

Original Research Article

Profile of Tropical Chronic Pancreatitis (TCP): A Study of 16 YearsDr. Sarat Chandra Jayasingh¹, Dr. Chitta Ranjan Sarangi², Dr. Satya Prasad Samantray³, Dr. Pradip Kumar Mallick⁴, Prof. Mihir Kumar Mohapatra⁵¹Assistant Professor, ²Associate Professor, ³Assistant Professor, ⁴Assistant Surgeon, ⁵Professor & Head
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Abstract: A study had been conducted on 454 patients of Tropical Chronic Pancreatitis within a period of 16 years (from 2000 to 2016) in the department of Surgical Gastroenterology of SCB Medical College and Hospital, Cuttack (Odisha). The contents of the scientific article reflect the outcomes of our study in relation to epidemiology, clinical features, investigations and surgical approach which are helpful in the management of TCP and its further evaluation.

Keywords: Tropical Chronic Pancreatitis, Gastroenterology

INTRODUCTION

Tropical Chronic Pancreatitis (TCP) is a form of chronic pancreatitis of uncertain etiology seen mostly in developing countries of tropics affecting young malnourished individuals characterized by pancreatic calculi with ductal dilatation, abdominal pain and defective digestion. It pursues a long and progressive course to finally develop Diabetes Mellitus which is then termed as Fibro - calculus Pancreatic Diabetes (FCPD) [1].

TCP has been described by a variety of names like Chronic Calcific Pancreatitis of Tropics (CCPT), Idiopathic Chronic Calcific Pancreatitis, Non - alcoholic Tropical Pancreatitis, Nutritional Pancreatitis and Juvenile Tropical Pancreatitis Syndrome. This condition was first reported from Indonesia in 1959 by Zuidema and subsequently by others from different tropical countries of the world. The largest series being described by Geevarghese from South India, it is also seen in other parts of India [2].

MATERIALS AND METHODS

All the patients included in our study were subjected for routine haematological and biochemical tests like DC, TWBC, HB%, PCV, FBS, 2-hour PGBS, Serum Amylase, Lipase, Urea Creatinine, Calcium and LFT along with imaging like plain X- Ray and ultra sonography of abdomen and pelvis.

Clinical Profile

Although etiology of TCP remain obscure, certain features are quite important in its creation as well as progression.

RESULTS

In our study males predominate (78.2 %) over females (21.8%) and maximum patients (57%) come under the age group of 21 years to 40 years. About 42 % patients are found between the age of 11 years to 30 years. (table-1).

Table 1: Distribution of cases as per age group

Age(Yrs)	Number of Patients	Percentage
<10	4	1
11-20	55	12
21-30	136	30
31-40	123	27
41-50	77	17
51-60	45	10
>60	14	3

There is a delay of average 6 months to 3 years for diagnosis of TCP after initiation of abdominal symptoms as revealed from the study. Typical onset of symptoms of TCP at young age as described in the literature is not seen in our series. Table no. 2 Reveals different clinical features of our series in which

abdominal pain is the commonest complaint in 98 % of patients which on many occasions becomes quite difficult to cure. Moreover, presence of abdominal pain in TCP reflects disease activity in general.

Table-2: Different clinical features of our series

Symptoms	Number of patients	Percentage
Pain	445	98
Diabetes	50	11
Steatorrhoea	9	2
Jaundice	18	4
Pseudocyst	22	5
Ascites	4	1
Malignancy	13	3

DISCUSSION

Malnutrition:

Protein calorie malnutrition is incriminated as the causative factor of TCP. But, there are no convincing data to support malnutrition because hematological parameters like hemoglobin, PCV, Serum Protein, Serum Albumin, AG ratio and BMI are within normal limits at the time of presentation of patients to our department. Malnutrition might be secondary to less intake of food due to abdominal pain and malabsorption of nutrients resulting from exocrine pancreatic deficiency. Hence, it is not the cause of TCP but should be considered as its effect [3]. However, Tripathy *et al.*; from Odisha reported that 85 % of FCPD patients belong to poor socio-economic status which suggests poor calorie intake and debility in such patients [4].

Alcohol:

Only 7.92 % of cases of our series give history of alcohol intake, but not on regular basis. This occasional intake of alcohol associated with chronic pancreatitis should not be confused with Alcoholic Pancreatitis.

