

Exocrine Pancreatic Function in Familial Chylomicronaemia Syndrome: A Narrative Review

Ekitumi Robert Ofagbor^{1*}, Paul Okediji², Temitope Toluse Selowo³ and Olatunde Olayanju⁴

¹Consultant Chemical Pathologist, Royal Free London, NHS Foundation Trust, United Kingdom

²Public Health Specialty Registrar, National Institute for Health and Care Excellence, London, United Kingdom

³IMT core trainee, Queen Elizabeth Hospital, King's Lynn, Norfolk, United Kingdom

⁴Consultant Chemical Pathologist, Ben Carson College of Health and Medical Science, Babcock University, Ilisan, Nigeria

***Corresponding author:** Ekitumi Robert Ofagbor, Consultant Chemical Pathologist, Royal Free London, NHS Foundation Trust, United Kingdom

ARTICLE INFO

Received:  August 26, 2026

Published:  September 02, 2026

Citation: Ekitumi Robert Ofagbor, Paul Okediji, Temitope Toluse Selowo and Olatunde Olayanju. Exocrine Pancreatic Function in Familial Chylomicronaemia Syndrome: A Narrative Review. Biomed J Sci & Tech Res 66(4)-2026. BJSTR. MS.ID.010369.

ABSTRACT

Familial chylomicronaemia syndrome (FCS) is a rare autosomal recessive disorder characterised by severe hypertriglyceridaemia which may present with recurrent episodes of hypertriglyceridaemia-induced acute pancreatitis, which can progress to recurrent acute and chronic pancreatitis. While the association between FCS and pancreatitis is well established, the impact of repeated pancreatic inflammation on exocrine pancreatic function remains poorly understood in these patients. This narrative review aimed to synthesise the available evidence on exocrine pancreatic insufficiency (EPI) in patients with FCS. A structured two-phase literature search was conducted across MEDLINE, EMBASE, and the NHS Knowledge and Library Hub.

No studies were found that directly evaluated EPI in FCS patients, highlighting a significant gap in the literature. The literature however consistently demonstrated that FCS is associated with recurrent acute pancreatitis and progression to chronic pancreatitis which are established causes of EPI. EPI may be under-recognised in FCS because gastrointestinal symptoms overlap with those of FCS. Potential consequences of undiagnosed EPI include malnutrition, vitamin deficiencies, sarcopenia and reduced quality of life. As EPI is treatable, the absence of evidence and lack of routine screening represent an important unmet clinical need. In conclusion, EPI is a biologically plausible but underacknowledged complication of FCS. There is an urgent need for prospective studies and incorporation of pancreatic exocrine assessment into FCS care pathways to address this unmet need.

Abbreviations: FCS: Familial Chylomicronemia Syndrome; LPL: Lipoprotein Lipase; VLDL: Very-Low-Density Lipoprotein; EPI: Exocrine Pancreatic Insufficiency; FFA: Free Fatty Acids; EPI: Exocrine Pancreatic Insufficiency; PERT: Pancreatic Enzyme Replacement Therapy; s-MRCP: Secretin-Enhanced Magnetic Resonance Cholangiopancreatography

Introduction

Familial chylomicronemia syndrome (FCS) is a rare Mendelian autosomal recessive disorder characterized by extreme and sustained hypertriglyceridemia [1] affecting approximately 1 to 10 individuals per million [2]. The condition is associated with a high risk of recurrent triglyceride induced acute pancreatitis [3]. FCS commonly results from profound reduction of lipoprotein lipase (LPL) activity (60% - 80% of cases) but could also result from defects in other related genes [1]. Individuals with FCS experience fasting chylomicronaemia i.e. severe hypertriglyceridaemia which could be intermittent

and persists after fasting [1]. Abnormalities in catabolism of other triglyceride rich lipid sub-fractions like very-low-density lipoprotein (VLDL) also contribute to the severe hypertriglyceridemia seen, but accumulation of chylomicrons represents the most distinctive characteristic with triglyceride level on a fasting blood sample often >10mmol/l (≥885 mg/dL). In addition to the milky appearance of plasma or serum, other observable features like eruptive xanthoma, abdominal pain, hepatosplenomegaly, and lipaemia retinalis may be recorded [2]. Recurrent episodes of acute pancreatitis defined as two or more episodes of acute pancreatitis with a resolution of symptoms between episodes predispose patients to chronic pancreatitis with 35

