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Chapter

Epidemiology and Risk Factors of Pancreatic Cancer

Michele Molinari, Hao Liu and Christof Kaltenmeier

Abstract

Pancreatic cancer (PC) is among the most common tumors of the gastrointestinal system in the world. In the United States and in other industrialized countries, it represents the fourth leading cause of cancer-related mortality. The incidence of PC increases with age and most patients are diagnosed after the age of 50. The overall prognosis of PC is poor. Most tumors are silent and they often present when metastatic. Only less than 15% of patients can undergo surgery, which represents the only potential cure for PC, and less than 10% of patients are alive after 5 years. In this chapter, we present the epidemiology of PC and its most common risk factors.

Keywords: pancreatic cancer, risk factors, epidemiology

1. Introduction

Worldwide, pancreatic cancer (PC) is the 12th most common cancer [1] and the fourth leading cause of cancer-related mortality in the United States with estimated 42,500 new cases and 35,000 deaths each year [2] (Figure 1). The age-standardized incidence of PC is 4.9 per 100,000 individuals [1] (Figure 2). There are significant variations in the incidence of PC among different geographical areas (Figure 3). In high-income countries, the incidence of PC is much higher than in low-income countries (11 vs. 3 per 100,000 individuals). PC ranks fifth after colorectal cancer, gastric cancer, hepatic cancer, and esophageal cancer among all gastrointestinal malignancies [3] (Table 1). Over time, the mortality rate for males has decreased by 0.4% while the mortality rate for females has increased by 4.4% [2]. More than 80% of PCs are diagnosed in patients older than 60 and almost 50% have distant metastases at the time of their clinical presentation [3–5]. Men are more frequently affected than women (Relative Risk (RR) = 1.3) and individuals of African American descent are at a higher risk in comparison to Caucasians (RR = 1.5) [3]. Despite some improvements in early diagnosis, surgical therapy, neoadjuvant therapy, adjuvant therapy and palliative interventions, the overall survival of patients with PC is still quite poor with only 6-9% of all patients being alive after 5 years (Figure 4) [6].
Figure 1.
Estimated age-standardized mortality rates of the most common types of malignancies in 2020 for patients older than 50 years in Canada and in the United States (data from the World Health Organization; https://gco.iarc.fr/).

Figure 2.
Age-standardized incidence of pancreatic cancer in the world (4.9 per 100,000 individuals) and in selected countries with high incidence of the tumor (data from the World Health Organization; https://gco.iarc.fr/).

Figure 3.
Estimated age-standardized incidence of pancreatic cancer in the world for individuals older than 50 years (data from the World Health Organization; https://gco.iarc.fr/).
### Table 1.
Estimated number of patients diagnosed with cancer with exclusion of nonmelanoma skin cancer, worldwide in 2020.

