Original Research Article

Gastric cancer in Cameroon: Epidemiological profile and histopathological appearance of 574 cases

Abstract:

Objective: To describe the epidemiological and histopathological aspects of stomach cancer in Cameroon.

Material and methods: This was a retrospective, descriptive study of histologically confirmed stomach cancers recruited from histopathology laboratories in Cameroon spread over a period of 13 years (2004-2016). The variables studied were: the frequency, age, gender, risk factors, location and histopathologic type.

Results: At the end of our study, 574 cancers of the stomach were recorded. Men were predominantly affected with 312 cases (54.36%), the sex ratio of men to 1.19. The mean age of onset was 52.95 +/- 16.27 years for all sexes, with extremes ranging from 20 months to 92 years. Patients aged between 50 and 59 years were the predominant age group (24.39%) compared to the other groups. Traders and retirees represented the most affected groups with 20.97% and 14.52% respectively. The main risk factors were: chronic Helicobacter gastritis Pylori 59.73%, chronic smoking 16.11% and chronic alcoholism in 7.12%. Upper digestive endoscopy with biopsy and histological examination was the main means of assertion, 84.18%. The antral location was the most represented with 52.67%. Adenocarcinoma was the most frequent histological type with 419 cases (72.99).

Conclusion: Stomach cancer is an important public health problem in Cameroon. Its annual frequency increases since 2009. This cancer affects all 10 regions of Cameroon. The western region is the most represented. The mean age of onset is 52.95 years with a male predominance. Chronic gastritis with helicobacter pylori is the main risk factor. The most common histological type is adenocarcinoma

Keywords: Gastric cancer; epidemiology; histopathology; Cameroon.

1. INTRODUCTION

Gastric cancer is a malignant tumor that develops from its histological structures (primary tumors) or from other organs (secondary tumors). It represents 6.8% of all cancers and ranks fifth among the most common malignant tumors behind lung cancer (13.0%), breast cancer (11.9%), colorectal cancer (9%), 7%) and prostate cancer (7.8%) [1]. The International Agency for Research on Cancer (IARC) in 2012 estimated more than 950000 new cases of stomach cancer with a sex ratio (m / f) at 2 / 1, the average age of onset is 70 years [1]. The highest incidence was found in Asia (Republic of Korea with 49 cases per 100,000 inhabitants), in Central America (Guatemala with 24 cases per 100,000 inhabitants), and in Eastern Europe (Albania, 29 cases per 100000 inhabitants). In Africa, Mali has the highest incidence (9.5 cases per 100,000 inhabitants) [2]. Other studies in Africa on the frequencies of stomach cancer have been conducted. In Togo Bagny et al in 2015 reported a frequency of 14% of stomach cancers compared to digestive cancers with a mean age of 58.82 years ± 13.43 years. The ulcero-budding form was predominant (22.85%), the antrum being the most affected zone [4]. Cancer has become a major public health issue, globally in both developed and developing countries as it is one of the leading causes of death. In 2012, stomach cancer was the third leading cause of cancer death in the world with 723,000 deaths behind lung and liver cancers

[2]. Mortality rates for the two highest sexes were found in Asia (Mongolia 25.3 per 100,000) and Eastern Europe (Albania 24.7 per 100,000). In Zimbabwe, 17.22 deaths per 100,000 were found [2]. Several risk factors have been implicated in the genesis of gastric cancer, particularly Helicobacter pylori and Epstein barr virus infections. Some lifestyles (tobacco, alcohol, smoking, excessive salting), cadherin 1 gene, Biermer's disease and chronic gastritis taking long-term anti-inflammatory drugs [5-6]. Upper gastrointestinal endoscopy with biopsy and histopathological examination allow to diagnose stomach cancer at early stages. More than 90% of malignant tumors of the stomach are adenocarcinomas according to the Lauren classification [7]. The main therapeutic methods of gastric cancer are: surgery for loco regional forms, chemo and radiotherapy, cytoreduction surgery followed by intraperitoneal chemotherapy, palliative chemotherapy, targeted therapy [8-12]. Despite this therapeutic progress, stomach cancer remains very deadly with an overall survival rate of 5 years not exceeding 25% due to the insidious evolution often correlated to a late diagnosis [13]. In Cameroon, Ankouane et al in 2015 reported in Yaoundé a frequency of 42.9% of stomach cancers compared to other digestive cancers with a sex ratio (H / F) 3: 1. The average age of diagnosis was 53.4 years. [14]. In Douala, in 2016, Engbang et al found 48% of stomach cancers out of 414 digestive cancers with a mean age of 56.97 years [15]. Cameroon does not have a functioning national cancer registry yet, its importance in guiding cancer policies is well known. These observations led us to carry out this work in order to contribute to the production of epidemiological and histopathological data at the national level.