in 100 patients progressing to chronic pancreatitis [4] with potential declining exocrine pancreatic function. The assessment of pancreatic exocrine function may therefore be critical for optimal care of these patients. However, this assessment is not routinely done as guidelines for their care do not recommend it despite this potential for exocrine pancreatic insufficiency (EPI) in them [1,2]. EPI is a clinical syndrome resulting from failure of the pancreas to deliver sufficient digestive enzymes, particularly lipase to the small intestine, leading to impaired digestion and absorption of nutrients, especially fats and fat-soluble vitamins [5]. EPI is most associated with chronic pancreatitis but also occurs in up to 60.5% of patients following severe acute pancreatitis, with occurrence in up to a third of patients on long term follow up [6]. Exocrine pancreatic disorder symptoms are non-specific requiring a high index of suspicion for accurate diagnosis and timely intervention with enzyme replacement therapy [5]. The clinical consequences of EPI extend beyond gastrointestinal symptoms and can substantially affect long-term health and quality of life [4] and faecal elastase testing is recommended for diagnosis of the condition [6]. The objective of this narrative review is to critically examine the current evidence on EPI in patients with FCS, including its epidemiology, pathophysiology, clinical manifestations, diagnosis, and management. It also highlights existing knowledge gaps and identifies priorities for future research and clinical practice.

Methods

A comprehensive, two-phase literature search was conducted across MEDLINE (PubMed), EMBASE, and the NHS Knowledge and Library Hub for this narrative review. An initial search of the databases from inception to September, 2026 using key search concepts including “familial chylomicronaemia syndrome”, “faecal elastase”, and “exocrine pancreatic function”, retrieved no studies that directly addressed EPI in patients with confirmed FCS. A second broadened search was then conducted over the same period. Studies reported in English were eligible if they included patients with FCS and reported any measure of exocrine pancreatic function. Given the rarity of the condition, observational studies, case series, and case reports were all considered eligible. Full texts of potentially relevant records were subsequently retrieved and assessed against the predefined inclusion criteria - defined as studies directly measuring or reporting exocrine pancreatic function in patients with FCS. No studies meeting the inclusion criteria were identified from either phase of the search. The absence of eligible primary literature is itself reported as a principal finding of this review, providing direct and unambiguous evidence of an unaddressed gap in the clinical and research literature. A narrative synthesis was performed to explore themes related to the association between EPI and FCS. Expert opinion, clinical guideline recommendations, and pathophysiological reasoning were incorporated, enabling a nuanced exploration of the subject.

Review

FCS and Pancreatitis

The most serious complication of FCS is acute pancreatitis, which is often recurrent and can lead to the sequelae of chronic pancreatitis [3]. Many FCS patients experience 1 episode of acute pancreatitis every 3-5 years [1]. Although, the precise mechanism by which triglyceride mediated pancreatitis occurs and progresses remains unclear, but it has been hypothesized that high levels of serum triglyceride result in triglyceride-induced hyperviscosity from large lipoprotein chylomicron particles which impair pancreatic capillary blood flow [1]. This vascular congestion can lead to ischaemia, necrosis, apoptosis and acidosis, triggering the ectopic release of pancreatic lipase [5]. Another theory is based on the activity of lipase from the exocrine pancreas which liberates free fatty acids (FFA) by triglyceride hydrolysis. The released FFA is bound to albumin but once this binding capacity is exceeded, adverse consequences like mitochondrial toxicity, oxidative stress, endothelial dysregulation, inflammation and acinar cell damage can be seen [7]. Seminal studies by Saharia et al showed that perfusion of an ex vivo-isolated pancreas with triglyceride results in an oedematous pancreas with accompanying hemorrhage [8]. Additionally, it has been demonstrated that triglyceride administration into the pancreas perfusate causes elevation of serum amylase and lipase levels depending on the triglyceride dose indicating that acute pancreatitis can be induced by hypertriglyceridaemia [9]. The action of pancreatic lipases in pancreatic capillaries is also thought to contribute to acute pancreatic damage due to the local liberation of pro-inflammatory lysolecithin and free fatty acids by the hydrolysis of lecithin and triglyceride from triglyceride-rich lipoproteins [2]. Furthermore, the liberated lipase hydrolyzes triglyceride within the pancreatic vascular system producing toxic-free radicals that can directly damage acinar cells, leading to a vicious cycle of inflammation, cell death, and, eventually, acute pancreatitis [1].

