomena and abusive language. Carphologia observed 3 in patients helped to diagnose cause of acute delirium. Graph 1 shows comparative features of different types of Delirium. 95.23% Patients were males. [n=21, M=18/F=3]. Highest scores allotted was 63 for each question and lowest was 0. Highest scores were obtained for affection of Sleep-wake cycle in 95.23% patients, followed by loss of orientation in time, place and person, short term memory loss 90.22%. Loss of Visual-spatial ability, Motor retardation gained lowest 0. Sleep wake cycle affected severely total score of 63. The pre-and post-scores are indicated in columns no E & F of Table no 1. 47% patients had shown symptoms of hallucination due to anticholinergic drugs. Hallucinations decreased after treatment or withdrawal of Atropine. All patients with ingestion of natural anticholinergic showed hallucinations. Attention was severely affected in all cases. Delusions were absent in both groups. Language was undistorted in both groups but due to extreme dryness of mouth, slowing and slurring of speech, was seen. Motor agitation was observed in both groups equally and restraint was necessary. Carphologia was observed in 14% patients of Delirium. Patients of Natural toxin exposures with delirium showed reversal and recovery from agitated Delirium after treatment with average 3 doses of Iv physostigmine/Neostigmine 0.5mg/dose not more than 0.02mg/kg. Females had delayed onset of drug induced delirium as compared to males. [N=3/post 24 hrs]

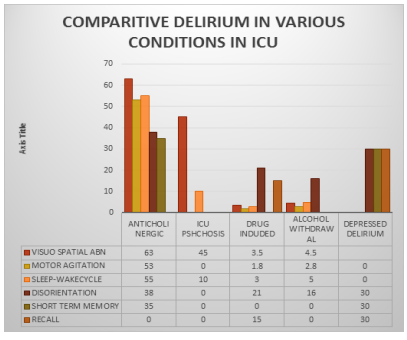
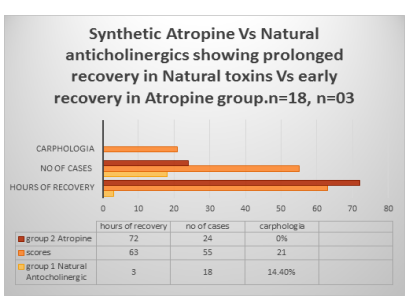
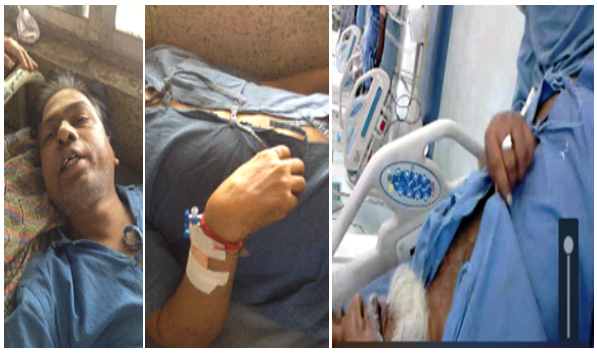


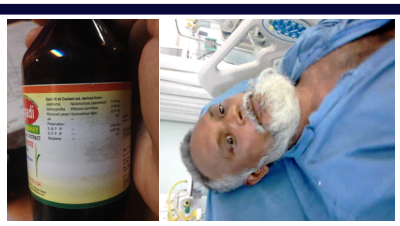
Chart 1 above showing comparative scores of different kinds of Delirium with same scale DRS R98-16point scale.

Secondary observations were, the dose range of IV Atropine required for the patient to develop psychosis in Organophosphate toxicity was as low as mg 7.2mg found in a 16year old female. Delirium in Patients administered atropine, resolved without treatment after drug was withdrawn. Mean duration of Delirium was 22.86 hrs. No residual effects recorded before discharge. Mean indoor stay till complete recovery was 9.14 days. EEG recordings were normal. Anticholinergic required in high doses coincided with low serum cholinesterase activity. Agitated Delirium was seen earlier in patients with normal cholinesterase activity. **chart 2** below shows recovery time after treatment or withdrawal of anticholinergic drugs and Carphologia seen only among patients who had delirium due to Natural anticholinergic extracts.



The range of dose of atropine required to produce delirium was_0.18 mg/kg -3. 17mg/kg.





Video 1, 2 &3, showing rare clinical sign Carphologia with Delirium.

