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Case Report

A Case Report on Prednisolone Induced Acute Pancreatitis

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Abstract

Acute pancreatitis is defined as an acute inflammation of the pancreas that may also involve peripancreatic tissues and/or remote organ systems. Although the common causes of acute pancreatitis are gall stones and alcohol consumption, drug induced cases are also reported frequently. Whereas steroids induced pancreatitis cases were less and rarely reported so the true incidence is unknown, quality of the evidence is limited since the availability of studies is very less. Prednisolone is a commonly used intermediate acting corticosteroid. It is a challenge to physicians to diagnose the drug induced pancreatitis than other causes. Here in this case report we describe a 22-year-old male patient who had received prednisolone tablet for Nephrotic syndrome. According to Naranjo's ADR probability scale, the reaction was probable cause for acute pancreatitis.

Introduction

Acute Pancreatitis (AP) is an inflammatory disorder of the pancreas characterized by upper abdominal pain and pancreatic enzyme elevations [1]. Drug-induced Pancreatitis (DIP) is assumed to be a relative rare condition, and its incidence is reported between 0.1 and 2% of AP cases. However the, accurate incidence depends on the facts from clinical trials and documented case reports of DIP [2]. Drugs may be considered a possible source of the disease in patients who take medications that have been associated with AP. The majority of the data is derived from case reports, case series or summaries of them. According to World Health Organization (WHO) database 525 suspected drugs that cause AP are reported. The causality for these drugs remains indefinable and for only about 36 drugs has a definite, 10 drugs has a probable causality. Among these 36 drugs, steroids are been established as definite causality [3]. Prednisolone is a synthetic glucocorticoid, a derivative of a steroid hormone cortisol. It is indicated in Nephrotic syndrome to induce a diuresis or remission of proteinuria [4]. The risk among users of prednisolone compared to other steroids was 41% [5]. A helpful tool to establish the relation of a drug with pancreatitis could be the Naranjo Scale [6]. We describe a detail case report of predinsolone induced acute pancreatitis.

 Table 1: Treatment pattern of patient with prednisolone tablet based on the alternations of protein and creatinine levels in urine sample.

Month	Urine protein	Urine Creatinine	Protein:Creatinine ratio	Predinsolone dose
1	342mg/dl	99mg/dl	3.4	20mg OD (2tablets)
2	04mg/dl	70mg/dl	0.05	20mg OD (alternate day)
3	02mg/dl	63.3mg/dl	0.03	5mg OD
4	35mg/dl	101mg/dl	0.3	10mg OD
5	450mg/dl	109.2mg/dl	4.1	20mg OD
6	200mg/dl	69.1mg/dl	2.89	10mg BD
7	66mg/dl	51mg/dl	0.2	10mg OD
8	04mg/dl	55.5mg/dl	0.07	10mg OD (alternate day)
9	215mg/dl	89.9mg/dl	2.28	20mg OD
10	140mg/dl	60.5mg/dl	2.3	20mg OD (alternate day)
11	370mg/dl	121.3mg/dl	3.0	20mg BD
12	02mg/dl	39.8mg/dl	0.02	5mg OD
13	750mg/dl	89.8mg/dl	8.35	20mg OD (1 1/2 tablet)
14	450mg/dl	109.2mg/dl	4.1	20mg OD
15	200mg/dl	69.1mg/dl	2.89	10mg OD
16	66mg/dl	51mg/dl	1.2	10mg OD
17	02mg/dl	93mg/dl	0.02	5mg OD
18	0.6mg/dl	91mg/dl	0.06	10mg OD

BD: Twice Daily, OD: Once Daily.

