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Concurrent Cancer of the Oesophagus and Stomach

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Abstract: 557 patients with oesophageal carcinoma, who were studied, revealed 16 cases (2.87%) of synchronous Gastric carcinoma.13 cases (2.33%) were diagnosed preoperatively by endoscopy and Biopsy. Two cases (0.36%) who had so much stenosed cancer oesophagus that the endoscope could not be passed through into the stomach, these two were found histologically cancer in the stomach resected with the oesophagus, one case of carcinoma oesophagus who had endoscopically normal stomach was found histologically cancer in stomach resected with the oesophagus. Therefore intra operative examination and postoperative histology of the resected segment of the stomach should be done in such cases.

Introduction

Multiple primary cases have been reported in many papers 1 & 2 gastric and oesophagial cancers can be associated reported by Maeta et al. It is possible to detect gastric cancer at their earliest stages because of improvement of diagnostic procedures. Gastric cancers can be concomittent with oesophageal cancers in 20% cases reported by Nakayamo & Abo.The high oesophageal cancers was also reported by Okudaira at al. The stomach is the organ which plays the most important role in restoring oesophageal continuity after oesophageal resection for malignancy; but the operation is not a straight forward in their concurrent cancers.

We hereby report on the diagnostic and pathological features of lesions in 16 patients with concurrent oesophageal and gastric carcinomas. The biological differences between oesophageal and gastric carcinomas have been discussed.

Materials and Methods

Between 1983 to 2001, 557 patients of oesophageal carcinoma were examined in the Department of Surgery, Chittagong Medical

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College (CHC) and Hospital, and Bangabandhu Memorial Hospital (BBMH) of University of Science and Technology, Chittagong (USTC). History, clinical findings, endoscopy, biopsy and barium meal examination findings were analysed.

Among 557 cases 16 (2.87%) had synchronous oesophageal and gastric carcinoma, 13(2.33%) cases were not operated as they required oesophagectomy and gastrectomy and continuity of the gullet and gut were not technically feasible.

Two patients were operated as preoperative gastric carcinoma were not diagnosed. In ten cases including these two patients the Gastrofileroscope could not be inserted into the stomach for examination, due to oesophageal stenosis caused by carcinoma.

In these two cases cancers were found in the resected segment of the stomach histopathgologically.

In one case carcinoma oesophagus was seen endoscopically and cofirmed by histology, but stomach was found normal endoscopically. In this case gastric carcinoma was found incidently in the stomach resected with the oesophagus.

Results

The findings of 16 patients are summarized in table one.They were 10 men and 6 women ranging from in age from 40 to 72 years (median 56 years). Dysphagia was the most common symptom in 10 patients, mild dysphagia and sticking of food in the gullet in 5 patients, and epigastric pain and vomiting immediately after food in 1 case. All 16 oesophageal cancer were histologically diagnosed by endoscopic biopsy.

Concomitant oesophageal and gastric carcinoma were diagnosed in 13 cases preoperatively and 2 cases postoperatively. In the later 2 cases preoperative. Endoscopic examination of the stomach was impossible due to oesophageal stenosis caused by carcinoma. In another case gastric carcinoma was diagnosed incidentally in the stomach resected with the oesophagus on postoperative histopathological examination.

Histologically all gastric cancers exhibited adenocarcinoma and the oesophageal cancers were squamous cell carcinoma. All the oesophageal cancers were in an advanced stage. The histological characteristics of gastric and oesophageal cancers were summarized in table 2.

Table-1: Clinical details and detection of Oesophageal and Gastric carcinoma

Serial No	Age	Sex	Symptom	First detection of cancer	Detection of cancer of Stomach by	
					Barium Meal	Endos copy
1	60	M	Dysphagia	Oesophagus	+	+
2	43	M	Epigastric pain&vomitting	Stomach	+	+
3	72	M	Dysphagia	Oesophagus	+	+
4	48	F	Dysphagia	Oesophagus	-	+
5	72	M	Dysphagia	Oesophagus	+	+
6	52	F	Food stuck in gullet	Oesophagus	+	+
7	47	M	Dysphagia	Oesophagus	+	-
8	55	F	Dysphagia	Oesophagus	+	+
9	59	M	Dysphagia	Oesophagus	+	+
10	58	F	Food stuck in gullet	Oesophagus	+	+
11	60	M	Dysphagia	Oesophagus	-	+
12	63	F	Dysphagia	Oesophagus	+	+
13	66	M	Food stuck in gullet	Oesophagus	+	+
14	69	M	Food stuck in gullet	Oesophagus	Inconclusive	Could not be done
15	40	M	Dysphagia	Oesophagus	Inconclusive	Could not be done
16	65	M	Food stuck in gullet	Oesophagus	+	+