<table>
<thead>
<tr>
<th>Type of cancer</th>
<th>Total number</th>
<th>Crude rate</th>
<th>ASR (World) (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All cancers</td>
<td>18,094,716</td>
<td>232.1</td>
<td>190</td>
</tr>
<tr>
<td>Breast</td>
<td>2,261,419</td>
<td>58.5</td>
<td>478</td>
</tr>
<tr>
<td>Lung</td>
<td>2,206,771</td>
<td>28.3</td>
<td>22.4</td>
</tr>
<tr>
<td>Colorectum</td>
<td>1,931,590</td>
<td>24.8</td>
<td>19.5</td>
</tr>
<tr>
<td>Prostate</td>
<td>1,414,259</td>
<td>36</td>
<td>30.7</td>
</tr>
<tr>
<td>Stomach</td>
<td>1,089,103</td>
<td>14</td>
<td>11.1</td>
</tr>
<tr>
<td>Liver</td>
<td>905,677</td>
<td>11.6</td>
<td>9.5</td>
</tr>
<tr>
<td>Cervix uteri</td>
<td>604,127</td>
<td>15.6</td>
<td>13.3</td>
</tr>
<tr>
<td>Esophagus</td>
<td>604,100</td>
<td>7.8</td>
<td>6.3</td>
</tr>
<tr>
<td>Thyroid</td>
<td>586,202</td>
<td>7.5</td>
<td>6.6</td>
</tr>
<tr>
<td>Bladder</td>
<td>573,278</td>
<td>7.4</td>
<td>5.6</td>
</tr>
<tr>
<td>Non-Hodgkin lymphoma</td>
<td>544,352</td>
<td>7</td>
<td>5.8</td>
</tr>
<tr>
<td>Pancreas</td>
<td>495,773</td>
<td>6.4</td>
<td>4.9</td>
</tr>
<tr>
<td>Leukemia</td>
<td>474,519</td>
<td>6.1</td>
<td>5.4</td>
</tr>
<tr>
<td>Kidney</td>
<td>431,288</td>
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<td>4.6</td>
</tr>
<tr>
<td>Corpus uteri</td>
<td>417,367</td>
<td>10.8</td>
<td>8.7</td>
</tr>
<tr>
<td>Lip and oral cavity</td>
<td>377,713</td>
<td>4.8</td>
<td>4.1</td>
</tr>
<tr>
<td>Melanoma of skin</td>
<td>324,635</td>
<td>4.2</td>
<td>3.4</td>
</tr>
<tr>
<td>Ovary</td>
<td>313,959</td>
<td>8.1</td>
<td>6.6</td>
</tr>
<tr>
<td>Brain and central nervous system</td>
<td>308,102</td>
<td>4</td>
<td>3.5</td>
</tr>
<tr>
<td>Larynx</td>
<td>184,615</td>
<td>2.4</td>
<td>2</td>
</tr>
<tr>
<td>Multiple myeloma</td>
<td>176,404</td>
<td>2.3</td>
<td>1.8</td>
</tr>
<tr>
<td>Nasopharynx</td>
<td>133,354</td>
<td>1.7</td>
<td>1.5</td>
</tr>
<tr>
<td>Gallbladder</td>
<td>115,949</td>
<td>1.5</td>
<td>1.2</td>
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<tr>
<td>Oropharynx</td>
<td>98,412</td>
<td>1.3</td>
<td>1.1</td>
</tr>
<tr>
<td>Hypopharynx</td>
<td>84,254</td>
<td>1.1</td>
<td>0.91</td>
</tr>
<tr>
<td>Hodgkin lymphoma</td>
<td>83,087</td>
<td>1.1</td>
<td>0.98</td>
</tr>
<tr>
<td>Testis</td>
<td>74,458</td>
<td>1.9</td>
<td>1.8</td>
</tr>
<tr>
<td>Salivary glands</td>
<td>53,583</td>
<td>0.69</td>
<td>0.57</td>
</tr>
<tr>
<td>Vulva</td>
<td>45,240</td>
<td>1.2</td>
<td>0.85</td>
</tr>
<tr>
<td>Penis</td>
<td>36,068</td>
<td>0.92</td>
<td>0.8</td>
</tr>
<tr>
<td>Kaposi sarcoma</td>
<td>34,270</td>
<td>0.44</td>
<td>0.39</td>
</tr>
<tr>
<td>Mesothelioma</td>
<td>30,870</td>
<td>0.4</td>
<td>0.3</td>
</tr>
<tr>
<td>Vagina</td>
<td>17,908</td>
<td>0.46</td>
<td>0.36</td>
</tr>
</tbody>
</table>

All ages, crude, and age-standardized rates per 100,000 individuals (data from the World Health Organization; [https://gco.iarc.fr/]).
2. Risk factors

The strongest risk factor for PC is age. The incidence of PC increases significantly after the age of 50 and over 80% of PCs are diagnosed in patients older than 60 [7] (Figure 5).

Figure 4.
Estimated age-standardized mortality rate of pancreatic cancer in different parts of the world for individuals older than 50 years (data from the World Health Organization; https://gco.iarc.fr/).

Figure 5.
Incidence of pancreatic cancer in Canada during the period between 2011 and 2013 per 100,000 individuals stratified by age.
2.1 Smoking

The risk of PC in smokers ranks second to lung cancer [8] and it is proportionate to the frequency (≥30 cigarettes per day: Odds Ratio (OR) = 1.75), duration (≥50 years: OR = 2.13), and cumulative smoking dose (≥40 pack/years: OR = 1.78) [9]. A meta-analysis of 82 studies from 4 continents has shown that cigarette smokers were diagnosed at significantly younger ages and had a 75% increased risk of developing PC in comparison to the regular population [10], and the risk persisted for 5 to 15 years after cessation [11]. In a case–control study of 808 PC patients matched against 808 healthy controls, in comparison to male counterparts, female smokers were at increased risk of developing PC as they suffered from a synergistic interaction between cigarette smoking, diabetes mellitus (OR = 9.3), and family history of PC (OR = 12.8) [12].