The pancreatitis risk seen in patients with FCS begins to increase when triglyceride is >10 mmol/L (>885 mg/dL) and sharply increases when triglyceride is greater than 20 mmol/L (>1770 mg/dL). Sixty to ninety percent of patients with FCS experience acute pancreatitis in their lifetime [1] as they have a 360-fold greater risk of acute pancreatitis compared to the general population [3]. These patients also report a median of 34-lifetime episodes of acute pancreatitis and recurrent episodes of acute pancreatitis can lead to chronic pancreatitis, pancreatic pseudocysts, pancreatic abscesses, pancreatogenic (Type 3c) diabetes mellitus, and exocrine pancreatic insufficiency [3]. These incidents of recurrent pancreatitis in FCS patients could result in 30 workdays missed yearly compared with 3.5 workdays missed yearly for other individuals [1]. In recognition of this risk, the principal goal of treatment for individuals with FCS is to reduce pancreatitis risk [2] primarily by controlling triglyceride level. Multiple studies, including

systematic reviews have established a link between EPI occurrence and pancreatitis [4], but pancreatic exocrine function is not regularly assessed in people with FCS who have a high risk of pancreatitis. This means that unlike other conditions e.g. cystic fibrosis [10] where EPI is screened for and pancreatic enzyme replacement therapy prescribed, screening and management of EPI for people with FCS is not currently recommended.

Clinical Course of Hypertriglyceridemia Induced Acute Pancreatitis

Severe hypertriglyceridaemia not only precipitates acute pancreatitis but may also worsen the disease course in acute pancreatitis from other causes like gallstones and alcohol consumption [9]. This is because elevated serum triglyceride concentrations are associated with an increased risk of organ failure and greater disease severity in acute pancreatitis. Triglyceride levels exceeding 2 mmol/L at 3-4 days after admission have been identified as an independent predictor of severe acute pancreatitis, with a stepwise increase in the incidence of severe disease observed as triglyceride concentrations rise [11]. Hypertriglyceridaemia induced pancreatitis results in more complications than alcoholic pancreatitis [12] with respiratory distress syndrome, acute kidney injury, deep venous thrombosis, and multiple organ dysfunction seen more frequently in the severe hypertriglyceridaemia pancreatitis group than in those with biliary pancreatitis [9]. These observations underscore the prognostic significance of hypertriglyceridaemia in acute pancreatitis, as higher triglyceride level is consistently associated with an increased risk of pulmonary failure and intensive care unit admission, regardless of disease aetiology [9]. Notwithstanding the increased risk of complications and severity of acute pancreatitis in hypertriglyceridaemia [7], there remains a divergence of opinions on the impact of severe hypertriglyceridaemia on disease course and mortality in acute pancreatitis including in patients with FCS.

Exocrine pancreatic insufficiency (EPI)

EPI is a clinical syndrome caused by reduced or inappropriate secretion or activity of pancreatic juice, its digestive enzymes and bicarbonate. This leads to impaired digestion of fats, proteins, and carbohydrates [5]. The condition arises when pancreatic enzyme output falls below the threshold required for effective intraluminal digestion, typically due to structural or functional pancreatic disease [13]. Subsequently, these patients can develop diabetes mellitus when α and β cell mass is critically depleted from repetitive pancreatic injury. EPI is therefore not just a preceding event, but it is a component of the diagnostic triad for Type 3c diabetes mellitus proposed by Ewald and Bretzel [14]. In addition to chronic pancreatitis, EPI can also be seen in post-pancreatic surgery states, and in metabolic or systemic diseases affecting pancreatic function [15]. Over 50% of patients with chronic pancreatitis develop EPI with an increasing risk depending on the disease's duration. In addition, chronic alcohol use, smoking,

pancreatic ductal obstruction, islet cell atrophy, duct calcifications, and diabetes mellitus increase the likelihood of EPI in chronic pancreatitis with the risk of EPI exceeding 80% in those with these additional risk factors. EPI typically occurs after 5-10 years of the disease and EPI pooled prevalence during follow-up ranges from 27% to 62% [16]. If untreated, EPI significantly impairs quality of life, making early recognition and treatment clinically important [4].

Rationale for Exocrine Pancreatic Insufficiency (EPI) in FCS

There is a strong clinical rationale for actively investigating EPI in patients with FCS, rather than assuming gastrointestinal symptoms are dietary or nonspecific. The rationale is pathophysiological, clinical, and therapeutic for these patients.