Picture 1 –. Carphologia persistent Even after 24 hrs. Patients who had consumed natural anticholinergic and presented with delirium, recovered after treatment with neostigmine given in ICU setting. Picture 1 showing traditional drug extracts containing Hyocimus Niger taken in unsupervised manner. Some examples of unsupervised medications taken chronically

Table no 1. Documents/Delirium_drdeepabanjan.xlsx

TYPE	VISUO SPATIAL ABN	MOTOR AGITATION	SLEEP-WAKECYCLE	DISORIENTATION	SHORT TERM MEMORY	RECALL	LANGUAGE	ATTENTION	CONSCIOUSNESS			Natural toxin
ANTICHOLINERGIC	63	53	55	38	35	0	0	45	0	21	cases	3 odd cases
ICU PSHCHOSIS	45	0	10	0	0	0	20	63	52	6	cases	
DRUG INDUDED	3.5	1.8	3	21	0	15	0	63	43	18	cases	
ALCOHOL WITHDRAWAL	4.5	2.8	5	16	0	0	0	36	24	5	cases	
DEPRESSED DELIRIUM		0	0	30	30	30	30	30	30	4	cases	
										54	total	

CASE NO	MALE	FEMALE	BODY-WT	ATROPINE	HEART RATE	ONSET	DURATION	TOTAL STAY	OUTCOME	per/k/bw	serum pseudocholinesterase levels		SWC	MA	HAL	DIS	VSA	STM	LTM	ATTN	AFFECT	DEL	MR	LANGAUGE	
1	30m		51	162mg	84-122	12-14 HRS	24	5	DCHG	3.1mg/kg	306	SCRORING	3	3	0	3	3	3	3	3	0	0	0	0	0
2	38m		55	19.8mg	64-113	12-14 HRS	24	11	DCHG	0.36mg/kg	3200		3	3	0	3	3	3	0	3	0	0	0	0	0
3		16 f	40	7.2 mg	109-113	24-32HRS	24	4	DCHG	0.18mg/kg	5549	delayed	3	3	0	3	3	3	0	2	0	0	0	0	0
4	34m		51	31.2mg	80-107	12-14 HRS	24	14	DCHG	0.61mg/kg	5456		3	3	0	3	3	3	0	1	0	0	0	0	0
5	45m		68	216mg	64-109	36-48HRS	36	7	DCHG	3.17/kg	<200		3	3	2	3	3	3	0	1	0	0	0	0	0
6	57m		55	48mg	80-120	12-14 HRS	12	4	DCHG	0.87mg/kg	644.6		1	3	0	3	3	2	0	1	0	0	0	0	0
7	35m		49	84mg	80-124	20-24HRS	24	11	DCHG	1.71mg/kg	303	Delayed	0	3	0	3	3	1	1	1	0	0	0	0	0
8		30f	52	43.2mg	74-113	24-30HRS	24	9	DCHG	0.83mg/kg	5000	no aggression	3	3	2	3	3	1	3	3	3	0	0	0	0
9	38m		56	100.8mg	64-120	12-14 HRS	24	13	DCHG	1.8mg/kg	704		3	3	0	3	3	1	1	1	3	0	0	0	0
10	24m		52	32.4mg	73=103	12-14 HRS	24	12	DCHG	0.648mg/kg	2045		3	3	0	2	3	1	1	2	0	0	0	0	0
11	26m		50	52.2mg	73-120	12-14 HRS	0	16	DCHG	1.044mg/kg	2877		3	3	0	2	3	2	2	1	0	0	0	0	0
12	34m		55	41.4mg	84-133	8-12HRS	12	13	DCHG	0.75mg/kg	5644		3	3	0	2	3	1	1	2	0	0	0	0	0
13	38m		62	46.8mg	71-107	12-14 HRS	0	11	DCHG	0.73mg/kg	2838		2	3	0	2	3	3	3	2	0	0	0	0	0
14		46F	64	72mg	82-118	28-32HRS	12	8	DCHG	1.12mg/kg	4000	delayed	3	3	2	3	3	1	2	2	0	0	0	0	0
15	30m		60	39.6mg	82-110	12-14 HRS	42	9	DCHG	0.66mg/kg	2500		3	3	0	2	2	1	1	1	0	0	0	0	0
16	24m		55	58.8mg	72-113	12-14 HRS	24	12	DCHG	1.06mg	3200		3	3	0	2	2	1	1	1	0	0	0	0	0
17	52m		58	159.6mg	62-96	12-14 HRS	24	7	DCHG	2.75mg/kg	300	delayed	2	3	1	2	1	1	1	1	0	0	0	0	0
18	54m		62	NIL	75-90	12-14 HRS	6	3	DCHG	HERBAL	5560		1	3	1	1	3	2	1	1	0	0	0	0	0
19	56m		51	NIL	72-86	36-48HRS	72	13	DCHG	HERBAL	7000		3	3	1	1	3	2	1	1	0	0	0	0	0
20	68m		72	NIL	90-96	72-96HRS	36	15	DCHG	HERBAL	6500		3	3	1	3	3	2	1	1	0	0	0	0	0
21	36m		48	20.4mg	77-110	12-14 HRS	24	7	DCHG	0.42MG/KG	5560		2	3	0	2	1	1	1	1	0	0	0	0	0
TOTAL	18m	3 f	1166	KGS		12-96 HRS	480 HRS	192	days		ATROPINE=0.6MG/ML	0.6MG	53	63	10	51	55	38	25	32	3	0	0	0 mean	20.81
MEAN			55.52	KGS			22.57 HRS MEAN	9.14	Average stay in hospital		days	/ML	2.523	mean 3	2	2.4	2.3	1.8	1.1	1.66	1	0	0	0	17.76