2. MATERIAL AND METHODS

This is a retrospective descriptive and analytical study of histologically proven malignant colon tumors, diagnosed between January 2004 and December 2016. The study took place in the main public and private pathological anatomy laboratories in Cameroon. We needed the reports of histopathological examinations of the various laboratories solicited, all the necessary documentation relating to our subject (books, journals, specific publications ...), and a well-defined office equipment. The samples generally come from previously unresolved surgery, cancerology or gastroenterology departments. Once in the pathology departments, they are fixed at 10% formalin, and then the macroscopic study in which the pieces are cut. The pieces are dehydrated by passing through several tanks of alcohol at increasing concentrations, then included in paraffin, then cut with a microtome to a thickness of 5 micron. They are then deparaffinized by xylene lightening, and the staining is done with haematin-eosin followed by a reading made using a microscope. The parameters studied were frequency, age, sex, histological type of the tumor. Data entry was done using computer based statistical Package for Social Sciences (SPSS) version 20. The elements of descriptive statistics were used to calculate the frequencies and proportions.

3. RESULTS

3.1.Frequency

We collected 1047 cases of cancers of the digestive tract in Cameroon, among which gastric cancer is in the first position (574 cases; 40.80%) (Figure 1).

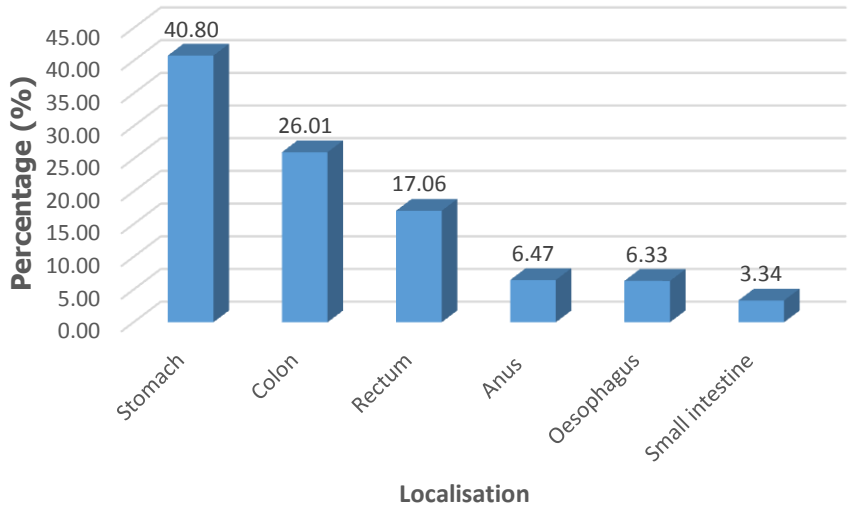

Figure 1. Distribution of cancers according to the segment of the digestive tube

3.2.According to chronological evolution

The distribution of our patients over the last 13 years showed a variation in frequency from one year to another. The highest frequency was noted in 2011 with 66 cases. The lowest frequency was recorded in 2008 with 22 cases.

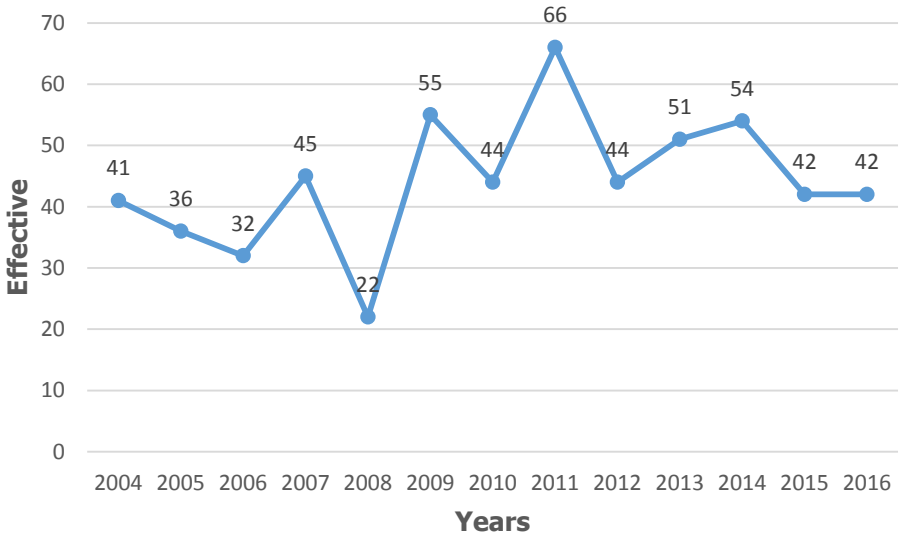

Figure 2: Evolution of colon cancer in the years from 2004 to 2016 (n = 366)

