The Milk Vomiting Baby in the First Few Months of Life

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COMMENTS ARTICLE

There are two main diagnoses which come to mind when babies vomit or regularly regurgitate in the first few months of life.

a. Pyloric Stenosis of infancy. (PS)
b. Oesophago-gastric Reflux. (Reflux)

Reflux may be associated with a sliding hiatal hernia. In that event the prognosis may not be benign and oesophageal strictures and prolonged problems are possible. Surgical correction may be required.

Simple reflux without hernia is a benign condition sometimes referred to as (“possetting”) with the babies being described as “happy spitters”. Simple reflex quickly self-cures with time. Up to 70% of babies posset within the first months and by 1 year of age the frequency falls to less than 5%.

The condition is usually diagnosed entirely clinically. There are usually no confirmatory tests. The absence of projectile vomiting and visible peristalsis is often all that is required. Minimal medical treatments with very occasional antacid drugs are sufficient to ensure a time-controlled self-cure.

PS. shares the same time presentation with the same tendency to self-cure. John Thomson in 1921 first described the phenomenon of mild cases of PS in an analysis of 100 cases. Milder cases may not have obvious projectile vomiting or visible peristalsis. The mild cases were described by Dr. Thomson as “not uncommon” and self-cured with only minimal medical treatment (1). Temporary modern antacid drugs would have a particular part to play in facilitating cure (2).

Could both conditions sometimes share the same cause?

All babies develop a peak gastric acidity at around 17 days of life (3). The evidence-based reason is an immaturity of the negative feed-back between gastrin and acid secretion (3). Acidity progresses to a peak until the negative feed-back become established. Thereafter acid secretion becomes under control.

Acidity causes pyloric sphincter contraction; work-hypertrophy and delayed gastric emptying. Is it possible that this phenomenon acts also in the cases of reflux which may be diagnosed by barium studies but, in some cases, will be due to gastric outlet hold-up because of a hypertrophied sphincter. There is suggestive evidence that this might indeed be true.

Forshall in 1955 focused on simple reflux without hiatal hernia. Clinical diagnosis was supplemented by barium studies to confirm simple reflux in 58 babies. More male babies were affected and all self-cured clinically and radiologically with minimal medical treatment.

Almost half had projectile vomiting and one in 5 had visible peristalsis.

14% required pyloromyotomy. 6 of the 8 operated cases were confirmed as PS with 2 having equivocal thickening of the sphincter. This frequency of PS is at least at least 20 times the normal (4).

Thus the doctor faced with a baby vomiting or regurgitating milk in the first few months must find it a very difficult task to properly exclude PS. The treatment of PS, certainly in the progressive case, is radically different with surgery justifiably reigning supreme.

Ultra-sonic assessment of the sphincter is quick and easy and relatively precise.

Why not make this examination mandatory in all non-trivial cases of babies diagnosed with persistent gastro-oesophageal reflux.
REFERENCES