

Clindamycin Induced Dysphagia – A Rare Concurrence

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Abstract: Although dysphagia is a mechanical impedence in phase specific mechanism, drug induced dysphagia is an adverse event often caused due to esophagitis. Clindamycin is well known to cause esophagitis; itself causing dysphagia is not reported in literature. Herein, we report one such case recently seen by us who was diagnosed with Clindamycin induced dysphagia post debridement surgery for acute necrotizing fasciitis.

Keywords: Dysphagia, Drug Induced, Clindamycin, Swallow reflex.

INTRODUCTION

Dysphagia is a biomechanical disorder defined as “an inability to swallow, or a sensation that solids or liquids do not pass easily from the mouth to the stomach” [1]. It is an uncommon adverse event of drugs that may either cause or exacerbate an existing dysphagia. Medications can induce dysphagia secondary to esophageal injury caused by esophagitis. Therapeutic modalities like aspirin, bisphosphonates, potassium chloride, quinidine, tetracycline and drugs with ferrous combinations are drugs which are well known to result in esophageal injury or impaired esophageal motility in upto 50% of cases [2]. Clindamycin is a commonly used antibiotic with Pseudo membranous colitis as its grave complication along with others. There are a few reports of Clindamycin causing esophagitis [3, 4]. However, it per se causing dysphagia is unknown. Herein we report one such case of dysphagia induced by Clindamycin.

CASE REPORT

A 40-years old male farmer presented with three days history of inability to swallow, odynophagia, aspiration with intake of solids and itchy erythematous maculopapular rashes over body. The patient did not present with any history of neurological, sensory or other systemic problems like hypertension, and diabetes mellitus. There was no previous history of esophageal disease, drug allergies, smoking and alcoholism. The patient had undergone surgical

debridement of right lower leg for Necrotizing Fasciitis and was treated with oral Clindamycin (600 mg/6th hourly) for 10 days. After six days during the course of treatment, patient noted a progressive onset of difficulty in swallowing and odynophagia on consumption of solids and presented to us on the 9th day. Apart from these, he did not have any signs and symptoms of esophagitis or extra pyramidal disturbance.

A comprehensive protocol referred to as the ‘Clinical Swallowing Evaluation template’ given by American Speech and Hearing Association (ASHA) was used for clinical examination of dysphagia in the patient. The signs and symptoms revealed pharyngeal and esophageal dysphagia in this case. A detailed diagnostic evaluation of the patient revealed, independent self feeding on mechanically soft solids, pudding, thin liquids and was observed to have a fair endurance during meals. An oro-motor, respiratory and phonatory system examination revealed normal structure and function of the speech mechanism. Repeated Saliva Swallowing Test (RSST) [5] revealed a delayed third swallow i.e. at 39 seconds which was suggestive of aspiration, Table 1. The values in the table reveal the time taken for three swallows for two repeated trials. Other associated features suggestive of dysphagia noted in this subject were xerostomia, odynophagia, effortful swallow, oral regurgitation, aspiration, delayed oral onset of swallow and presence of oral residue cleared with piece meal deglutition. The reaction time for a swallow reflex for liquids was 4.4 seconds and an oral transit time of 17 seconds.

A subjective assessment of various swallow functions was carried out, following which the patient was rated on the basis of degree of impairment by

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Table 1: Findings of Repeated Saliva Swallowing Test (RSST)

Swallow Trials	First Swallow (in seconds)	Second Swallow (in seconds)	Third Swallow (in seconds)
Trial 1	5	15	29
Trial 2	16	29	39

observing his swallowing abilities using different consistencies of the solids and fluids. The response reliability was cross checked by three repetitive trials using the same fluid or solid being tested during assessment. All the swallow parameters were rated on the basis of a 5 – point rating scale with 1 referring to normal swallow (no impairment), 2 as mild impairment, 3 as moderate impairment, 4 as severe impairment and 5 to profound impairment in swallowing. Figures 1 and 2 represents the patient characteristics during trials with different consistencies viz, semi solids, thickened and thin liquids and Solids, respectively. The patient had moderate to severe difficulty with thick and thin liquids, which can be inferred from the abnormal pharyngeal swallow reflex, cough reflex, nasal regurgitation and aspiration. On trial with solids, the patient showed moderate impairment in aspiration and severely impaired swallow functions like delayed oral onset time, oral residue and increased oral transit time. Although oral dysphagic features were observed during assessment, symptoms were less severe when compared to that of pharyngeal dysphagia. Based on these examination and clinical findings, a confirmed diagnosis of pharyngeal and esophageal dysphagia was made.

Cutaneous examination revealed erythematous maculopapular rash over trunk, extremities, palms &

soles. Mucosal examination was within normal limits. Axillary lymphadenopathy was present. A dermatology consult sought for same inferred a drug rash secondary to Clindamycin.

Consequently, patient was asked to stop Clindamycin and was suggested palliative care for management of dysphagic symptoms and tapering doses of oral corticosteroids added for the improvement of drug rash. No other alternative antibiotic was prescribed since the patient did not show any worsening of his underlying clinical condition even after stopping Clindamycin. Following this, patient also noticed a marked improvement in swallowing both solids and liquids which were a part of his regular diet, with changes starting as early as four days. The skin rash also cleared within a week.