3.3.Distribution by sex

Of the 574 cases of stomach cancer found, the male sex is represented by 312 cases (54.36%) or 22.17% of all digestive cancers and the female sex is represented by 262 cases (45.64%) or 18.62% of all digestive cancers. The sex ratio H / F is 1.19. (Table I)

Table I: Distribution of digestive tract cancers by sex

Organ	Stomach		Colon		Rectum		Anus		Œsophagus		S intestine		Total
Sex	**H**	**F**	**H**	**F**	**H**	**F**	**H**	**F**	**H**	**F**	**H**	**F**	
Effective	312	262	193	173	130	110	42	49	67	22	24	23	1407
% Effective	22.17	18.62	13.72	12.30	9.24	7.82	2.99	3.48	4.76	1.56	1.71	1.63	100
Total	574		366		240		91		89		47		1407
% Total	40.80		26.01		17.06		6.47		6.33		3.34		100

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3.4. Age distribution

As schown in figure 3, the age group 50 to 59 was the most represented. The average age was 52.95 +/- 16.27 years old, regardless of gender, with extremes ranging from 20 months to 92 years.

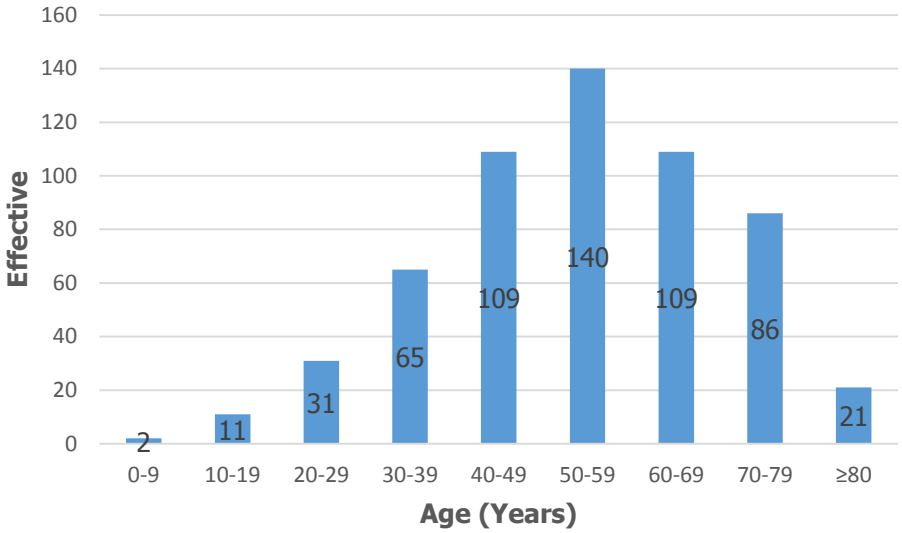

Figure 3: Distribution of patients by age group.

3.5.Distribution according to risk factors

In our series, risk factors were found in 298 cancers. These were mainly: chronic gastritis with Helicobacter pylori with 59.73% or 178/298, chronic smoking with 16.11% of cases or 48/298, chronic alcoholism with 7.27% of cases or 23/298, an excess of spice with 4.36% is 13/298, an excess of salt with 4.36% or 13/298.

Table II: Factors Associated with the Risk of Stomach Cancer

	Effective	Percentage (%)
Smoking	48	16,11
HP + chronic gastritis	178	59 ,73

Alcoholism	23	7,72
Spices	13	4,36
GOR	4	1,34
Polyp	2	0 ,67
Excess of salt	13	4 ,36
Excess of smoked food	1	0,34
EBV	7	2,35
Partial gastrectomy	5	1,68
Presence of other cancer	4	1,13
Total	**298**	**100,00**

HP – Helicobacter pylori; GOR - Gastroesophageal reflux; EBV - Epstein-Barr Virus

3.6. Tumor localization

We identified endoscopic localization in 442 cases out of 574. The gastric antrum was the most represented zone with 233 cases (52.67%). Anthropyloric and antroptic locations were the least represented with 13 cases or 2.94%.