ACUTE FIRST EPISODE

REVERSIBILITY

MORE OF MOTOR AGITAION THAN SENSORY PERCEPTION OF AFFECT OR DELUSIONAL THOUGHT PROCESS .

LANGUAGE CONTENT GRANMAR, FLOW , LOGIC , IS CONCRETE EVEN UNDER INFLUENCE OF ANTICHOLINERGIC.

ATTENTION BECOMES NORMAL TO TOUCH & NOT SOUND OR SIGHT.

SHORT TERM MEMORY AFFECTED SEVERELY.

FEMALES AFFECTED LESS

MIN DOSE OF ATROPINE= 0.18MG/KG BW-MAX DOSE = 3.17MG/KG BW

LOW PSEUDOCHOLINESTERASE ASSOCIATED WITH DELAYED INDUCTION OF DELIRIUM

PROPOSED DELIRIUM SCALE OF ONLY 5 PARAMETERS FOR ICU - ATROPINE INDUCED TOXICITY TO MODERATE DOSE OF

ATROPINE IN ICU SETTINGS.

SW CYCLES

MOTOR AGITATION

VISUO SPATIAL DISORIENTATION

LANGUAGE

High scores indicate better outcomes.

PROPOSED DELIRIUM SCALE OF ONLY 5 PARAMETERS FOR ICU - ATROPINE INDUCED TOXICITY TO MODERATE DOSE OF

ATROPINE IN ICU SETTINGS.

SW CYCLES

MOTOR AGITATION

VISUO SPATIAL DISORIENTATION

LANGUAGE

High scores indicate better outcomes.

sw=Sleep wake cycle
MA=motor agitation
VSA= Visuo spatial disturbance
ATTN= Attention
DEL= Delusios
STM= Short term memory
LTM= long term memory
HAL=Hallucination
MR=Motor retardation
DCHG=Discharge
HRS=Hours

DISCUSSION & CONCLUSIONS.