2.2 Diabetes

Nearly 80% of PC patients have either frank diabetes or impaired glucose tolerance at the time of their diagnosis [13]. Diabetes is often found concomitantly or during the two years preceding the diagnosis of PC [14]. Several studies have assessed the link between diabetes and PC with conflicting results. A meta-analysis of 11 cohort studies found that the relative risk for diabetic patients was 2.1 (95% CI 1.6–2.8) in comparison to nondiabetic individuals [15]. These findings were supported by another cohort study of 100,000 Danish diabetic patients that found a standardized incidence ratio of 2.1 (95% CI 1.9–2.4) [16]. A large prospective cohort study of 20,475 men and 15,183 women in the United States, has shown that the relative risk of dying from PC adjusted for age, race, history of cigarette smoking, and body mass index (BMI) was proportionate to the severity of diabetes. The RR was 1.65 for post-load plasma glucose levels between 6.7 and 8.8 mmol/L, 1.60 for levels between 8.9 and 11.0 mmol/L, and 2.15 for levels equal or more than 11.1 mmol/L [17]. Diabetes can present as an early manifestation of PC. Approximately 1% of new onset of diabetes in patients older than 50 is linked to PC [18]. Despite these findings, there is no evidence that screening patients for PC when newly diagnosed with diabetes [19] could reduce their mortality risk [5].

It is important to highlight that the link between abnormal glycemia and PC exists only for type II diabetes. A meta-analysis of 36 studies indicated that the OR of PC for patients with type II diabetes was 2.1 [20] while there are no reports showing an association between PC and type I diabetes [21].

Family history of diabetes does not appear to be a risk factor for PC [22]. On the other hand, a recent prospective study found that women with gestational diabetes are at a higher risk of developing PC with an estimated relative risk of 7.1 (95% CI = 2.8–18.0) [23]. Gapstur and colleagues [17] have proposed that high levels of insulin can cause abnormalities in the regulation of the insulin-like growth factor I (IGF1) receptor [19] that down-regulates the IGF binding protein 1, (IGFBP1) [20] causing an increase cell growth in PC cell lines [24, 25].

2.3 Alcohol

The role of alcohol in the predisposition of PC is controversial. Several studies have shown inconsistent findings due to multiple associations between alcohol consumption and other confounders such as cigarette smoking, lower socioeconomic status [26], and history of pancreatitis and diabetes [25]. A recent pooled analysis of 14 cohort studies with a sample of 862,664 individuals has shown a slight positive association...
between PC and alcohol consumption when larger than 30 gm/day (RR 1.22; 95% CI 1.03–1.45) [27]. On the other hand, a smaller epidemiological European study of 555 patients did not show any association between PC and alcohol consumption [28]. Yet, there is some evidence that compared with light drinkers, men consuming a large amount of hard liquor suffered from 62% increased risk of PC (95% CI 1.24–2.10) [11, 29] but this did not pan out for women and for beer and wine drinkers [29].

Although moderate alcohol consumption is not a risk factor, African Americans seem to be at a significantly higher risk of developing PC after adjusting for their drinking habits suggesting that racial differences play a role [30].

2.4 Pancreatitis

Several studies have shown a positive association between PC and history of pancreatitis. However, the magnitude of this phenomenon remains poorly understood [31, 32]. An international epidemiological study reported that both genders with chronic pancreatitis had an increased risk of developing PC independently from the cause of the disease [32]. A large case–control study indicated that chronic pancreatitis lasting more than 7 years was associated with a higher risk of PC (RR = 2.04; 95% CI 1.53–2.72) [33]. A large Italian study from 1983 to 1992 found similar results reporting that the risk increased after 5 or more years of chronic pancreatitis (RR in the first 4 years = 2.1, RR after 5 years = 6.9) [29]. These findings have been challenged by a more recent international study that showed that the risk was significantly increased only in the early years after the onset of pancreatitis. This observation suggested that pancreatitis might represent a manifestation of PC that becomes apparent only several years later rather than an independent risk factor for PC.