Table-2: Histopathological characteristics of Gastric and Oesophageal cancer

No.	Gastric cancer		Oesophageal cancer	
	Location in Stomach	Histology Differentiation of Adenocarcinoma	Location in Oesophagus	Histology Differentiation of Squamous cell carcinoma
1	Body	Well	Lower 1/3	Poorly
2	Body	Well	Middle 1/3	Moderately
3	Body	Moderately	Upper 1/3	Moderately
4	Fundus	Well	Middle 1/3	Moderately
5	Antrum	Well	Middle 1/3	Well
6	Antrum	Well	Lower 1/3	Adenocarcinoma
7	Fundus	Poor	Middle 1/3	Moderately
8	Antrum	Well	Middle 1/3	Moderately
9	Body	Well	Middle 1/3	Poorly
10	Antrum	Moderately	Lower 1/3	Poorly
11	Antrum	Well	Middle 1/3	Moderately
12	Fundus	Poor	Middle 1/3	Moderately
13	Antrum	Well	Middle 1/3	Well
14	Antrum	Well	Upper 1/3	Moderately
15	Body	Moderately	Lower 1/3	Moderately
16	Body	Poor	Upper 1/3	Poorly

Discussion

Duel cancers of oesophagus and stomach are not common. The incidence can range from 1.5% reported by Mori et al and 5.2% by Oohara et al to 20% reported by Nakayama and Abo. In our series it is oesophageal cancers start first. It is more likely that the tumour starts first in the oesophagus, but it is not known whether the gastric cancer starts first as denovo. In our study, all oesophageal cancer were more advanced than the gastric cancer.

Advances in the diagnostic technology have improved early detection of the cancers of the stomach and oesophagus. Endoscopy,

biopsy and histology are the most effective way to detect the early stage of gastric and oesophageal carcinoma. It is important that adequate gastric examination be done preoperatively in a case of oesophageal cancer. Endoscopic examination and biopsy of the stomach are not possible in presence of severe stenosis due to advanced oesophageal carcinoma. Barium series are futile in such condition. Hence intraoperative gastric observation is important when endoscopic observation of the stomach cannot be done. If the upper G-I series reveal certain abnormalities one must examine the stomach during operation. Even that malignant cells may be found in the resected segment of the stomach in such condition. In the current study, one gastric carcinoma was found incidentally in the stomach resected with the oesophagus although the stomach was found normal endoscopically. It is obvious that endoscopically normal stomach must have micro-matastasis. Hence terminal ends of the resected segment of the stomach and the oesophagus must be examined histologically.

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Neurofibromatosis with Malignant Transformation

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Neurofibromatosis is one of the common genetic disorders and can affect anyone, regardless of family history, race, gender and ethnic background. We report a case of plexiform neurofibroma with malignant transformation responding to radiotherapy.

Key words: Neurofibromatosis, Plexiform, Malignant transformation.

Introduction

Neurofibromatosis (NF) is an unpredictable inherited autosomal disorder that varies in its medical, physical and psychological manifestations, first discovered by Prof. FD Von Recklinghausen in 1882. A century later in 1982 Ricardi classified it into 7 types. NF type-1 sometimes referred as peripheral NF is a common autosomal dominant genetic disorder (the responsible gene is located on chromosome 17q11.2) with a prevalence of 1 in 3000 to 5000 where there is evidence of multiple skin nodules along with cafe'-au-lait spots anywhere over the skin. NF Type-2 sometimes referred as central NF is less common, occurring 1 in every 40,000 births and is defined by the presence of bilateral acoustic neuromas.The NF Type-2 gene is located on chromosome 22q12. NF Type 3 is of a mixed type. NF Type 4 resembles NF type-2 but may have more neurofibromas. NF Type-5 defined by segmental NF covered by post zygotic somatic mutation. NF Type-6 presents with only café-au-lait spots having no neurofibroma. In type-7 neourofibromas appear late usually after the age of twenty. Whether it is inherited or not is unknown. Histologically neourofibroma is a benign proliferation of nerve sheath cells mainly of Schwann type having spindle shaped and faintly eosinophilic cytoplasm with spindle to oval of nuclei. As both the melanocytes and neurogleal cells are neural crest origin, it is believed that the protein code responsible for their