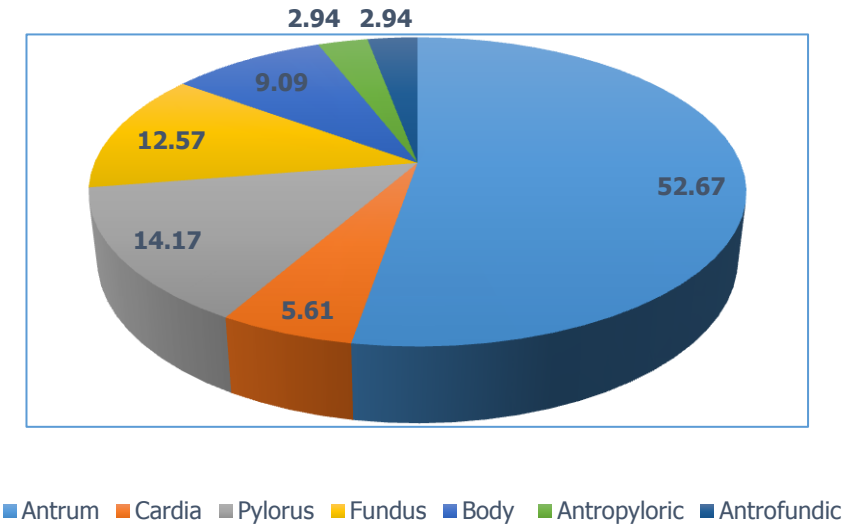

Figure 4: Distribution by localization

3.7.Anatomopathology

3.7.1. Type of sample

In our series, 84.18% (483 cases) of anatomical specimens submitted for analysis were taken from biopsies.

3.7.2. Macroscopic aspect

At macroscopy, the ulcerated aspect was the most represented with 230 cases (40.38%), followed by the ulcero-budding appearance with 138 cases or 24.04%.

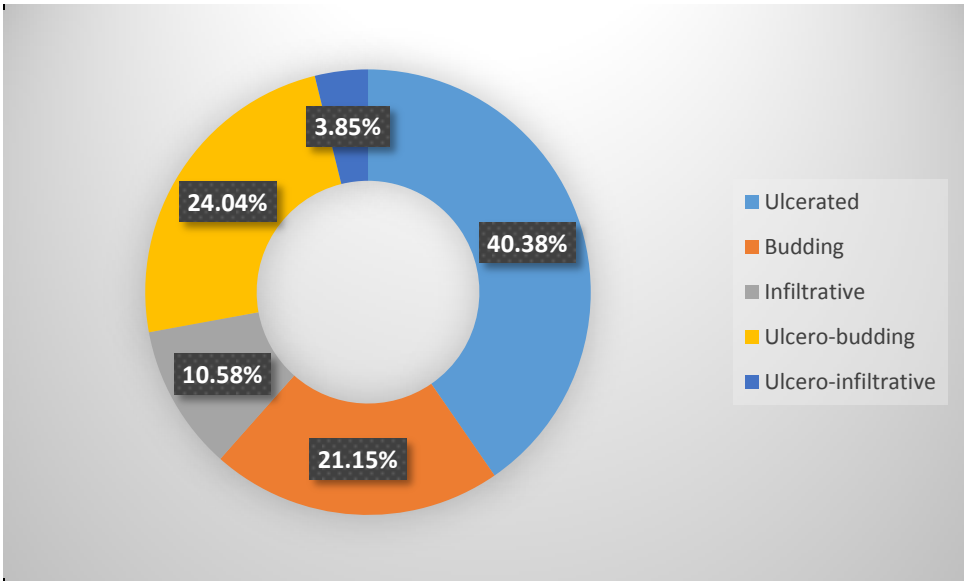

Figure 5: Case distribution according to the macroscopic aspects of the tumor.

3.7.3. Histological types

Whether from biopsy or from room of gastrectomy, histological status was known in 574 of our patients. In 419 cases, this was adenocarcinoma (ADK), the most represented 72.99% histological type. According to the Lauren and WHO classifications, 169 cases of adenocarcinoma were identified, the intestinal type being the most represented with 164 cases (Papillary 43, mucinous: 49 Tubular: 42). On the other hand 5 cases of diffuse type were found. We did not find any precision in 250 adenocarcinomas. Kaposi's sarcoma was present in 15.67% of cases and lymphomas in 9.58% of cases. And the other rarer types such as: Leiomyosarcomas 0.87%, Malignant hemangioma, Plasmacytoma, Carcinoid stromal tumor and Malignant Schwanoma with 0.17%. The table below shows the frequency of each histological type.

