

Complications of 'Slow-K' therapy

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SUMMARY. (1) The failure of 'Slow-K' tablets to disintegrate prevents rapid release but allows them to be trapped by their bulk in the intestine.

(2) Two cases are reported. In the first the tablet was trapped in a caecal diverticulum and the patient developed an abscess. In the second, abdominal pain developed which subsided when 'Slow-K' was stopped. Later 'Slow-K' was again started and the patient developed dysphagia.

(3) The possibility of abdominal complications with this treatment should be remembered.

(4) Effervescent KCl preparations may replace 'Slow-K' but KCl supplementation may be necessary only in cardiac disease.

Introduction

Prescribing potassium chloride (KCl) has been greatly improved by the introduction of 'Slow-K' tablets. Previously, the enteric coating of tablets of potassium chloride rapidly dissolved somewhere in the intestine. The high local concentration of KCl caused severe inflammation, ulceration, stenosis,* and even perforation of the bowel (*The Lancet*).

Sometime, up to many months after starting treatment, post-prandial colicky pains, distension, nausea, and vomiting started (Ashby, 1965). Many patients needed surgical treatment even if the treatment was stopped (Ashby, 1965; Grant, 1972).

Messrs Ciba then partly coated the KCl crystals with inert wax. The tablets were prepared from this mixture, coated with sugar. When the sugar coating dissolved, the KCl was gradually dissolved, leaving behind the spongy, honeycomb-like wax matrix. The tablets did not disintegrate. In simulated intestinal juice it took four hours until full dissolution of the KCl. The enteric-coated tablets totally disappeared after 15 minutes (Wynn, 1965). Metabolic studies showed that KCl was completely absorbed from the 'Slow-K', but not from the enteric-coated tablets (Nordin, 1970; Wardener, 1969; Wynn, 1965). Most other firms have since similarly adopted the slow-release principle. ('Hydrosaluric-K' still has KCl as an enteric-coated core within the sugar-coated tablet).

Sporadic complications were reported during the past ten years, but only in very special circumstances i.e. in the postoperative stage after cardiac surgery. The enlarged left atrium compressed the oesophagus against the spine and held up the 'Slow-K' tablet. A high KCl concentration developed, causing ulceration and stenosis of the oesophagus (Howie, 1975; Chesshyre, 1971).

However, complications may arise in other more common circumstances. The tablets may be trapped further down in the gastrointestinal tract and cause similar damage. The diagnosis may easily be missed if this possibility is not remembered, because these patients are usually taking multiple medication.

Two cases of complication in general practice

During the last year I have observed two patients whose complications may be attributable to 'Slow-K' treatment. In one, the tablet was trapped in a small diverticulum of the caecum. She developed an abscess, diagnosed as an appendix abscess. In the other, severe dysphagia developed, but immediately subsided on stopping the treatment.

First patient

A 49 year-old woman had mitral stenosis with incompetence. On digoxin (0.125 mg daily) she remained symptomless for years. In February 1974 she developed congestive heart failure

* Observed in other of our patients.

due to fibrillation. Frusemide (40 mg daily), 'Slow-K' (1,800 mg daily), 'Eraldin' (200 mg daily) were started.

Soon she began to complain of abdominal discomfort, pain, distension, and diarrhoea. Previously she was rather constipated. She blamed the "new tablets" for her complaints. Her symptoms were widely fluctuating, though persisting.

In August, 1974 the pain became worse. There was some epigastric tenderness, the W.B.C. was 16,800. This raised the suspicion of 'Slow-K' damage, but again she improved and on 12 September 1974, with a W.B.C. 10,300, gastroenteritis was diagnosed.

In October, 1974 the pain became severe and she vomited. There was tenderness in the epigastrium and in the right iliac fossa. The 'Slow-K' was immediately and finally stopped. However, peritoneal infection became clear, (W.B.C.: 14,300) and she was admitted to hospital (25 October 1974), a tender lump became palpable. She was treated with antibiotics as an appendix abscess and improved. The lump disappeared and she was discharged in November 1974.

