the discoloration started to resolve. The pulse oximeter now displayed values normally when applied. A Doppler ultrasound revealed a normal study of radial and ulnar arteries and echocardiography did not reveal any vegetation in the heart. The patient was advised low molecular weight heparin for one week as a prophylactic measure and she recovered without any adverse sequel.

Kramer’s algorithm was used to determine whether the clinical manifestation described was an adverse drug reaction. Cleofol had a cumulative score of +3 while the rest of the agents injected through the same intravenous cannula had a score of +1. Thus, Cleofol was established as the most likely cause of this possible adverse drug reaction.

Tissue necrosis following extravasation of propofol has been reported in the literature. We had ruled out direct extravasation in this case. It is known that the kallikrein-kinin system in plasma is activated by contact with propofol to generate bradykinin. This bradykinin acts locally and makes the vein dilated and permeable. Perhaps by this mechanism the constituents of the drug had escaped the venous system and caused damage to the surrounding tissues, thus triggering the oedema and subsequent arterial compromise.

Very high incidence of thrombophlebitis (93%) has been reported following a formulation that did not contain soyabean oil. Cleofol is also known to damage the bivalve used for administration and this has so far not been a problem with the lipid emulsion preparations. Thus, in the case of propofol, a change in formulation seems to alter the safety profile.

The availability of this lipid-free solution avoids bacterial contamination of propofol and is welcome in clinical situations where lipid load is not desirable. But the present report poses a question mark on its safety in clinical anaesthesia.

**Dubey PK, Kumar A**

Department of Anaesthesiology and Critical Care Medicine, Indira Gandhi Institute of Medical Sciences, Patna, India

**Correspondence:** Prakash K. Dubey, E-mail: pkdubey@hotmail.com

**References**


**PubMed ID**: 15793348

---

**Ileal metastases from oesophageal carcinoma causing intestinal obstruction**

Sir,

A 56-year-old male presented with dysphagia. Endoscopy revealed an ulcerative lesion in the lower oesophagus, extending from 30 cm to 35 cm. At surgery, the tumour involved the oesophagus without infiltration of the adjacent structures. A transthoracic oesophagectomy was performed and a tube of the stomach was anastomosed to the oesophagus to restore gastrointestinal continuity. Histopathological examination revealed a squamous carcinoma of the oesophagus with involvement of the paraesophageal and perigastric lymph nodes (pT3N1M0). He received 54 Gray of radiotherapy to the mediastinum postoperatively.

Eight months later, the patient presented with abdominal distension. X-rays of the abdomen suggested small bowel obstruction. A computed tomogram scan of the abdomen showed an ill-defined bowel mass in right sub-hepatic region, with minimal ascites. The liver did not reveal any metastasis. These findings prompted a laparotomy that showed a well-localized abscess in the right paracolic gutter. After draining the abscess and separating the adhered bowel loops, a mass was noticed in the ileum, approximately 6 to 7 cm proximal to the ileocaecal junction, with adhesions to the adjacent small bowel and caecum. This mass was causing stenosis of the ileum here, with proximally dilated bowel. A perforation was also present at the site of the mass. There was no other gross evidence of intra-abdominal disease. The mass was excised and a diverting ileal mucous fistula was fashioned in view of the peritoneal contamination and poor nutritional status.

Histopathological examination of the excised mass revealed keratinized squamous carcinoma involving the full thickness

Figure 1: Tissue section of ileum showing metastatic deposits of squamous carcinoma involving the sub-mucosa. (Tumour was also present in the muscularis propria.) H/E, x10

---
of the ileal wall (Figure 1). The suspicious omental areas which were resected showed non-specific inflammation, and the peritoneal fluid revealed pus cells and Escherichia coli on culture; however, no malignant cells were seen.

In view of the metastatic disease and the poor general condition of the patient, no further adjuvant treatment was offered. At an eight-month follow-up, the patient is alive and asymptomatic and has learnt to manage his ileostoma well.