Pathophysiologic Rationale

Kessler et al's study evaluating the experience of pancreatitis in patients with hypertriglyceridaemia reported a mean of 9 (Range 1 - 30) lifetime episodes of pancreatitis in them [17]. These repeated episodes of pancreatitis from extreme chylomicronaemia could result in chronic pancreatitis which is a progressive fibroinflammatory condition characterised by acinar cell damage, pancreatic necrosis, fibrosis and declining pancreatic endocrine and exocrine function [18]. The most common cause of EPI in pancreatitis is due to loss of functioning pancreatic parenchyma with decreased secretion which could also result from pancreatic ductal obstruction, reduced endogenous stimulation and postprandial asynchrony [6]. This reduction in pancreatic exocrine function from chronic pancreatic damage as well as acute and recurrent acute pancreatitis can result in EPI which may present with clinical symptoms [19]. EPI is reported in 94% of patients with chronic pancreatitis within 10 years of disease onset [6]. The clinical symptoms that are commonly seen in FCS like steatorrhea, malabsorption, fat-soluble vitamin deficiency, etc. [20] overlap with EPI symptoms and can be misattributed to dietary fat restriction, chronic illness, or "functional" symptoms. This uncertainty about the symptoms means that without targeted investigations (e.g. faecal elastase), EPI is easily under-diagnosed in FCS [6]. This tendency for underdiagnosis of EPI in FCS is consistent with broader gastroenterology experience that EPI diagnosis is frequently missed in high-risk pancreatic conditions [5,15].

Clinical Rationale

Nutritional vulnerability from EPI is amplified in FCS as these patients are already at high nutritional risk due to the prescribed severely restricted fat intake (often <10-15 g/day), reliance on specialised diets and supplements and risks of fat-soluble vitamin deficiency (A, D, E, K) from reduced dietary intake [1]. Undiagnosed EPI increases this nutritional vulnerability in FCS patients with a heightened risk of protein-calorie malnutrition, micronutrient deficiencies and sarcopenia all resulting from impaired pancreatic enzyme activi-

ty and a resultant reduction in health-related quality of life [16,21]. In severe EPI, pancreatic enzyme production falls below 10% of normal capacity and patients develop clinically significant malabsorption and malnutrition [6]. Assessment of pancreatic exocrine function in FCS patients is therefore important to confirm pancreatic sufficiency, determine the severity of pancreatic enzyme deficiency if present, identify nutritional consequences, and guide enzyme replacement therapy for the pancreas [5]. Clinical evaluation of the patient should also include assessment of nutritional status, body mass index, fat-soluble vitamin deficiency, and other biochemical markers of malabsorption.

Therapeutic Rationale

EPI when diagnosed is treatable and can be effectively treated with pancreatic enzyme replacement therapy (PERT). Patients receiving PERT maintain global health status and PERT has been shown to improve gastrointestinal symptoms and digestion in patients with pancreatitis. PERT has also been shown to improve fat and nitrogen absorption, nutritional parameters and GI symptoms [6]. FCS patients with EPI may therefore also experience these improvements from PERT use. It could also aid weight, nutritional stability and reduce the requirement for ultra-low-fat diets [6]. Failure to recognise EPI on the other hand may represent a missed opportunity to address a modifiable contributor to morbidity in patients with FCS.

Clinical trials of new triglyceride-lowering therapies like Apolipoprotein C-III inhibitors [22] and Fibroblast growth factor-21 [23] demonstrate a substantial reduction in triglyceride level in FCS patients and longer-term studies are ongoing to establish a reduction in pancreatitis risk [2]. As survival for these patients improve, the diagnosis and management of long-term pancreatic sequelae such as EPI becomes more clinically relevant in FCS.

Nutritional and Metabolic Consequences of EPI in FCS

The hallmark consequence of EPI is fat malabsorption, as lipase deficiency is usually the earliest and most clinically relevant enzymatic deficit [16]. In this present review, no research evaluating the prevalence of EPI in FCS patients and the nutritional and metabolic consequences that can be seen was found. Hence, the relation between nutrient deficiency and EPI remains inconclusive in FCS patients. However, deficiencies of fat-soluble vitamins A, D, E and K are frequently demonstrated in patients with EPI and screening for this is now part of the standard diagnostic work-up for EPI [15]. This lipid soluble vitamin screening is already part of the recommendations by the National lipid association [1]. In a meta-analysis of twelve studies including 548 patients with CP, the pooled prevalence rates for vitamin A, D and E deficiency were reported as 16.8% (95% CI 6.9–35.7), 57.6% (95% CI 43.9–70.4) and 29.2% (95% CI 8.6–64.5) respectively. Levels of magnesium, pre-albumin and retinol-binding protein were also found to be below the population reference values [15].

The persistent malabsorption state also leads to calorie deficiency resulting in protein-calorie malnutrition, sarcopenia, frailty, impaired immune, declining muscle bulk and reduced neuromuscular function [15]. Metabolically, EPI can coexist with or exacerbate pancreatogenic (type 3c) diabetes mellitus [24], further complicating nutritional management and increasing morbidity. The consequences of EPI are known to be systemic with long-term widespread metabolic and nutritional implications [13] and these consequences may also be seen in FCS patients.