Although no further antibiotics were given, the pain did not recur. Barium meal and intravenous pyelogram during the following four months were negative. Barium enema showed incomplete filling of the appendix.

In February, 1975 interval appendicectomy was performed. There were adhesions around the appendix which were removed. There was a small diverticulum on the anterior wall of the caecum which contained a faecolith. This was dislodged and the diverticulum invaginated. Her recovery was uneventful.

Since 'Slow-K' was stopped there has not been any recurrence of pain. Histologically the wall of the appendix was normal, without any evidence of subacute infiltration, scarring or increase of connective tissue. This should have been present after an eight months' history of abdominal pain with persistent leucocytosis, if the abscess had been caused by appendicitis.

Comment

Even though the diverticulum was not opened to identify the "faecolith", it is likely that a 'Slow-K' tablet was trapped in the diverticulum and the "faecolith" was its remaining matrix. Within the small space of the diverticulum a high concentration of KCl could easily develop and its thin wall allowed the permeation of the infection into the peritoneum. Adhesions were likely to develop and to be found at the operation whatever the origin of the abscess. Such situations also arise with a leaking duodenal ulcer, cholecystitis, or adnexitis. The abscess was "perityphlitic" (the diverticulum was on the "typhlon").

The conspicuous fluctuation of her symptoms was probably due to only temporary "trapping" of tablets; when they were released from the diverticulum, the symptoms subsided. Symptoms started soon after 'Slow-K' was started and subsided when they were stopped.

Second patient

A 60-year-old man, a heavy drinker had chronic bronchitis with emphysema. He had had a partial gastrectomy for peptic ulcer in 1953. In 1970, he developed cardiac failure due to cor pulmonale. Afterwards he needed hospital admission on numerous occasions. He improved on 'Brinaldix-K' (one tablet daily), digoxin (0.25 mg daily), salbutamol (12 mg daily) and prednisolone (5 mg daily).

In October 1972, he developed ankle oedema when 'Brinaldix-K' was replaced by frusemide (40 mg) and 'Slow-K' (1,800 mg). The oedema disappeared. In January 1973, he developed vague but increasing abdominal pain. The grossly enlarged liver occupied the whole of the epigastrium. Ascites was demonstrated. 'Aldactone-A' (100 mg) was started. In April 1973, a barium meal was reported. There was a circumscribed dilation of the oesophagus proximal to a stenosed area in its lower third where tertiary peristaltic waves could be seen. No hiatus hernia or reflux was demonstrated. The patient had no dysphagia. He complained only of "heartburn", relieved by 'Aludrox'. In April 1974, he was admitted to hospital for pneumonia when tricuspid incompetence was also diagnosed. 'Slow-K' was stopped; otherwise his medication was left unchanged. Again he improved. In September 1974, he fell and sustained a fracture of the tenth left rib. After that he began to deteriorate and became practically bed-ridden. His ascites increased. In April 1975, he became anaemic (10.5 gm/100 ml) and a barium

swallow was arranged. There were no oesophageal varices. The dilation and stenosis remained unchanged, but there were no tertiary peristaltic waves seen. There was a small hiatus hernia without reflux. He was hypokalaemic (3.6 milli eq.) and 'Slow-K' (1,800 mg) was again added to his medication.

In May 1975 (two weeks later), he developed severe dysphagia and even vomited fluids. On further questioning he admitted occasional very mild dysphagia during the past two years but "not worth even mentioning". 'Slow-K' was immediately stopped. Immediately he improved and three weeks later he was seen to take chips, beans, and bread for lunch, without any difficulty.

Dysphagia has not since recurred, but ascites continued to increase. Arrangements for paracentesis were made, but he died at home in his sleep (June 1975). A post-mortem examination was not performed.

Comment

In retrospect, it seems possible that his abdominal pain was due to 'Slow-K' treatment. It started within three months, gradually increased, but disappeared when this was stopped in April, 1974. The tertiary peristalsis (April, 1973) might have been due to mucosal irritation. Gross hepatomegaly, the developing ascites and constipation might have slowed down or temporarily arrested the 'Slow-K' tablets and caused the abdominal pain.

