Guillain-Barré syndrome (GBS) is a rare manifestation of acute hepatitis B virus infection. In most cases, the event occurs simultaneously or after the onset of icterus; rarely it is preicteric. GBS occurring with antecedent HBV infection is exceedingly rare.

A 56-year-old man presented with sudden-onset weakness and mild numbness in both lower limbs; 4 days following low grade fever that lasted 2 days. There were no prodromal features. There was no other neurological symptom, jaundice, diarrhea, heavy metal poisoning, risk factors for viral hepatitis, or immunosuppression. He was non-diabetic and a teetotaller.

Examination revealed stable vitals and no icterus or organomegaly. Cranial nerves were normal. There was symmetric flaccid weakness of both lower limbs, greater distally, with areflexia and without fasciculation or atrophy. There was mild diminution in touch, pain, vibration and position sense distally in the lower limbs. Plantar reflex was flexor bilaterally and breathing was normal. Upper limb examination was normal.

Investigations: Hemogram, blood sugar and renal profile were normal; serum bilirubin was 0.8 mg/dL, proteins 6.6 g/dL (albumin 3.3), AST 154 IU/mL, ALT 434 IU/mL (normal up to 35), alkaline phosphatase 504 IU/mL (normal up to 170). Prothrombin time was 16 s (control 11). HBsAg and IgM anti-HBc (ELISA) and HBV DNA were positive. Serology for HAV, HBV, HCV, HIV, Epstein-Barr virus and ANF were negative. Ultrasonography revealed mild hepatomegaly without splenomegaly or ascites, and normal portal vein diameter. Upper GI endoscopy was normal. X-ray chest, cervical and lumber sacral spine were normal. Cerebrospinal fluid (CSF) revealed 5 cells per cu mm (all lymphocytes), protein 70 mg/dL, sugar 74 mg/dL and negative culture and AFB smear; HBsAg was positive in CSF. Nerve conduction study was suggestive of demyelinating axonal neuropathy involving bilateral peroneal, posterior tibial and sural nerves with subclinical involvement of left median and ulnar nerves. F waves were absent in lower limbs and normal in upper limbs.

The patient was managed conservatively and recovered strength in his legs over 1 month. He never had clinical icterus through the course of his illness. At discharge, he could walk by himself. After 2½ months, he remains HBsAg-positive although liver enzymes and prothrombin time have normalized.

In the pre-HBsAg era, the incidence of viral hepatitis was reported as 1% in a large series of patients with GBS. Our case is unique in that the patient never had any prodrome or icterus. Most cases reported have recovered from their paralyses.

The mechanism by which HBV causes GBS is conjectural. Postulates include molecular mimicry between HBV DNA and myelin basic protein with host immunity attacking both, HBsAg-mediated immune complex vasculitis, or direct damage by HBV. HBsAg has been demonstrated in CSF, as in our case.

We recommend routine testing of liver chemistry and HBsAg in all cases of GBS.

References

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Multiple squamous cell carcinomas of esophagus

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Multiple tumors of the esophagus are reported only rarely. We describe three patients with double carcinoma of esophagus. They developed a second squamous cell carcinoma of esophagus more than 1½ years after external radiation for a primary squamous cell carcinoma at a different site in the esophagus. [Indian J Gastroenterol 2003;22:228-230]

Key words: Carcinoma esophagus: metachronous, radiation-induced, synchronous
these individuals. Shibuya et al. found that the incidence of multiple primary malignant tumors rose from 1.26% in 1958-69 to 4.08% in 1973-78.

Patients with squamous cell carcinoma of the esophagus have increased risk of harboring a second malignant tumor at another site. Only rarely is the second malignancy in the esophagus itself. Most such lesions are synchronous tumors, with features of squamous cell carcinoma. There are also reports with smooth muscle tumors. However, occurrence of a second squamous cell carcinoma of the esophagus after an interval of many years is rare. We report three such cases seen by us, which are among over 650 patients with carcinoma of the esophagus operated on by one of us.

**Case 1:** A 56-year-old woman visited us with progressive dysphagia for four months. She had lost 2.0 Kg weight in the last two months. Physical examination did not reveal any abnormality. Barium swallow revealed a 4-cm polypoidal growth in the middle third of the esophagus, while the rest of the esophagus was normal. At endoscopy the growth was seen to extend from 28 cm to 32 cm. Brush cytology and biopsy revealed a well differentiated squamous cell carcinoma. She received external irradiation (5000 rads over five weeks) with complete symptomatic improvement.

She remained well till four years later when she developed dysphagia again. Endoscopy showed narrowing of the lumen from 25 cm to 28 cm with smooth mucosa, and an ulcerative growth beginning at 38 cm causing partial luminal obstruction. Biopsy and brush cytology from the growth revealed poorly differentiated squamous cell carcinoma, while histology from the proximal region showed esophageitis. She was subjected to esophagectomy, when a tumor (2.5 cm x 3.0 cm) was found localized to the lower end of esophagus (Fig.) with no evidence of local or distant dissemination. Histology showed squamous cell carcinoma with no involvement of resection lines. The site of previous tumor and the intervening esophagus did not reveal evidence of malignancy.

She remained well for another eight years when she again developed dysphagia and was found to have squamous cell carcinoma of the cervical stump. She declined further treatment and died a month later.