2.5 Hereditary pancreatitis

Hereditary pancreatitis affects 0.3 per 100,000 [34]. In 1996, it was found that hereditary pancreatitis was due to an autosomal dominant defect of the cationic trypsinogen gene (PRSS1) in 7q35 chromosome region [35]. Since then, more than 30 different PRSS1 mutations have been identified and reported in a few families. The risk of developing PC is particularly high for patients affected by hereditary pancreatitis who are at 53 times higher risk of developing the tumor in comparison to individuals without history of hereditary pancreatitis [36]. This observation was confirmed by another study that estimated a 40% cumulative risk of PC in patients with hereditary pancreatitis by the age of 70 [37]. For patients with paternal inheritance of hereditary pancreatitis, the cumulative risk of PC was even higher with a risk of up to 75% [37]. Patients with hereditary pancreatitis have high concentrations of cytokines, reactive oxygen molecules, and pro-inflammatory compounds that can lead to DNA damage, and despite DNA repair systems, these mechanisms seem responsible for the higher risk of genetic mutations leading to PC [33, 38].

2.6 Intraductal papillary mucinous neoplasms

The definition of intraductal papillary mucinous neoplasm (IPMN) is applied to a family of benign pancreatic cysts that can transform into PC [39]. The risk factors and the true incidence of IPMN are still unclear. These cysts produce mucin and are divided into two groups: IPMNs that affect the side branches of the pancreatic ducts and IPMN that affect the main pancreatic duct. Some patients have mixed IPMNs as they have
cystic lesions in both the side branch and main pancreatic duct. IPMNs are responsible for 20–30% of PC cases. Current evidence suggests that only IPMNs affecting the main pancreatic duct are at high risk for malignant transformation. A recent meta-analysis [39] of 2411 patients with low-risk IPMNs and 825 patients with high-risk IPMNs has shown that the cumulative incidence of PC was significantly different between the two groups. For low-risk IPMNs, the cumulative incidence of PC was 0.02% at 1 year, 1.4% at 3 years, 3.1% at 5 years, and 7.7% at 10 years. On the other hand, for high-risk IPMNs, the cumulative incidence of PC was 1.9% at 1 year, 5.7% at 3 years, 9.7% at 5 years, and 24.6% at 10 years.

2.7 Genetic predisposition for pancreatic cancer

The presence of genetic predisposing factors for the development of PC has been an area of intense research during the last few decades. Case reports of families with multiple members diagnosed with PC suggest that for some patients, PC might be hereditary [40]. A large population study on twins identified hereditary factors for prostatic, breast, and colorectal cancers, however, this was not detected for PC [41]. A Canadian study on patients with suspected hereditary cancer syndromes found that the standardized incidence of PC was 4.5 (CI 0.54–16.) when cancer affected one 1st degree relative; the standardized incidence increased to 6.4 (CI 1.8–16.4) and 32 (CI 10.4–74.7) when two and three 1st degree relatives were affected, respectively [42]. This translates to an estimated incidence of PC of 41, 58, and 288 per 100,000 individuals, respectively, compared to 9 per 100,000 for the general population [43].

Brentnall et al. [44] and Meckler and colleagues [45] described examples of autosomal dominant PC in individuals presenting at early age (median age 43 years) and with high genetic penetrance (more than 80%). A mutation causing a proline (hydrophobic) to serine (hydrophilic) amino acid change (P239S) within a highly conserved region of the gene encoding paladin (PALLD) was found in all affected family members (family X). Another study has shown that the P239S mutation was only specific for the family X while it was not a common finding in other individuals with suspected familial PC [46]. Currently, genetic predisposition is thought to be responsible for 7–10% of all PC [47]. Genetic factors including germline mutations in p16/CDKN2A [48], BRCA2 [49–51], and STK 11 [52] genes are thought to increase the risk of PC. The combination of all these known genetic factors accounts for less than 20% of the familial aggregation of PC, suggesting that other genes play a role in the development of familial PC.