While the small intestine accounts for nearly 75% of the total length of the GI tract and more than 90% of the mucosal surface area, less than 25% of all alimentary tract neoplasms and less than 2% of all malignant tumours originate from the small intestine.[1] This could be due to the relatively rapid transit time, liquid content of stools, low bacterial population, and a high concentration of IgA within the lumen.[1] Metastatic tumours of the small bowel outnumber the primary tumours and usually occur as part of generalised peritoneal carcinomatosis.[4] Rarely are these metastatic tumours solitary. The different mechanisms postulated for isolated small bowel metastasis include retrograde lymphatic spread following blockade of paraaortic or mediastinal lymph nodes, lymphatic embolisation, haematogenous spread or peritoneal seeding, including direct implantation.[2] The ileum is the commonest site of metastatic lesions in the small intestine, probably because of the large number of Peyer’s patches, which make it the best trapping zone for hematogenic metastasis.[1]

Ileal metastasis of squamous carcinoma may arise from the cervix, lung and other sites, but metastasis from oesophageal carcinoma is rare.[5] Although equally uncommon, the possibility of a primary squamous cell carcinoma of the ileum was excluded since these usually arise in the presence of bowel duplication, and more importantly, the patient already had a primary tumour in the oesophagus.[1,5]

Despite the inevitable concern of finding generalised abdominal carcinomatosis, laparotomy is indicated to relieve intestinal obstruction, either by a palliative intestinal resection or by a bypass surgery.[24] Although the overall prognosis is poor, significant palliation or occasionally, long-term survival may be achieved in the absence of disseminated disease.[23]

**Neve RS, Qureshi SS, Mistry RC**
Department of Thoracic Services, Tata Memorial Hospital, Parel, Bombay - 400012, India

**Correspondence:**
Dr. Rajesh Mistry, E-mail: musica@vsnl.com

**References**


**Severe autonomic dysfunction as a presenting feature of Wilson’s disease**

Sir,

Wilson’s disease (WD) is known for its protean clinical manifestations. Though neuropsychiatric symptoms are common in WD, autonomic nervous system involvement has received inadequate attention. However, few recent reports describe the clinical and electrophysiological features of autonomic involvement in WD.[1,2] We describe a young man, initially presenting with features of severe autonomic dysfunction with the diagnosis of WD being made later, when he developed dystonia and Parkinsonism. The frequency of autonomic dysfunction in WD and its possible mechanisms are discussed.

28-year-old man presented with history of postural giddiness, impaired sweating, palpitations, urgency of micturition and erectile dysfunction for four months. He had two episodes of syncopal attacks on rising up from recumbent position. On examination, optic fundi, motor, sensory, and cerebellar systems were normal. Detailed bedside autonomic function tests were abnormal (Table 1). Investigations showed normal haemogram and biochemistry. Detailed motor and sensory nerve conduction studies were normal. However, sympathetic skin responses (SSRs) were absent in all four limbs. Upper gastrointestinal endoscopy performed after an overnight fasting showed residual food particles in the stomach suggestive of gastroparesis.

As there was no obvious cause for dysautonomia, he was symptomatically treated with fludrocortisone. Two months later, he had persistent autonomic symptoms; in addition, he had developed dystonia of hands and feet, tremors of hands and bradykinesia. WD was suspected and confirmed on finding a Kayser-Fleischer ring in cornea and serum ceruloplasmin of 8 mg% (Normal >60 mg%). MRI of brain showed symmetrical T2-weighted hyperintense lesions in lentiform nuclei. He improved with d-penicillamine and was asymptomatic at 6-month follow up. Autonomic function tests also improved (Table 1). The SSRs normalized.

Common neurological manifestations of WD include dysthria, dystonia, silly smile, tremors, Parkinsonism, cognitive impairment and cerebellar ataxia.[4] Autonomic dysfunction is not commonly thought to be a manifestation of WD. However, literature review reveals evidence to the contrary. In a prospective study evaluating cardiac involvement in WD, 34% patients had electrocardiographic abnormalities.[1] 20% had orthostatic hypotension and one-third had abnormal response to Valsalva maneuver. In another study, autonomic nervous system involvement was assessed with the help of SSRs and RR interval variation. SSRs were abnormal in 52%. However, parasympathetic function as assessed with RR interval variation in Valsalva maneuver was abnormal in only 3 patients.