**Case 2:** A 46-year-old man had presented to us with progressive dysphagia for six months and recent onset of anorexia and weight loss. He did not have pallor, lymphadenopathy or hepatomegaly. Barium swallow study showed a short irregular stricture with destruction of mucosa in the mid-esophagus. At endoscopy an ulcerated growth was seen at 26 cm to 32 cm from the incisors. Endoscopic biopsy showed features of moderately differentiated squamous cell carcinoma. He received external radiation (5000 rads over five weeks) and had gradual improvement in symptoms. He was being evaluated periodically with barium swallow and/or endoscopy. Endoscopy two years after initial presentation revealed mild narrowing at 27 cm to 32 cm, the distal esophagus was normal. Endoscopic biopsies and brushing showed no evidence of malignancy. He remained asymptomatic for another two years, when he was readmitted with dysphagia since one month.

Barium study showed a stenotic growth at the gastroesophageal junction. At endoscopy an impassable stricture was seen at 38 cm, with gross mucosal irregularity. The proximal esophagus was normal. Brush cytology and biopsies from the lesion revealed well differentiated squamous cell carcinoma. Multiple biopsies from the original tumor site and the intervening segment did not show evidence of malignancy. CT scan showed a 5-cm tumor spreading till the gastroesophageal fat planes. The liver was free of metastases. At laparotomy, the tumor was found to be resectable, with fixity to surrounding structures and spread up to celiac group of lymph nodes. Feeding gastrostomy was done and he received 5000 rads palliative radiotherapy. He died three months later.

**Case 3:** A 45-year-old lady presented with progressive dysphagia and weight loss since three months. There were no positive physical findings. Barium swallow examination revealed a malignant stricture in the middle third of the esophagus. At endoscopy a polypoidal growth was seen from 27 cm to 30 cm with normal distal esophagus. Brush cytology and biopsy showed features of keratinizing squamous cell carcinoma. She received external radiotherapy (5000 rads over five weeks) followed by four courses of combination chemotherapy (vincristine, cyclophosphamide, mitomycin and bleomycin) at monthly intervals. Endoscopy showed complete dissolution of the tumor. Periodic follow up for the next three years with barium swallow and endoscopy showed normal esophagus.

She developed dysphagia again 4½ years later. Barium swallow showed polypoidal growth at 35 cm, occluding the lumen by about 50%. Brush cytology and biopsy showed features of keratinizing squamous cell carcinoma. Ultrasound and CT scan showed the tumor to be localized. At laparotomy, a 6.5-cm-long ulceroproliferative lesion with spread up to submucosa and involvement of lymph nodes around the left gastric artery was found. She underwent transthoracic esophagectomy and cervical esophagogastic anastomosis. Histology of the resected specimen showed squamous cell carcinoma. Pulmonary sections of the proximal 10 cm of esophagus and resection lines did not reveal evidence of malignancy. She remained well for four years till her last follow up.

Synchronous squamous tumors of the esophagus can be explained on the basis of simultaneous widespread carcinomatous transformation or the origin of...
tumor as part of multicentric invasive squamous carcinomatosis. The presence of in-situ carcinoma or severe atypical epithelium contiguous with the invasive carcinoma can be taken as evidence in support of multicentric origin. Because of the peculiar lymphatic drainage of the esophagus, occurrence of multiple malignancies raises a question about submucosal spread of the primary tumor. Malignant cells entering submucosal lymphatics can result in tumor emboli to sites 2 cm or more from the growth, producing the phenomenon of "skip" or "satellite" nodules. Rarely a double carcinoma can be caused by this phenomenon.

Our patients developed a metachronous tumor several years after the first one. There was no evidence of residual tumor at the primary site. We did not undertake ultrastructural characterization of the tumors in any of our patients, nor did we do genetic studies.

All our patients had received external radiation for the primary tumor. Esophageal carcinoma has been rarely seen as a sequel to therapeutic irradiation of adjacent lesions. Of 14 patients with radiation-associated malignancy of esophagus reported, 11 had developed squamous cell carcinoma. Only one patient had received radiotherapy for carcinoma esophagus and he later developed leiomyosarcoma.

Another cause of second squamous cell carcinoma of esophagus is previous gastrectomy. Kitabayashi et al suggested that gastrectomy with subtotal esophagectomy precipitates gastroesophageal reflux, which induces development of squamous dysplasia and then carcinoma. However, such an occurrence is rare.

References

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Gastric leiomyoma presenting as gastric volvulus

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We report a 45-year-old lady who presented with recurrent vomiting, retching and epigastric pain, and was diagnosed to have gastric volvulus. After correction of the volvulus by endoscopic detorsion, a tumor was seen along the greater curvature. This was excised surgically. Histology showed leiomyoma. [Indian J Gastroenterol 2003;22:230-231]

Key words: Gastrointestinal stromal tumor

Pre-disposing factors for gastric volvulus include lax ligamentous attachment of the stomach to surrounding structures, diaphragmatic hernia, eversionation of the left hemidiaphragm, gastric tumor, or masses in adjacent organs. We report a patient with gastric volvulus secondary to gastric leiomyoma.

A 45-year-old lady presented with history of recurrent vomiting, retching and epigastric pain since one year. There was no history of abdominal surgery. On examination she was anemic. There was no lump palpable and no visible peristalsis.

Upper GI endoscopy could not be negotiated beyond mid body of the stomach, and gastric folds appeared twisted. Barium study showed incomplete mesentero-axial gastric volvulus (Fig). At repeat endoscopy the endoscope was negotiated along the gastric lumen and the antrum entered. After the scope was straightened by torque method, with gastric detorsion, a large growth was seen along the greater curvature with two ulcerations on its surface. Barium study was repeated after detorsion (Fig).

Surgery was done three weeks later after correction of anemia. The tumor was excised along with 5 cm of normal gastric wall. The serosa over the growth was normal. There was no regional lymphadenopathy. Histologically the tumor

Fig: Barium study showing gastric volvulus before (left) and after detorsion. Growth is seen along the greater curvature after detorsion.