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proliferation is also a same one. As a result there is excessive growth of melanocytes and neuroglial cells, often referred as Hamartoma. Proliferation of melanocytes is documented by cafe'-au-lait spots, hyperpigmented skin, mucous membrane, iris, choroids and that of neuroglial tissue is manifested externally by nodules and internally by neuromas with pressure effects. Physical manifestations of NF may also include scoliosis, spinal cord and brain tumours, precocious or delayed puberty, visual impairment or blindness, speech impediments, deafness or hearing impairment, balance problems, headache, severe itching, hypertension, pain, vomiting, chronic constipation and diarrhoea. Variability is extremely high between individuals. Many persons have none or only a few symptoms.

Case reports

A 15 years old boy presented with a 4 months history of a rapidly enlarging painful swelling over the left hemi-face, progressive loss of vision in left- eye, weight loss and anorexia. According to his statements this swelling is the enlargement of a small plexiform swelling on lateral aspect of left upper eyelid, which was painless and existing almost in same size since childhood. (Fig :1)

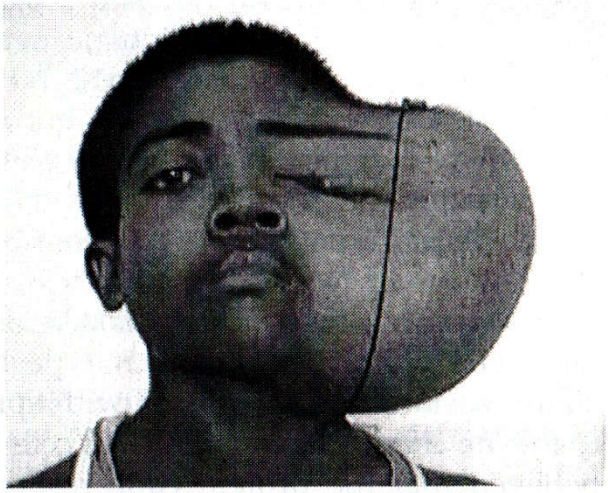

Figure: 1

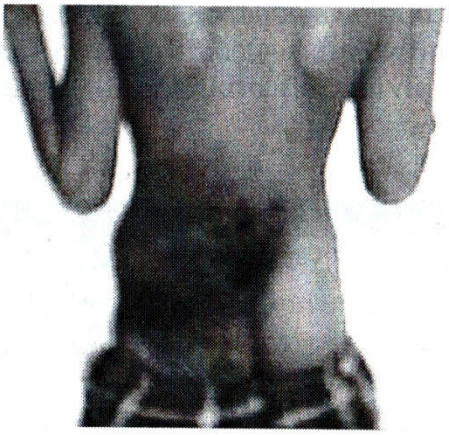

Figure: 2

He has also multiple skin nodules and pigmented patches all over the body since childhood. His father who died at the age of 50 due to other reason not related to this disease had also similar skin nodules and pigmented patches all over the body. On examination he is found to be normotensive, of short stature, and well behaved. There are multiple café-au-lait spots all over the body that are flat, light brown in colour and sharply demarcated, varying in size from a few millimeters to several centimeters. Axillary freckles, which are clusters of small café-au-lait spots. Neurofibromas over the lateral aspect of left upper eyelid, forearm, back of the sacrum are subcutaneous, less well defined and soft in consistency. Plexiform neurofibroma over the sacral region is associated with pigmentation and abnormally excessive in hair growth and distribution. (Fig.: 2) The hugely enlarged, slightly tender, swelling of left hemi face extending up to the parieto-occipital region is almost spherical in shape measuring about 30 cm x 25cm x 15cm in size. (Fig.: 1). It is soft in consistency, well outlined having smooth shiny surface and slightly moveable in both directions. Transillumination and fluctuation tests are negative. A large bone defect is palpated over the right parieto-occipital region .No bruit is present over the swelling. Ocular examination reveals pthysis bulbi of left eye, the palpebral fissures of which was pulled and elongated in a S-shaped manner. On right eye examination visual acuity, PL, PR, and tonometry are within normal limit. In addition to this there are multiple iris lisch nodules, which are sharply demarcated. There is also a dome shaped excrescence over the iris. On blood examination there is leucocytosis, ESR is 100mm in first

hour, USG of abdomen reveals no abnormality, X- ray chest is normal. X-ray skull shows absence of flat bone in occipital region (Figure: 3, between two arrows). At the base of the swelling there is bony erosions involving left orbital wall, zygomatic & sphenoid bones. (Figure: 3)