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Day	Observations	Therapy
		Cefotaxime 1gm OD IV
1	C/O abdominal pain, vomitings	Pantoprazole 40mg OD IV
	O/E patient was conscious	Tramadol 50mg TID IV
	P/A-Tenderness+	IVF NS with multivitamin 500ml
	Temperature - N, BP - 150/100 mmHg, PR - 72 bpm, RR - 22 per min	DNS 500ml
		RL 500ml
2	C/O abdominal pain, fever with rigors	CST
	O/E patient was weak	Acetaminophen 150mg/ml IM Stat
	P/A-Tenderness+	Ketorolac 30mg/ml IM Stat
	Temperature - 102°F, BP - 160/100 mmHg, PR - 74 bpm	Pheniramine 10ml IM Stat
3	C/O abdominal pain, fever, nausea, vomiting	CST
	O/E patient was drowsy	Metoprolol 50mg OD PO
	P/A-Tenderness+	Metronidazole 100ml TID IV
	Temperature - 101°F, BP - 170/120 mmHg, PR - 96 bpm	Tramadol 50mg SOS
4	C/O abdominal pain	CST
	O/E patient was conscious and coherent	Stop: Cefotaxime 1gm OD IV
	P/A-Tenderness+	Ketorolac 30mg/ml IM Stat
	Temperature - 99°F, BP - 180/100 mmHg, PR - 88 bpm	
		Ofloxacin 100ml BD IV
	C/O abdominal nain loose motions	Metoprolol 50mg OD PO
		Metronidazole 100ml TID IV
5	D/A Tenderness	Lactobacillus 1tab BD PO
	P/A-Tendemess+	Racecadotril 100mg TID PO
	Temperature - 99°F, BP - 140/100 mmHg, PR - 96 bpm	Pantoprazole 40mg OD IV
		Tramadol 50mg SOS
	C/O abdominal pain (mild), loose motions	
6	O/E patient was conscious	CST
0	P/A-Normal	
	Temperature - N, BP - 160/100 mmHg, PR - 88 bpm	
7	C/O abdominal pain (mild)	
	O/E patient was conscious	CST
	P/A-Normal	Stop: IV fluids
	Temperature - N, BP - 140/90 mmHg, PR - 88 bpm	
8	C/O abdominal pain decreased	
	O/E patient was conscious	CST
	P/A- Normal	Stop: Racecadotril, Lactobacillus
	Temperature - N, BP - 130/90 mmHg, PR - 78 bpm	
9	C/O abdominal pain decreased	Ofloxacin 100ml BD IV
	O/E patient was conscious	Metoprolol 50mg OD PO
	P/A- Normal	Metronidazole 100ml TID IV
	Temperature - N, BP - 120/80 mmHg, PR - 76 bpm	Pantoprazole 40mg OD IV
	Adv. : Discharge	Tramadol 50mg SOS

Table 2: The day wise observations and treatment pattern for Acute Pancreatitis.

Adv.: Advice, BD: Twice Daily, C/O: Complaints of, CST: Continue Same Treatment, IV: Intravenously, IM: Intramuscularly, O/E: On examination, OD: Once Daily, P/A: Per Abdominal, PO: Per Orally, SOS: If needed, STAT: Immediately, TID: Thrice Daily.

Case Report

A 22-year-old male patient of 50kg weight visited gastroenterologist with abdominal pain, vomiting, symptoms with suspected Acute Pancreatitis. Continuously verifying his past medical history we came to know that the patient was on treatment for nephrotic syndrome with medication of prednisolone along with pantoprazole and multivitamin. The patient was continuously taking prednisolone tablet since 18 months, based on urine protein, creatinine protein levels and their ratio the dose was altered every month (Table 1).

During his treatment after 12 months patient came to gastroenterologist with complaints of pain in abdomen sporadically since 6 months (pain was controlled by OTC medication) with blood stools, vomiting, peritoneal discomfort, and dyspepsia since 3-4 days and patient was on prednisolone 5mg at time of visit. Physician advised to stop predinisolone for 1 month and patient observed that the pain was significantly reduced. The dechallenge of prednisolone was done after 10 days because of abnormal protein and creatinine levels in urine and patient developed abdominal pain after incessant

use of prednisolone for 6 months. Patients was presented with chief complains of severe abdominal pain radiating towards back since 3 days and 3 episodes of vomiting since 2 days at the time of recent visit to gastroenterologist.

At the time of admission after 18 months treatment for nephrotic syndrome with prednisolone patient was conscious and vital signs temperature, blood pressure, pulse rate, respiratory rate were normal on examination. His hematological report, electrolytes, liver function tests, random blood sugar levels, blood urea, serum creatinine, urine analysis was found to be normal. Serum amylase (573 U/L) and serum lipase (294.7 U/L) levels are elevated. Ultrasound report revealed acute pancreatitis, mild ascites, minimal left pleural effusion, multiple left renal cyst and this was confirmed from CT scan report which shows that acute pancreatitis, ascites, pleural effusion, right renal cortical cyst, left renal calculus (Table 2).

The medication prescribed on discharge was Tramadol 50mg BD PO, Pantoprazole 40mg OD PO, multivitamin tablet OD, and patient was counseled about lifestyle changes which includes intake of balanced diet with fresh fruits and vegetables, avoid meat and do regular physical exercises to maintain weight.

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Conclusion

Drug Induced Pancreatitis was very uncommon cause but should be considered when other reasonable causes of pancreatitis like gall stones and alcohol consumption are not present. In the present case report the patient was non alcoholic without any complaints of gall stones but the symptoms and diagnostic tests showed that the patient was diagnosed with Acute Pancreatitis. However the DIP are rare, we recommend routine monitoring of abdominal pain which are the golden standard predictors for acute pancreatitis and dose adjustments of corticosteroids is recommended for avoiding pancreatitis. Further we conclude that the prednisolone may be a cause for DIP. The BISAP (Bedside Index of Severity in Acute Pancreatitis) score 3 and Pain score 7 are evaluated which are used to know the severity of disease and pain respectively were moderate. It concludes that the continuous intake of prednisolone may cause acute pancreatitis.

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