SUMMARY
Percutaneous Endoscopic Gastrostomy (PEG) tubes were often offered to patients requiring long term enteral feeding. Even though the procedure is relatively safe, it is associated with various complications such as peritonitis or even death. We presented a case of a 54-year-old gentleman with underlying ischemic stroke and pus discharges from a recently inserted PEG tube. Computed Topography (CT) scan confirmed abdominal wall necrotising fasciitis complicated with hyperosmolar hyperglycaemia state (HHS) and later succumbed after 48 hours of admission. Our case illustrated the rare complication related to the insertion of PEG tube; abdominal wall necrotising fasciitis that was associated with mortality.

INTRODUCTION
Percutaneous Endoscopic Gastrostomy (PEG) was first introduced in 1980 and has been an essential armament for physicians in treating chronic illness patients. The procedure for PEG tube insertion is relatively brisk without the need for general anaesthesia and is cost effective comparing to other means of enteral feeding. With easy accessibility, it has been on demand for patients with swallowing disability such as stroke, Parkinson disease and others. However, the procedure is marginally associated with variable complications and our case illustrated a rare complication of abdominal wall necrotising fasciitis, which is often associated with poor prognosis.

CASE REPORT
A 54-year-old man with underlying hypertension and diabetes mellitus presented with right sided hemiparesis, global aphasia and a computed topography (CT) brain revealed left frontal, pons and parietal infarcts. His initial admission was complicated by aspiration pneumonia and assessment by speech and language therapist indicated he was experiencing silent aspiration. Thus, he was commenced on nasogastric feeding. Due to recurrent aspiration pneumonia, enteral feeding via PEG was recommended and the procedure was performed uneventfully.

Two weeks post PEG tube insertion, he was down with fever, reduced consciousness and pus discharges from the PEG’s site. Family members denied the PEG tube was ever pulled or dislodged. The discharges were noted a week post procedure and was treated with a course of oral antibiotic by a general practitioner. Despite completing the course of antibiotic, his condition worsened with continuous foul smelling discharge that warranted hospitalisation. Nonetheless, the PEG tube remained functional and intact since insertion.

Clinical examination revealed a septic looking patient with temperature of 38.5°C and hypotensive at 88/48 mmHg. Dextrostix was 58 mmol/L but no ketone was noted from the urinalysis.

On abdominal examination, the PEG tube was in situ but surrounded by erythema, pus and foul smelling discharges. No tenderness was elucidated.

Initial blood investigations showed white cell count elevated at 12.7 x 10^9/L, C-reactive protein was raised to 8.51 mg/dl with creatinine and urea elevated to 228.1 µmol/L and 43.6 mmol/L respectively; indicating acute kidney injury. Random blood sugar was 51.1 mmol/L with serum osmolarity of 433 mOsm/kg. Arterial blood gas indicated metabolic acidosis with pH of 7.326, HCo3 of 20.4 mmol/L and pCO2 of 39.6 mmHg.

Initial abdominal imaging on ultrasound (Figure 1) showed presence of subcutaneous and intra-abdominal collection surrounding the PEG site. Subsequent CT abdomen (Figure 2) indicated extensive hypodense collection with peripheral enhancement within the subcutaneous and intra-muscular layer extending from the left hypochondrium to the left lumbar region. Associated air pockets were seen. The collection measured 4.1 x 12.2 x 14.3 cm.

Considering the clinical, biochemical and radiological findings, he was diagnosed with septicaemic shock secondary to abdominal wall necrotising fasciitis. It was complicated with hyperosmolar hyperglycaemia state and acute kidney injury.
Intravenous Piperacillin/Tazobactam was initiated together with aggressive fluid resuscitation. Insulin infusion was commenced with close monitoring of his dextrostix. The PEG tube was removed and the patient was planned for the ultrasound-guided drainage of the subcutaneous collection. However, his condition deteriorated and died less than 48 hours post presentation.

**DISCUSSION**

Percutaneous Endoscopic Gastrostomy (PEG) insertion is a relatively simple procedure and should be considered for long-term enteral feeding. However, Lockett et al. have demonstrated that PEG tube insertion may lead to various complications such as bowel perforation, peritonitis, leakage and local infection or abscess. Furthermore, these complications were recorded as high as 16.3% with 0.6% risk of death during the procedure. Approximately 7.8% of patients died after 30 days post PEG procedure. The frailty and multiple co-morbidities of patients requiring PEG contributed to the increased risk for complications.

A rare but severe complication post PEG tube insertion is abdominal wall necrotising fasciitis, often conferring poor prognosis. Maclean et al. illustrated a high mortality rate of 33.3% due to PEG related necrotising fasciitis. Higher mortality rate was observed with underlying diabetes mellitus, malnutrition, immunosuppressed patients, old age, frequently dislodged PEG tube and impaired kidney or liver function.

In term of microbiological data, Rolston et al. reported that the most common organisms associated with PEG tube sites infection were Staphylococcus aureus, Candida species and Pseudomonas aeruginosa with 55% of cases caused by Gram positive organisms. It also highlighted the incubation of Gram-negative organism such as Pseudomonas aeruginosa during polymicrobial infection. Moreover, out of 37% polymicrobial organisms isolated from PEG tube infection, 17% of those were methicillin resistant organisms. Therefore, treatment may pose a challenge with the increasing incidence of polymicrobial infection and methicillin resistant organism. Hence, broad-spectrum antibiotics either high dose penicillin or third generation cephalosporin together with anaerobic cover such as metronidazole were suggested for empirical treatment.

In addition, rapid surgical intervention with debridement of necrotic tissue together and soft tissue reconstruction should be considered, since it is associated with better outcome.
Urgent operative debridement, even for patients in septic shock, should be considered within hours of presentation. However, resuscitation of the patient is paramount prior and during surgery. Ensuring adequate nutritional support with increased calorie intake is recommended due to high catabolic state associated with the infection.

In terms of prevention, the current accepted practice is to infuse third generation cephalosporin prior to PEG tube insertion as a prophylactic measure. This practice originated from the observation that only 5.4% of patients with prophylactic antibiotics developed peristomal infection compared to 38.5% patients without prophylaxis. However, our patient still developed abdominal wall necrotising fasciitis despite prophylactic measures. This may be explained by his diabetes mellitus (HbA1c of 12.6%), which contributed to increase susceptibility to infection and sepsis.

CONCLUSION
Post PEG insertion abdominal wall necrotising fasciitis is a rare complication with devastating outcome. Greater awareness, prompt intervention and a multidisciplinary approach involving medical practitioners, surgeons, nursing staff and dieticians are required to improve patient care. Even though the procedure is relatively safe, prophylactic measures helps to prevent complicated outcome.

REFERENCES