Table III: Distribution by histological type

Types	Fréquencies	Percentage (%)
Adénocarcinoma	419	72,99
Kaposi Sarcoma	90	15,67
Lymphoma	55	9,58
Leiomyosarcoma	5	0,87
malgnant Hemangioma	1	0,17
Plasmocytoma	1	0,17
stromal Tumor	1	0,17
Carcinoïd	1	0,17
malignant Schwanome	1	0,17
Total	**574**	**100,00**

According to the differentiations of adenocarcinomas, 133 cases were found. 79 cases were well differentiated.

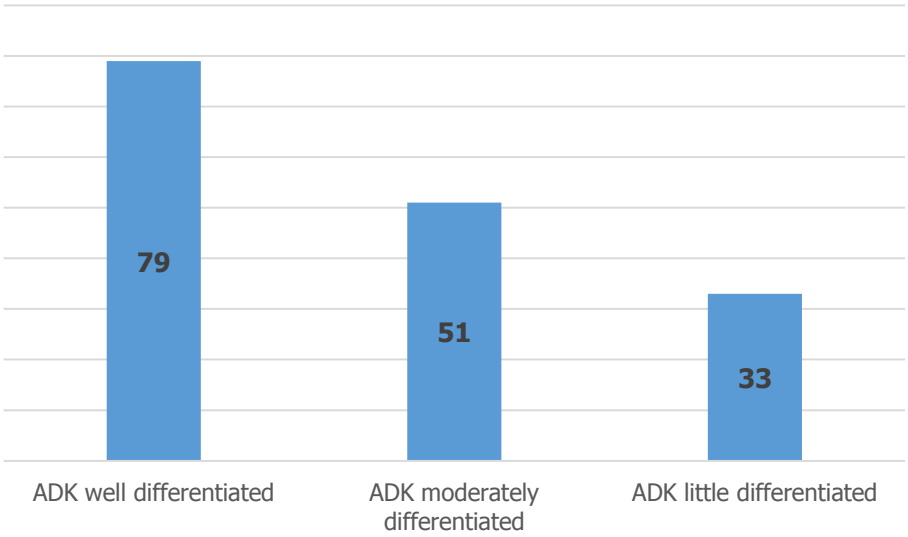

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Figure 6: Differentiation of adenocarcinomas

Discussion

In the study, we found 1407 cases of digestive cancers in Cameroon among which the stomach ranks first with 574 cases or 40.80% (men 312 cases and women 262). This frequency is similar to that of Ankouane et al which in 2015 in Yaoundé found 42 gastric cancers or 42.9% [14] and that of Engbang et al which 2016 in Douala found 199 cases out of 414 digestive cancers or 48% [15]. It should be noted, however, that these two studies were regional studies while ours is of a national character. Higher frequencies were reported by Diarra in 2005 [16] in Mali and Effi et al in 2011 [17] in Côte d'Ivoire: 103/127 or 80.10% and 722/1620 cases respectively, 44, 6%. This apparently reduced frequency in our series can have several causes. Thus, some patients with suspicious lesions of gastric cancer, whose malignant nature was not confirmed histologically, were excluded from our study. In addition, the diagnosis of gastric cancer can also be made in a surgical environment, some patients have escaped our selection. Most patients consult late because many of them resort to self-medication and practice of traditional medicine. Consequently, they are seen in consultation during a complication phase, where it is no longer possible to perform esophago-gastroduodenal fibroscopy, and therefore to make the diagnosis of gastric cancer.

From January 1, 2004 to December 31, 2016, the number of cases per year saw many variations sawtooth. The number of cases diagnosed per year was 44.15 cases / year. In the first 4 years, this number has decreased from 42 cases per year in 2004 to 22 cases in 2008. This fall can be explained by the fact that endoscopic diagnosis was not sufficiently well understood. From 2009 to 2014, it was noted that an increase in the number of cases per year with a peak of 66 cases in 2011. This peak is correlated with a Moroccan study that in 2011 found 48 cases [18].

The male sex was the most represented in our study with a sex ratio of 1.19. This result is comparable to several studies in Africa as well as in other continents [19, 20, 21] where gastric cancer affects men more than women, with values of 1.62; 1.45; 1.26. It has been suggested that female hormones have a protective role [22].

The most affected age group in our study was 50-59 years old with 24.39%. Extreme ages ranged from 20 months to 92 years. The average age was 52.95 +/- 16.27 years old. These results are similar to Ankouane in Yaoundé, which found an average age of 53.4 years [14]. In Africa, our results are consistent with those found by Tounkara with an average age of 57 years [19] and Coulibaly W with 55.90 years in Mali [23]. They are also close to those obtained by Ouattara et al with 58 years in Burkina Faso [24] and by Afuwape who found 52.6 years in Nigeria [20]. On the other hand, our average age is lower than those of other authors outside Africa: Saito with 62 years in Japan in 2008 [25]; David D found an average age of 71 in the USA in 2009 [26]; Cathy B in Holland and Pinto in Italy were 68 and 63.5 respectively [26, 27].