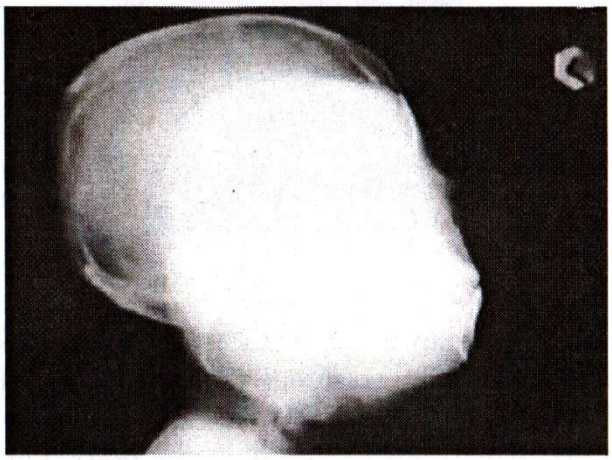

Figure 3: Flat bony defect. (Arrow)

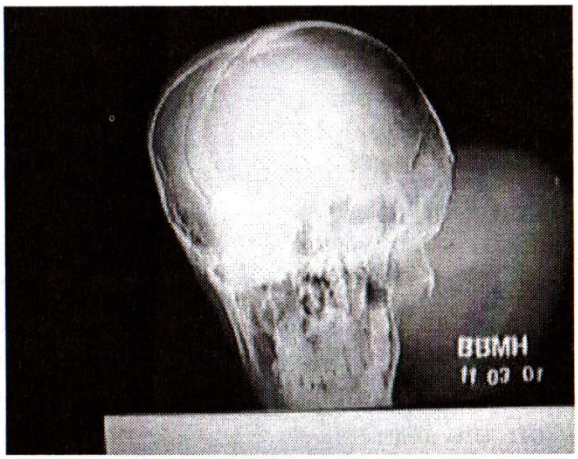

Figure 4: Bony erosion & soft-tissue shadow

Wedge incision biopsy from the core of the growth including the overlying skin revealed moderately differentiated anaplastic spindle cells with proliferation of vessels, which indicates malignant transformation of the tumour. He underwent a course of radiotherapy amounting to 4000 rads in fractionate doses for a period of six weeks as he was referred to oncologist. This results in a

significant reduction in the size of the bulk of the tumour facilitating a possibility of total debulking of the lump followed by craniofacial reconstructive surgery, which may prolong the period of survival rate.

Discussion

In the past NF was often diagnosed solely on the basis of multiple skin nodules and Café-au –Lait spots. Now it is recognized that a few individuals with these findings never develop other stigmata of this disease and may have offsprings with a similarly limited condition suggesting the existence of a genetic disorder distinct for NF-1. Currently NF-1 is diagnosed only when two or more criteria of the following group of signs are fulfilled:

1. Six or more café-au-lait sports > 5mm in diameter in prepubertal individuals or > 15mm diameter in post pubertal individuals.
2. Two or more neourofibromas of any type or one plexiform neourofibroma.
3. Freckling of axillary, inguinal or other intertiginous areas.
4. Optic nerve glioma.
5. Two or more iris lisch nodules.
6. A distinctive osseous lesion, such as flat bone dysplasia or thinning of the long bone cortex with or without pseudoarthrosis.
7. A first-degree relative with NF-1 according to the above criteria.

The discussed case fulfills most of the criteria mentioned above in favour of NF-1. 30% of NF-type-1 undergo malignant transformation which is capable of wide spread metastases. The chance is more in plexiform neurofibroma. 10% plexiform neurofibroma involves face, upper eye-lid and orbit. Involved eye-lid is thicker than normal and causes mechanical ptosis. Greater involvement on its temporal portion causes S-shaped configuration. In the discussed case the plexiform neurofibroma over left eye-lid has transformed into malignancy.

Conclusion

Incidence of NF is quite frequent in general population. The risk of malignant transformation should not be under weighed and those who develop are unfortunate ones. Genetic counseling is the only hope to reduce its incidence in next generation. Perhaps in near future gene therapy could stop this mutant gene responsible for malignant transformation during future innovation.

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