Geographical Distribution:

Most of the cases of TCP in our study belong to costal districts like Cuttack, Kendrapara, Jagatsinghpur, Jajpur, Khurda, Puri, Bhadrak, Balasore, and Nayagarh of the state of Odisha [5].

It signifies the influence of environmental factors in the causation of TCP. Most probably the environmental factors trigger genetic expression for developing TCP. Therefore further inquiry should be made as to the role of genetic factors (especially SPINK- 1) in all cases of TCP. Typically the patients of TCP present with

abdominal pain, malnutrition, emaciation, indigestion (steatorrhoea) , Jaundice and pancreatic mass.

Diabetes mellitus:

Patients of TCP develop diabetes during the course of the disease. These cases are termed as FCPD although previously classified as MRDM (Malnutrition Related Diabetes Mellitus) by WHO (World Health Organization) [6]. Patients of TCP with calcifications are more prone to develop diabetes. Those diabetic patients invariably require insulin for control but characteristically they are resistant to ketosis on withdrawal of insulin.

In our series, impaired GTT (Glucose Tolerance Test) is found in 10 % and Diabetes mellitus in 11.01 % of cases. It may be due to the fact that the patients in our series have presented early in the course of disease and the destruction of B- cells of pancreas has not occurred significantly.

Malignancy:

The patients of TCP are highly susceptible to develop pancreatic carcinoma. In a study, it is mentioned that they are at least eight times more prone than controls to develop pancreatic carcinoma. In our series development of carcinoma is detected in 3 % cases by clinical as well as ultra sonological examinations. In doubtful cases of mass lesion in pancreas (detected by USG) malignancy has been excluded / confirmed by diagnostic laparoscopy and biopsy. Hence, it may be opined that development of malignancy in TCP is not significant in our state and mass lesion in established cases of TCP require surgical intervention.

Imaging Profile

X- Ray abdomen:

It reveals radio-opaque calculi in the pancreatic region of upper abdomen on AP view. It also excludes renal calculi on lateral / oblique view. Presence of more calcifications in head region helps for planning of surgical operation (i.e. head coring etc.) if required for the disease condition.

Ultrasound (USG):

It helps in measuring the size of pancreas, its echo-texture, site, size and number of calculi, pancreatic duct morphology, mass lesion and conditions of surrounding structures (like GB / CBD calculi), presence of ascites and pseudocyst etc. Ultrasound helps in follow-up of patients of TCP to detect small mass lesion and to determine the diameter of PD (Pancreatic Duct).

In our series all cases were subjected to ultrasonogram and X – ray of abdomen. Detection of calculi by USG is more than 90 % in comparison to X-Ray. Hence, in our opinion ultrasonogram is an essential tool for diagnosis, planning for surgery and follow- up. CECT and MRI are needed when TCP is associated with complications, mass lesion and to exclude other conditions when doubt arises.

Surgery

Surgery in TCP is individualized. Since the causative factor of TCP is unknown, the role of surgery is only palliative. However, in our experience, Longitudinal Pancreatico Jejunostomy with Pancreatic Head coring (Frey's procedure) serves as the best palliative measure for long term relief of pain (93 %) with minimal morbidity (3.6 %) in patients of TCP requiring surgery [7].

But, it is to be remembered that surgery is not the first line of therapy in TCP. Hence all patients need to be adequately evaluated and treated by medical means prior to surgery. Mere presence of pancreatic calculi is not the indication for surgery in TCP. Surgery does not cure Diabetes Mellitus in TCP but it modifies glycaemic status by reducing ductal pressure, cellular oedema, inflammatory congestions which in turn improve B-cell functions. So insulin demand is reduced following surgery for few months in some cases but it again goes up as revealed from our study.

CONCLUSION

TCP behaves differently in different geographical areas. Aetiology of TCP although unknown requires genetic evaluation to establish the cause. Diabetic state does not improve much following surgery for TCP or may even become worse in subsequent years. Pain management is crucial. No modality of treatment like medicinal or surgical or therapeutic endoscopy relieves pain permanently. Recurrence of pain after adequate surgery needs thorough evaluation. TCP with pancreatic mass invariably requires surgery (like Whipple's procedure)

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