Challenges of EPI Diagnosis in FCS

The medical consequence of EPI is significant, but the overlap of its symptoms with those seen in functional gastrointestinal disorders, and the nonspecific complications of chronic illness results in a diagnostic conundrum [22]. This diagnostic challenge is amplified in rare diseases like FCS, where baseline gastrointestinal symptoms are present [7] and may be incorrectly attributed to the underlying condition rather than pancreatic dysfunction. The most widely recommended first-line investigation for EPI is faecal elastase. This is a non-invasive test that reflects pancreatic enzyme secretion. The test is limited as values between 100-200 µg/g are indeterminate, and watery stools can cause falsely low results. A faecal elastase value < 100 µg/g is consistent with a diagnosis of EPI but is also not sensitive enough to reliably detect mild or early EPI [16]. The sensitivity of faecal elastase for EPI diagnosis is dependent on a significant decline in pancreatic elastase secretion which may not be seen in early-stage chronic pancreatitis patients. Other alternative tests for EPI include indirect tests like coefficient of fat absorption and ¹³C-labelled mixed triglyceride breath test as well as invasive direct pancreatic function tests such as the aspiration of duodenal fluid following hormonal stimulation with secretin, cholecystokinin, or cerulein [19]. Imaging studies like secretin-enhanced magnetic resonance cholangiopancreatography (s-MRCP) can qualitatively and quantitatively assess pancreatic exocrine fluid secretion but its use is limited due to the lack of availability of secretin in many countries [25]. Overall, these alternative tests are not used in routine clinical practice although they are used in controlled clinical studies [16], furthermore data on their use in FCS patients is sparse. In FCS and other rare diseases such as cystic fibrosis, or inherited pancreatitis, the low clinical index of suspicion from medics in addition to these limitations from faecal elastase testing can delay diagnosis. The diagnostic role of imaging studies is also limited as pancreatic imaging may also appear relatively preserved [5]. To address these challenges, clinical practice guidelines on EPI emphasise the importance of maintaining a low threshold for EPI screening in high-risk groups to improve diagnosis, particularly where nutritional compromise, weight loss, or persistent gastrointestinal symptoms are present [6,16,19].

Limitation of this Study

There is limited evidence of the prevalence and severity of EPI in patients with FCS since it is not routinely screened for, and faecal elastase is not able to detect all cases of EPI accurately. Additionally, there is paucity of evidence from EPI screening in FCS using other indirect or direct methods. These diagnostic challenges underlie the uncertainties regarding prevalence, diagnosis, management and severity of EPI in patients with FCS. Another limitation is the heterogeneity in the definitions and diagnostic criteria of EPI with current evidence suggesting under-recognition of EPI in rare conditions like FCS.

Conclusion

EPI is a biologically plausible but under-recognised complication of FCS. Recurrent hypertriglyceridaemia-induced acute pancreatitis, which is a hallmark complication of FCS, can result in progressive pancreatic injury, fibrosis, and loss of exocrine pancreatic function. Despite the well-established association between recurrent pancreatitis and EPI in other pancreatic disorders, no studies were identified that directly evaluated exocrine pancreatic function in patients with FCS, highlighting a substantial gap in the current evidence base. Unrecognised EPI may contribute to steatorrhea, malabsorption, fat-soluble vitamin deficiencies, weight instability, protein-calorie malnutrition, sarcopenia, reduced quality of life, and type 3c pancreatogenic diabetes mellitus in FCS patients. Given that EPI is a treatable condition, failure to identify it may represent a missed opportunity to reduce morbidity and improve nutritional outcomes in this already vulnerable patient population. Clinicians managing FCS should maintain a low threshold for considering EPI in patients with persistent gastrointestinal symptoms, unexplained weight loss, nutritional deficiencies, or a history of recurrent pancreatitis.

Recommendations

Future FCS registries and longitudinal cohort studies should incorporate pancreatic exocrine assessment to determine the prevalence, severity, and impact of EPI and to establish evidence-based recommendations for screening, diagnosis, and PERT treatment within FCS care pathways. Guideline committees for the care of patients with FCS are encouraged to provide pragmatic, standardized protocols for the diagnosis of EPI in FCS with recommendations on pancreatic enzyme replacement therapies. This will help to integrate EPI screening in triglyceride lipid clinic pathways with systematic pancreatic assessment in FCS care pathways.

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ISSN: 2574-1241

DOI: 10.26717/BJSTR.2026.66.010369

Ekitumi Robert Ofagbor . Biomed J Sci & Tech Res



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