This difference between continents could be explained by: firstly, less exposure to risk factors and favoring factors in developed countries; this by better management of gastritis and good preservation of food cold. Secondly, the youth of the African population in general and Cameroonian in particular. Indeed, according to a demographic study conducted in 2010 by the Central Bureau of Censuses and Population Studies (BUCREP), more than 70% of the Cameroonian population was under 50 years old [28]. In addition, according to WHO 2012, the life expectancy of men and women at birth is 51 years [1].

Helicobacter pylori infestation: It is the only bacterium recognized and classified as a carcinogen by the WHO. H. pylori was discovered in 1982 by Marshall and Warren in the human gastric antrum. It causes a proliferation of lymphoid follicles in the gastric mucosa, whereas it is normally devoid of lymphoid follicles and is the first step in the development of gastric lymphoma B of low malignancy type MALT (mucosae associated lymphoid tissue). The eradication of this bacterium would result in a regression of this type of lymphoma in 90 to 100% of cases. There is a relationship between gastric cancer and HP, through chronic gastritis, gastric atrophy, intestinal metaplasia and dysplasia leading to the onset of cancer. Hypochlorhydria promotes microbial proliferation and consequently the formation of carcinogenic nitrosamines [29]. Epidemiological studies have shown that the risk of gastric cancer is higher in H. pylori-infected persons than in H. pylori-negative persons and that H. pylori infection precedes the development of gastric cancer [30-31]. H. pylori is associated with adenocarcinoma of the distal (noncardia) stomach but not cancer of the proximal stomach. Experimental orogastric infection of Mongolian gerbils with H. pylori can result in the development of gastric cancer [32]. H. pylori colonizes the stomach and elicits a gastric mucosal inflammatory response termed “gastritis” in both humans and experimentally infected animals. Gastritis is one of the first detectable changes in a stepwise pathway of histologic abnormalities that can ultimately culminate in gastric cancer: inflammation, gastric atrophy (loss of specialized cell types such as parietal cells and chief cells), intestinal metaplasia (presence of intestinal-type epithelium in the stomach), and dysplasia [32, 3]. The development of gastric cancer in the setting of *H. pylori* infection is thought to be a long-term consequence of many alterations, including chronic inflammation (which contributes to the pathogenesis of many types of malignancy) , DNA damage, activation of gastric stem cells, In our study, 298 stomach cancers were associated with a risk factor. changes in cell proliferation and apoptosis, changes in epithelial differentiation and polarity, degradation of tumor suppressors, and impaired gastric acidification, leading to bacterial overgrowth with species not found in the normal acidic stomach [32, 33, 34]. It has been generally accepted that the risk of cancer is highest among patients in whom the primary colonization causes acute and then chronic inflammation [35]. To our knowledge, certain *H. pylori* strains seem to differently increase the risk of cancer, depending on the existence of certain bacterial genotypes (for example: *cagA*) [36, 37]. Bacterial-secreted CagA, inducing high levels of chronic inflammation, is the main factor increasing mutagenesis rate, oxidative-stress, and increased mismatch repair pathways, resulting in gastric carcinogenesis [38, 39]

Thus, 59.73% of adenocarcinoma and 60.00% of lymphomas were associated with chronic gastritis induced by Helicobacter pylori. Our results are similar to those of Ankouane et al, which in 2015 reported that Helicobacter pylori was associated with 100% intestinal type adenocarcinoma, 72.2% diffuse adenocarcinoma and 100% Lymphoma [14]. In 2013, Yaoundé Noah et al found an overall prevalence of Helicobacter pylori infection of 72.5% (124/171) [40].