A systematic review and meta-analysis of studies on familial PC has shown that individuals with positive family history have nearly two-fold increased risk of developing PC (RR = 1.80, CI 1.48–2.12) [53]. Therefore, families with two or more cases may benefit from a comprehensive risk assessment involving collection of detailed family history information and data regarding other risk factors for PC [54]. A case–control study of PC in two Canadian provinces (Ontario and Quebec) assessed a total of 174 PC cases and 136 healthy controls. Information regarding the ages and sites of cancer was taken in 966 first-degree relatives of PC patients and for 903 first-degree relatives of the control group. PC was the only malignancy in excess in relatives of patients with PC, compared to the control group (RR = 5, p = 0.01). The lifetime risk of PC was 4.7% for the first-degree relatives and the risk was 7.2% for relatives of patients diagnosed before the age of 60 [55].

Besides the isolated aggregation of PC in some families, several other hereditary disorders predispose the development of PC in known familial cancer conditions [56].
These include hereditary pancreatitis, Peutz-Jeghers syndrome (STK11 mutation), familial atypical multiple mole melanoma (FAMMM mutation), familial breast cancer (BRCA1, BRCA2, PALB2, CDKN2A, ATM mutations) and ovarian cancer, Li-Fraumeni syndrome (TP53 mutation), Fanconi anemia, ataxia-telangiectasia, familial adenomatous polyposis, cystic fibrosis, and possible hereditary nonpolyposis colon cancer (HNPCC) or Lynch syndrome (MLH1, MSH2, MSH6, PMS2, EPCAM mutations) [4, 54, 57–59].

2.8 Familial pancreatic cancer registries

As the prognosis of PC is generally poor, there has been a strong interest to detect genes or other markers that could help identify high-risk patients in early stage. Although a precise genetic marker for this scope is not currently available, geneticists and epidemiologists have been profiling traits of high-risk families enrolled in registries established in North America and Europe [60]. Even if there is no standardized definition for familial PC, most authors apply the term to families with at least two first-degree relatives affected by PC in the absence of other predisposing familial conditions [60]. The creation of familial PC registries has been used not only for identification of genetic mutations but also for the screening of high-risk individuals. In selected centers in North America and Europe, screening programs for high-risk individuals have been implemented with the use of endoscopic ultrasound and computed tomography (CT) scan or magnetic resonance imaging (MRI). Such early diagnosis of PC within a comprehensive screening program is hoped to ultimately result in improved survival [61]. The discovery of the genetic bases of inherited PC continues to be an active area of research and in 2001 a multi-center linkage was formed to conduct studies aimed at the localization and identification of PC susceptibility genes (PACGENE) [62]. The complex nature of pedigree data makes it difficult to accurately assess risk based upon the simple counting of the number of affected family members, as it does not adjust for family size, age of onset of PC, and exact relationship between affected family members. Therefore, computer programs have been developed to integrate these complex risk factors and pedigree data. In April 2007 the 1st risk prediction tool for PC, PancPro (https://projects.iq.harvard.edu/bayesmendel/pancpro) was released [63]. This model provides accurate risk assessment for kindreds with familial PC as the receiver operating characteristic (ROC) curve was 0.75, which is considered good for predictive models.

3. Nutritional status

Several studies have explored the relationship between body mass index (BMI) lifestyle, diet, and the risk of PC, but uncertainty regarding the strength of this relationship still exists. A recent case–control study of 841 patients and 754 healthy controls showed that individuals with BMI of 25–29.9 had an OR of 1.67 (95% CI = 1.20–2.34) in comparison to obese patients (BMI of ≥30) who had an OR of 2.58 (95% CI = 1.70–3.90) independently of their diabetes status [64]. The duration of being overweight was significantly longer among patients with PC than among controls. Being obese or overweight, particularly in early adulthood, resulted in earlier onset of PC (age at presentation of PC was 61 years for overweight patients and 59 years for obese) when compared to the median age of diagnosis being 64 in the general population [65]. A few studies reported that central weight gain measured by
waist circumference and/or waist-to-hip ratio had a statistically significant increased risk compared to those with peripheral weight gain (RR = 1.45, 95% CI 1.02–2.07) [66, 67]. All the known risk factors for PC are summarized in Table 2.

