Letters

STAPHYLOCOCCUS AUREUS ENTEROTOXINS IN PEOPLE WITH CYSTIC FIBROSIS (CF)

Editor,

Staphylococcus aureus (SA) is a Gram-positive bacterium, which produces several enterotoxins inducing nausea. We hypothesize that patients with cystic fibrosis (CF) who are chronically infected with enterotoxin-producing SA in their airways may expectorate sputum containing enterotoxins, especially during sleeping, which may be ingested subsequently leading to nausea. Therefore, we wished to examine if SA isolates obtained from CF sputum are enterotoxin-producers, which have the potential to cause nausea in their host.

We examined 16 clinical SA isolate from sputum of CF patients (n=16), who were infected with SA. SA cultures were examined for enterotoxins A, B, C, D and E by ELISA assay, in accordance with the manufacturer’s instructions (RIDASCREEN® SET Total (R-Biopharm AG, Darmstadt, Germany)). Of these, 10 (62.5%) isolates were positive for at least one enterotoxin, with the remaining six isolates negative for enterotoxin(s). There was no statistically significant difference (p=0.8) in lung function (FEV1%) between patients chronically/intermittently colonised with enterotoxin-producing Enterotoxigenic SA strains and non-enterotoxin-producing SA strains (Figure 1).

Figure 1: Comparison of lung function (%FEV1) in patients with cystic fibrosis infected with enterotoxin-producing Staphylococcus aureus and non-enterotoxin-producing Staphylococcus aureus (p=0.82)

The exact amount of SEs required to produce emesis is not specific, largely due to individual variations in sensitivity to enterotoxins, however a study looking at a staphylococcal gastroenteritis outbreak discovered that doses of around 0.1μg of SEA were sufficient to produce nausea. A review in 2012 found that most studies quoted the total amount of SEs required for symptomatic gastroenteritis to occur to be around 0.1μg, however one exception suggested the figure to be as high as 10-20μg. In terms of numbers of enterotoxin-producing SA required to produce GI symptoms including nausea, this has been estimated to be circa 10^5 colony forming units (CFUs) per gram of food. In the CF lung, previous work from our group has shown that with chronic SA infection, the mean number of organisms is 1.01 x 10^7 CFU per gram of sputum.

In our CF population, 46.1% of adults and 44.7% of children are infected/colonised with SA. Data from the current study demonstrated that 62.5% of SA isolates were enterotoxin producers, equating to an occurrence of 28.8% and 27.9% SA enterotoxin-producers in adults and children, respectively. Interestingly, a study of 48 SA isolates from young and healthy Irish students between 1995 and 2004 found that 66.7% of isolates harboured the classical SE genes (SEA – SEE).

Nausea in CF patients can be associated with several aetiologies, including distal intestinal obstruction syndrome (DIOS), chronic inflammation of the GI tract and antibiotic usage, as well as other less frequent causes of nausea, such as eosinophilic esophagitis. Given the relatively high occurrence of SA from sputum in the CF population, the high occurrence of enterotoxin producers within these SA isolates, combined with the frequent reporting of nausea, we are now exploring if this could be contributing to nausea in our CF patient population. Further work is now required to determine the stability of SA enterotoxins produced in the CF lung, particularly their persistence against denaturation by the proteolytic environment within the lung, including their intact passage from the lungs into the GI tract.

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OESOPHAGITIS DISSECANS SUPERFICIALIS – AN UNUSUAL ENDOSCOPIC FINDING

Editor,

Oesophagitis dissecans superficialis (ODS) is a desquamative oesophageal disorder, characterised by sheets of sloughed squamous tissue with normal underlying mucosa. It is extremely rare and benign. We describe a case of ODS and discuss the condition.

An 83-year-old female was admitted to hospital with a 4 day history of vomiting and central cramping abdominal pain.

On abdominal examination, there was epigastric tenderness with intermittent guarding. Abdominal radiograph showed faecal loading.

The impression was gastritis and constipation. A Computed Tomography scan of the abdomen and pelvis was carried out, due to suspicion of ischaemic bowel and showed no acute intra-abdominal pathology. The scan report noted that the stomach fundus appeared slightly thick-walled and advised an oesophago-gastro-duodenoscopy (OGD).

The OGD showed ODS in the oesophagus (Figure 1), and a small hiatus hernia. The stomach and duodenum appeared normal. Biopsies were taken. The oesophagus showed patchy acute mild inflammation with epithelial hyperplasia and parakeratosis. Periodic acid-Schiff stain showed scattered Candida organisms. The gastric body mucosa showed some cystic dilatation of glands suggestive of a fundic-type polyph, with no evidence of dysplasia.

The patient was prescribed laxatives and anti-emetics. Over several days, her nausea and constipation resolved.

ODS is a desquamative oesophageal disorder, involving sloughing of the superficial mucosa. It is extremely rare, with one study reporting an incidence of 0.03%. It usually affects adults after age 50 and is slightly more common in women than men.

ODS can be idiopathic or secondary to oesophageal mucosal injury which may be due to bisphosphonates and non-steroidal anti-inflammatory medications, certain foods, or repeated vomiting. It is also associated with systemic diseases, such as pemphigus vulgaris and coeliac disease. In this case, the patient was not taking any associated medications and did not have any associated systemic diseases.

It is usually asymptomatic and discovered incidentally, which was likely to be the case in our patient. It can occasionally be associated with dysphagia, nausea, bleeding, vomiting, heartburn, epigastric pain, and odynophagia. The abdominal pain in our patient’s case was felt more likely to be due to constipation rather than her ODS, as the pain improved following successful laxative use.

It has been suggested that meeting 3 of the following endoscopic criteria is consistent with ODS: “(1) strip(s) of sloughed oesophageal mucosa >2cm in length; (2) normal underlying oesophageal mucosa; and (3) lack of ulcerations or friability of immediately adjacent oesophageal mucosa.” Biopsies are not always necessary, but should be performed if the patient is symptomatic, a coexisting diagnosis may be present, or the endoscopic features are not classical.

The most common histological findings are parakeratosis and intraepithelial splitting, although these are non-specific. Biopsies may show inflammation, and there may be associated fungal elements. In our patient’s case, Candida was noted.

Whilst there are no clear guidelines for the management of ODS, it has been reported that stopping any potential causative medications and use of acid-suppressing medications results in resolution. ODS is benign and does not cause permanent damage.

It is important to raise awareness of ODS. One study reported that only 41.5% of cases were correctly identified at endoscopy. Gastroenterologists’ unfamiliarity with this condition may cause it to be mistaken for other diseases.