According to the European Prospective Investigation into Cancer and Nutrition (EPIC), a significant association between smoking and risk of gastric cancer has been identified. The risk of ever smokers was higher than that of former smokers; it also rose with the intensity and duration of smoking to decrease after 10 years of weaning [41]. In our series, 16.11% of cancers were associated with chronic smoking. Chronic alcoholism was found in 7.27% of cases. Alcohol and tobacco were found in 16.11% and 7.27% respectively. Acetaldehyde is the main metabolite of alcohol in the digestive tract. The production of acetaldehyde results from the metabolism of ethanol by bacteria present in the oral cavity and in the stomach in the event of achlorhydria-induced microbial proliferation. Most foods contain small amounts of alcohol that these bacteria convert to acetaldehyde. Acetaldehyde is also present in large quantities in food products whose production requires a fermentation process; It is used as an artificial flavor in the manufacture of many foods: yogurts, sweets, pastries, soft drinks, alcoholic beverages. Some polymorphisms of acetaldehyde dehydrogenase (ALDH2) and alcohol dehydrogenase (ADH) increase the risk of cancer in regular drinkers by increasing the mucosal exposure of the upper digestive tract to acetaldehyde. Smoking increases the risk of stomach cancer [42, 43]. Acetaldehyde has been shown to induce DNA lesions, generate free radicals, and bind to enzymes involved in DNA repair and antioxidant protection [44]. Heavy alcohol consumption (40 g/d) is known to induce expression of cytochrome P4502E1 in human liver and in rat gastrointestinal mucosa [42, 45]. Thus, alcohol-induced cytochrome P4502E1 could contribute to the formation of reactive oxygen species in the gastrointestinal tract and to the activation of procarcinogens such as nitrosamines that may be present in beer (and in processed meats and tobacco smoke), as mentioned above [44].

The risk of alcohol consumption in developing cancer is elevated when it combined with tobacco smoking; smoking changes the oral bacterial flora, also increases acetaldehyde [46]. The association between tobacco smoking and gastric cancer has been investigated and confirmed by several studies [47, 48], tobacco smoke has been found to have more than 5000 chemical compounds, of which about 93 compounds including PAHs, carbonyls, tobacco specific nitrosamines (e.g. NNN and NNK) and toxic metals, have been identified as harmful and potentially harmful compounds, and most of which are implicated in development of several kinds of cancers due to the activation of the toxicity pathways that lead to these cancers [49–50]. Recent study has shown association between hookah use and gastric cancer [51]. Thus, smokers are considered to have higher incidence of H. pylori infection compared to non-smokers [52]. Stomach cancer risk is 62% higher in male smokers compared with male never-smokers.[53] Stomach cancer risk is 20% higher in female smokers compared with female never-smokers.[53] Risk is higher in smokers for both cardia and non-cardia stomach cancer, and increases with number of cigarettes smoked per day.[54] Smokeless tobacco is not associated with stomach cancer risk.[55]. The mechanisms underlying higher gastric cancer risk for smokers are incompletely elucidated. Tobacco carcinogens may directly damage the gastric mucosa and, indirectly, smoking may favor H. pylori infection persistence and diminish efficacy of anti-H. pylori eradication treatment. [56, 57, 58]. Alternatively, the interaction of smoking with EBV-positive gastric cancer may be mediated by EBV reactivation. Cigarette smoke extract induces EBV reactivation in the EBV-positive cell lines Akata and B95-8 [59]. Smoking is also associated with risk of NPC, another EBV-associated malignancy, as well as with immunoglobulin A antibodies to the EBV viral capsid antigen in subjects without NPC [59]. In addition, smoking is

associated with risk of EBV-positive, although not EBV-negative, Hodgkin lymphoma The very spicy diet was found in 4.36% of cases [60].

Excess salt was found in 4.36% of cases in our series. The raw salt contains a high level of nitrates, its significant consumption more than 6 g per day, would be associated with a decrease in gastric acidity and a high frequency of gastric atrophy, thus creating a favorable environment for the development of Helicobacter Pylori [1]. *Helicobacter pylori* (HP), infection is one of the main predisposing factors for gastric cancer development. High salt intake increases the colonization by HP and induces mucosal damage on persistent HP infection [61, 62]. A number of experimental studies addressed the question of the possible mechanisms of the adverse effect of excess salt intake toward susceptibility to gastric cancer. A powerful interaction has been detected between excess salt intake and HP infection, with high salt intake increasing the rate of colonization of the gastric mucosa by HP, enhancing surface mucous cells, and reducing gland mucous cell mucin [61, 63]. A study in rats showed that high dietary salt intake reduced cell yield and produced an increase in the number of S phase cells, susceptible to mutagenesis. In the same species, salt administration induced dose-dependent damage of the surface mucous cell layer and an increase in replicative DNA synthesis [61]. Moreover, in gerbils with HP infection, high dietary salt up-regulated the expression of COX-2 and iNOS, potentiated the effects of HP infection, and caused gastric cancer progression [64, 65]. High salt intake was found to potentiate CagA expression (HP gene), increase the capacity of this gene to translocate into gastric epithelial cells, and improve the capacity of HP to alter the function of epithelial cells [66]. In addition, both hypergastrinemia induced by high salt intake in the presence of HP infection and the synergic effect of this chronic hypergastrinemia and HP infection may contribute to parietal cell loss and gastric cancer progression [63, 67]. Elevated salt intake may promote and/or enhance the effect of food-derived carcinogens, for example N-nitroso compounds , potent carcinogen that may induce tumors in several sites , by affecting the viscosity of the protective mucous barrier and damaging the gastric epithelium [61, 63].Some experimental investigations on animal models showed a synergistic effect of high salt intake and chemical carcinogens (MNNG and MNU) in the development of gastric cancer [61, 68].