DISCUSSION

Pill-induced esophageal injury is often under recognized or unnoticed as it is generally passed off as gastric disturbance by the patients. This may result in serious consequences like choking, aspiration pneumonia and asphyxia which might be fatal. The possibility of drug induced esophageal damage should be considered as a precipitating factor in patients with symptoms of dysphagia, aspiration, xerostomia,

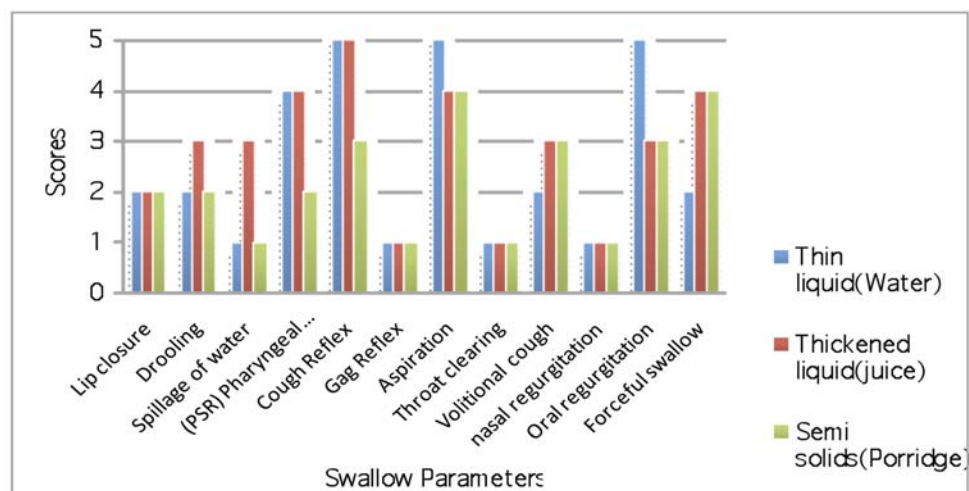


Figure 1: Patient Response Characteristics for Thin Liquid, Thickened Liquid and Semi Solids.

1-Normal (no impairment), 2-Mild impairment, 3-Moderate impairment, 4-Severe impairment, 5-Profound impairment.

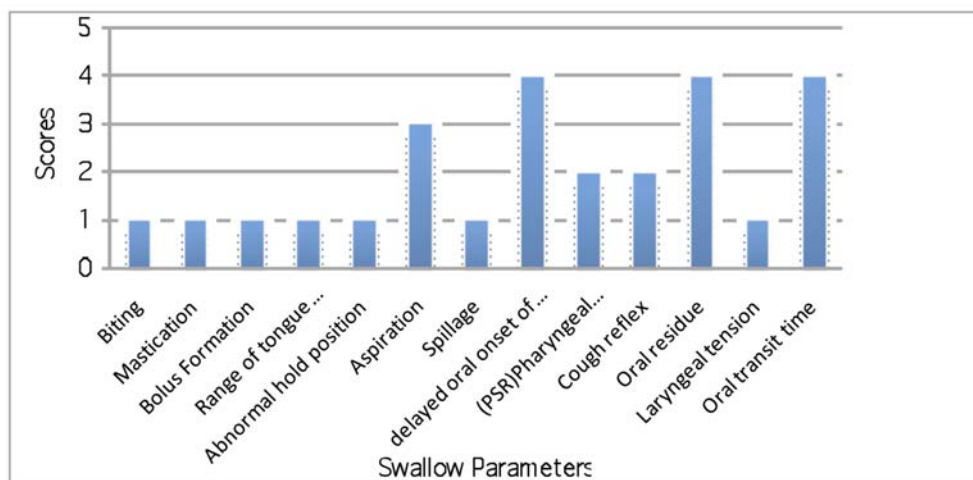


Figure 2: Patient Response Characteristics for Solids.

1-Normal (no impairment), 2-Mild impairment, 3-Moderate impairment, 4-Severe impairment, 5-Profound impairment.

odynophagia and retrosternal chest pains. Esophageal damage is possible if the patient is in a reclining position shortly after the intake of medication or because of inadequate fluid intake with the medication [6]. In both instances, the medication remains in the esophagus for too long, potentially causing esophageal damage, thus affecting swallowing.

Although Clindamycin is well known to cause grave side effects like pseudo membranous colitis, it being implicated as causing oesophageal injury like esophagitis (due to its acid containing properties) [3,4,6] is rare. The onset of dysphagic symptoms in the index case, correlated well with the intake of Clindamycin prescribed, in the absence of any other drug intake, prior to onset of illness and in the absence of signs and symptoms suggestive of extrapyramidal diseases, the findings confirms drug induced dysphagia. Subsequently, subsidence of dysphagia on stopping Clindamycin, presence of skin symptoms and dermatology consult suggestive of Clindamycin induced rash added to our suspicion as Clindamycin being the etiology agent for dysphagia. This did not warrant the usage of instrumentation to supplement our findings. Moreover, the patient was not co-operative for any probing measures even during the assessment phase due to which gastro intestinal endoscopy was not performed.

On the other hand, the most reliable and frequently used measure in the assessment of dysphagia is the bedside clinical evaluation [9]. Although clinical examination is considered to be less sensitive in identifying dysphagic features, several researchers do agree and believe that it provides valuable information

towards prognosis and management of patients with swallowing impairment [7, 8]. Mann, Lenius & Crary (2007) carried out a survey and their results revealed that clinical examination of swallowing is the primary method adopted for assessment by practicing clinicians [9]. Hence the dysphagic features diagnosed after the intake of Clindamycin in this patient based on clinical swallowing examination is reliable, as the withdrawal of the drug has proven effective in reducing the dysphagic symptoms and the palliative care showed tremendous progress in the swallowing abilities.

CONCLUSION

This report portends to exemplify yet another rarely representing side effect of Clindamycin, which is not reported to the best of the authors' knowledge. As Clindamycin is commonly prescribed in infective conditions, physicians need to be alert and cautious whenever it is being prescribed to patients. Sometimes a wholistic examination of the body would aid in diagnosis, as in this patient the presence of skin rash aided in fortifying our findings and confirming the diagnosis of Clindamycin induced dysphagia.

CONFLICT OF INTEREST

None.

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