**Table 2. Known risk factors for pancreatic cancer.**

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (more than 60 years)</td>
<td></td>
</tr>
<tr>
<td>Smoking:</td>
<td></td>
</tr>
<tr>
<td>Diabetes:</td>
<td>Type II</td>
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<tr>
<td></td>
<td>Gestational diabetes</td>
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<tr>
<td></td>
<td>Impaired glucose tolerance</td>
</tr>
<tr>
<td>Alcohol:</td>
<td></td>
</tr>
<tr>
<td>Pancreatitits:</td>
<td>Acute</td>
</tr>
<tr>
<td></td>
<td>Chronic</td>
</tr>
<tr>
<td>Genetic predisposition</td>
<td></td>
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<tr>
<td>Family history:</td>
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<tr>
<td>Hereditary disorders:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hereditary pancreatitis</td>
</tr>
<tr>
<td></td>
<td>Puoz-Jeghers syndrome</td>
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<tr>
<td></td>
<td>FAMMM</td>
</tr>
<tr>
<td></td>
<td>Familial breast and ovarian cancer</td>
</tr>
<tr>
<td></td>
<td>Li-Fraumeni syndrome</td>
</tr>
<tr>
<td></td>
<td>Fanconi anemia</td>
</tr>
<tr>
<td></td>
<td>Ataxia-telengectasiaistelangiectasia</td>
</tr>
<tr>
<td></td>
<td>Familial adenomatous polyposis</td>
</tr>
<tr>
<td></td>
<td>Cystic fibrosis</td>
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<tr>
<td></td>
<td>HNPCC</td>
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<tr>
<td></td>
<td>Lynch syndrome</td>
</tr>
<tr>
<td>Obesity:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Intraductal Papillary Mucinous Neoplasms</td>
</tr>
</tbody>
</table>

FAMMM: familial atypical multiple mole melanoma; HNPCC: hereditary nonpolyposis colon cancer.

waist circumference and/or waist-to-hip ratio had a statistically significant increased risk compared to those with peripheral weight gain (RR = 1.45, 95% CI 1.02–2.07) [66, 67]. All the known risk factors for PC are summarized in Table 2.

### 4. Screening for pancreatic cancer

The role of screening for PC is not recommended for asymptomatic average-risk individuals [68, 69] as it is estimated that it would generate more harm than good. With an incidence of only 1.6% of individuals developing PC during their lifetime, Lucas et al. [68] estimated that even with an ideal screening tool with 99% sensitivity and 99% specificity, 1000 false positive results would be generated when the test is applied to 100,000 individuals. Current guidelines recommend that only healthy individuals with at least a 5% or higher risk of developing PC should be considered for
screening programs [70] at age 50, or 10 years younger than the earliest diagnosis of PC in the family. These individuals must have two or more blood relatives diagnosed with PC with at least one affected first-degree relative [70, 71].

Guidelines also recommend that individuals with germline mutations in the genes listed above should consider screening beginning at age 50, or 10 years younger than the earliest pancreatic cancer diagnosis in the family, if they have a family history of PC. Some experts have recommended that all individuals with germline mutations in STK11 (which causes Peutz-Jeghers syndrome) or CDKN2A (which causes familial atypical multiple mole melanoma [FAMMM] syndrome), ATM, BRCA1, BRCA2, CDKN2A, PALB2, PRSS1, STK11, TP53, and the Lynch syndrome mismatch repair genes undergo screening for PC regardless of their family history. Peutz-Jeghers syndrome patients are recommended to begin screening at ages 30 to 35. FAMMM syndrome patients are recommended to begin screening for PC at age 40. Magnetic resonance imaging (MRI) or endoscopic ultrasound (EUS) is the two radiological modalities recommended for the screening of patients considered at increased risk of developing PC.

5. Conclusions

PC is among the most common malignancies of the gastrointestinal system. Its incidence increases after the age of 50. Most patients diagnosed with PC have advanced disease at the time of their presentation. Age, cigarette smoking, alcohol abuse, chronic pancreatitis, and genetic factors are well-known predisposing factors for PC. Screening protocols for PC in the general population are not recommended as the incidence of PC is relatively low. The use of screening programs for high-risk patients is still under investigation.
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