The very spicy diet was found in 4.36% of cases. A high level of spicy food intake was significantly associated with cancer risk [69]. Several possible underlying mechanisms may link the consumption of spicy food and the incidence of cancer. Capsaicin is a primary pungent and irritating agent found in chilies and red peppers, which are widely used as spices in many cultures worldwide.[70] Several animal studies have shown a carcinogenic dose–effect relationship. For example, chili extract has been shown to promote the development of stomach and liver tumors in BALB/c mice initiated by methyl (acetoxymethyl) nitrosamine and benzene hexachloride. Capsaicin also has a cocarcinogenic effect on TPA-promoted skin carcinogenesis in vivo ; this is mediated through the transient receptor potential vanilloid subfamily number 1 and the tyrosine kinase epidermal growth factor receptor. In the present meta-analysis, 19 studies indicated that high-level consumption of capsaicin-containing foods was associated with an increased risk of cancer [69].

The gastric cancers sit more frequently in the zones of mucous junction in the prepyloric region, in the antrum and the small curvature. The antral location represents 60%. This percentage matches that of our serie]s which was 52.67%. This frequency is similar to that found in Yaoundé in 2015 by Ankouane or 52.2% [5]. But it is lower than that found in Togo in 2015 (72%) [71]. In contrast, Tounkara in 2012 in Mali [72] and Sawadogo A et al in Burkina Faso 2000 found a predominance of antro-pyloric localization with 84.04% and 77%, respectively [73].

In Africa, we find a predominance of the distal (antral and antro-pyloric) localization of these cancers. This predominance of distal location would be related to the prevalence of H. pylori

infection. According to some authors, the decrease in the incidence of distal cancers is not due to a single factor, but to the interaction of several factors: the improvement of eating habits and methods of preserving food [74].

In our study, the ulcer aspect was the most represented macroscopic variant with 40.38%. This result is different from that of Diarra M [45] in Mali in 2005 where it is rather the budding ulcer aspect which predominates with 83.4% .However, in our study, fibroscopy evoked malignancy in all of our patients. This can be explained by the late stage at which most patients are seen.

The type of sampling was in 84.18% of the anatomical specimens subjected to histological analysis were from the biopsies and 15.82% fragments came from the operative parts. This result is similar to that of a Malian study where 94.6% of biopsies were found [75]. This predominance is explained by the fact that in the case of symptoms of gastric cancer, endoscopy with biopsies is the first examination required. Unfortunately these biopsies are made at an advanced stage of the disease.

In our series, 419 cases of adenocarcinoma (ADK) were found, ie 72.99% histological type most represented. This result is similar to that reported by Ankouane et al in Yaoundé in 2015, ie 60% of adenocarcinoma [5]. According to the Lauren and WHO classifications, 169 cases of adenocarcinoma were identified, the intestinal type being the most represented with 164 cases (Papillary 43, mucinous: 49 Tubular: 42). On the other hand 5 cases of diffuse type were found. We did not find any precision in 250 adenocarcinomas. This result is similar to the one that Ankouane et al found in Yaoundé 25 cases / 36 adenocarcinomas 69.44% intestinal type [71]. In Africa, our results are lower than those of Bouglouga et al in Togo, which in 2015 reported 94% adenocarcinoma but the total number was only 32cancers of the stomach [71]. We also identified 15.67% of Kaposi's sarcoma, this result is similar to that of Djomou et al which found 19.69% or 76 cases [76]. 9.58% of lymphomas were found this is consistent with results found in Togo [71] or 6% of lymphomas. And other histological types collected more rare with less than 1% of frequency.

In our series, the analysis of adenocarcinoma differentiation was found in 133 cases.79 cases of adenocarcinoma were well differentiated, 51 cases were moderately differentiated and 33 cases were poorly differentiated. These results are different from those found by Touhami [75] and Afifa [77] for whom the well-differentiated type was the most represented respectively 27% and 17.48%.

CONSENT

It is not applicable.

ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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