When 'Slow-K' was started again in 1975 he was bedridden when the mild oesophageal stenosis (which remained unchanged) was enough to arrest the tablets. Dysphagia developed with similar rapidity as in the cases previously reported (Chesshyre, 1971) but immediately subsided when 'Slow-K' was stopped.

Discussion

Paradoxically, the non-disintegration of 'Slow-K' is responsible for its complications, because the tablets can be arrested or trapped by their bulk. The enteric-coated tablets were arrested by intestinal spasm, when the high concentration of KC1 developed, as shown by the circumferential ulcers and strictures (Ansell, 1974; Ashby, 1965; Grant, 1975). In 'Slow-K' the reverse happens, the tablets are first arrested and then the KC1 concentration develops. In oesophageal complications dysphagia is probably caused by the obstructing effect of the tablets as well as by the secondarily developing high KC1 concentration.

It has previously been suggested that a tablet might arrest at a prepyloric stenosis (Ansell, 1974). In the available literature a case of ulceration of the mouth was reported when the patient was sucking the tablet instead of swallowing it (McAvoy, 1974).

In the second patient, who has had a partial gastrectomy, 'Slow-K' could have left the stomach through the anastomosis quicker than otherwise, but was slowed down after that by the pressure of the hepatomegaly and ascites, aggravated by constipation. The tertiary peristalsis showed that there must have been a reflux. Whether the mild oesophageal stenosis was pre-existing or caused by these tablets remains doubtful. However, it has not increased after two years, but there were no more tertiary peristaltic waves, because he has not had 'Slow-K' for a year.

Diverticulum of the caecum is not common and few if any of such patients were on KC1 supplementation; no such case was reported so far. There may be other conditions, say a blind loop (or others) where these tablets may be arrested, a possibility which has to be remembered.

In intestinal hyperaemia (venous congestion) the mucosa may be more sensitive to less high KC1 concentration than otherwise. Most of the reported cases were in cardiac failure. If an atypical, undefinable abdominal pain develops, stopping the 'Slow-K' may be diagnostic and therapeutic as well, as the symptoms may subside.

It will be mainly in general practice that untoward effects of 'Slow-K' can be detected, though also in long-term geriatric patients.

Potassium sparing diuretics may not be enough to prevent hypokalaemia, but the present effervescent KC1 preparations contain 12 meq K and 8 meq Cl ion. If the salt intake is not severely restricted, they may be adequate. They can be sufficiently diluted and can not be arrested.

The use of KC1 supplementation is now under review. It was found that morbidity, or

death were far more frequent in the group where KCl replacement and/or potassium sparing diuretics were given than in the other group where potassium wasting diuretics were given without supplementation (Lawson, 1974). In other surveys (Dargie, 1974; Wilkinson, 1975) of long-term frusemide treatment without potassium supplementation no significant hypokalaemia developed and KCl supplements were not retained in mild hypertension. However, in cardiac disease there is an inherent potassium depletion which may be aggravated by diuretics (Edmondson, 1974); KCl supplementation is therefore necessary. It is possible that KCl will be only selectively given in certain conditions, but not in others.

It will take time before the risks are finally clarified. It is important that in the meantime any serious side-effects of KCl supplementation should be avoided. General practice is an eminently suitable place to monitor this treatment to prevent untoward effects.

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RISK OF CONGENITAL ABNORMALITY AFTER INADVERTENT RUBELLA VACCINATION OF PREGNANT WOMEN

The risk to a fetus after rubella vaccination of its mother is unknown. The Centre for Disease Control has compiled information from the pregnancies of 343 women inadvertently given rubella vaccine shortly before or after conception. The pregnancies of 145 women were terminated by therapeutic abortion, and rubella vaccine virus was recovered from the products of conception of nine women, including six of the 28 known to be seronegative to rubella at the time of vaccination.

None of the 172 infants carried to term had either clinical evidence of rubella infection, including 38 infants of women known to be susceptible and 12 additional women estimated to be susceptible at the time of rubella vaccination. On the basis of the binomial distribution, the maximum risk of fetal infection after maternal rubella vaccination is between five and ten per cent. The actual risk is probably less.

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