Delirium: conclusions: severe Agitated type of Delirium was seen in anticholinergic toxicity or overdose. The delirium mainly presented with disturbances of sleep-wake cycle and was followed by motor aggression, loss of short term memory, loss of orientation in time, place and person. Restraint had been required to keep patient from aggression. The route of drug exposure had not affected the delirium both IV route and oral toxicity had similar delirium. The delirium score had improved dramatically in 24 hrs after treatment or withdrawal of the offending drug. Compared to the delirium found in patients' due to psychiatric causes like dementia, schizophrenia, Mania.[12] Hallucinations were short lived and not a prominent feature of drug induced acute delirium. Carphologia was a feature seen in 14% cases of Delirium due to anticholinergic consumption. The variation in state of delirium was very prominent as indicated by the difference in DSR-98 scores. Delusions were not a part of delirium due to anticholinergic toxicity. Attention was affected severely but on arousal, focus could be obtained by observer indicating no loss of cognition. Thus, the Delirium was of an acute presentation and short dose dependent duration. The Delirium due to natural anticholinergic required IV neostigmine 0.5 mgs in 3 doses to control agitation in addition to routine drugs like Haloperidol, Benzodiazepine, Olanzapine, Risperidone, Lorazepam, to control the anticholinergic toxicity. Response to IV Neostigmine administered under ICU setting was dramatic in patients who had consumed natural extracts of anticholinergic toxins. In clinical practice, a detail diet history and specific neurological examination are important part for identifying source of intoxication or drug exposure, in a patient presenting with symptoms suggestive of acute onset abnormal behaviour. A high degree of alertness must make the physicians as well as the psychiatrists in clinical practice, suspect toxin as an aetiology in acute onset behavioural neuropsychiatric diseases. The presenting symptoms often mimic psychiatric diseases. A cumulative toxicity of naturally occurring toxins also can mimic behavioural problems. Most anticholinergic compositions are present in organic forms in plants hence unknown plants and flowers must not be handled by children to avoid accidental exposure or ingestion. The toxic plants resemble edible natural green leafy vegetables hence sorting and cleaning of leafy vegetables before cooking, is advocated. Raw leaves need be used with caution while consuming. Traditional medical systems often use crude extracts of leaves, roots and stems of biological agents and if not administered under proper instructions or if misinterpreted by consumers, can lead to acute reactive or chronic accumulative, toxicity. Accidental exposure in population can occur during outdoor activity like Gardening, farming, Forestry, Landscaping, Camping, construction & fishing. Quacks often posing as traditional healers, use crude extracts of toxic leaves, roots and dried stem concoctions and experiment on innocent victims. Diphenhydramine is used as an anti- histaminic even in children. Unsupervised administration may trigger anticholinergic toxindrome often misinterpreted as hyperactivity in young children. Since serum acetylcholine levels are very difficult to quantify with any biochemical tests, only clinical observations form the basis of treatments. Currently no tests were available with the authors to measure serum acetyl choline levels or toxin levels from biological sources. The Authors, hence emphasizes that Carphologia becomes an important sign to be observed in patients presenting with confusing states for diagnosis of anticholinergic substance abuse, exposure or ingestion. This sign is not seen in toxins affecting cerebellum, peripheral nervous systems or myoneural junctions. Carphologia was observed specifically only in delirium due to biological extracts. [n=3] Patients of artificially induced delirious states in Atropine overdose for therapeutic treatment of

Organophosphorus poisonings did not show Carphologia as a sign during the period of delirium. [n= 18] The delirium seen in both groups was of extreme 'agitated type'. Therapies for delirium include treatment of underlying cause. Cochrane reviews 2005-2009 state the standard drugs like Haloperidol, Risperidone, Quetiapine, Midazolam or lorazepam don't work in some cases .[7] Benzodiazepines, anticonvulsants have been tried in controlling agitation due to alcohol withdrawal, as per evidence in the 3 cases, uncontrolled agitation in delirium responds to therapy of Intravenous neostigmine repeated after 20 minutes and 1 hour, to control agitation requiring restraint. The treatments essentially need cardiac monitoring, restraint and an intensive care setting and should be done under expert guidance and monitoring of vital parameters. In induced delirium due to therapeutic administration of Atropine sulphate, the reduction of dose was effective in controlling delirium and no specific therapy was required to revert delirium, indicating a cumulative effect of atropine sulphate resulting in toxicity. Carphologia is a significant clue to suspect anticholinergic substance or drugs overdose when patients come with acute confusion. This sign is not seen in synthetic analogues of drugs like Atropine sulphate, commonly used as antidotes for OPC poisonings.

The study emphasizes Key learning points as 1] Delirium in ICU needs a disciplined approach, different criteria to differentiate from delirium due to other causes. 2] Over the counter sale of traditional forms of medications should be discouraged and medications should be taken under guidance of registered practioner of traditional medicines.4] Therapeutic trial of physostigmine/neostigmine can be given to control agitation in delirium suspected to be due to natural anticholinergic toxicity. Especially when Routine drugs like haloperidol, Lorazepam, Benzodiazepines or anticonvulsants, fail to control agitation. Strictly with cardiac and respiratory monitoring.

Scope & Limitations: The study helped to classify features of various types of delirium seen in ICU settings. It provides insight for correlating dose of atropine in organophosphorus poisonings, with various co factors like bodyweight, frequency of administration, Mode of administration. Carphologia, an important rare seen clinical sign, is also highlighted in this study. This sample size was not so significant but provides evidence for quantitative research. It was Time consuming and required high end audio video equipment handy when such cases are treated. Doctors need to be technologically trained to use recording equipment effectively without affecting bias or breaching confidentiality.

No conflicts of Interest or financial interest to be declared. The Authors wish to thank the Dean of the institute for the study of cases. Informed Consent of patients as well as first relatives was taken for recording and of videos and use for academic purpose. No Ethical issues or financial interests to be declared. The Authors permit use of print Resource and article for academic purpose.

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