Unusual presentation of pain following partial splenic embolization-A case report.

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Introduction: Partial splenic embolization (PSE) has become an important therapeutic modality in the management of hypersplenism due to various causes including biliary atresia, in both children and adults. (1,2) Pain following PSE has been effectively managed with intravenous opioids, acetaminophen and sometimes with an epidural catheter. (1,3). Several complications including, abscess formation, sepsis and rupture have been described. However, there have been no reports of difficulty in pain management following PSE. We report the management of a case where recurrent episodes of extreme pain necessitated an extended hospital stay.

Case Report: A 17-year old female (wt.-60 kg) presented to an outside hospital with left flank abdominal pain, abdominal distension and nausea. A CT scan of the abdomen revealed a large spleen extending below the level of the umbilicus. The patient was then transported to our institution. Past medical history was significant for biliary atresia for which a Kasai procedure was performed during infancy, portal hypertension, hypersplenism, reactive airways disease, and seasonal allergies. The patient was awaiting a liver transplant. The patient was clinically stable and had a platelet count of 25,000. The following day she underwent PSE under general anesthesia. The goal of PSE was to increase the platelet count, decrease the splenic size, and to avoid open splenectomy so as to facilitate an easier surgical approach to liver transplantation in future. A morphine PCA was ordered for post-embolization pain control. On post-operative day (POD) one, the morphine PCA was replaced with a hydromorphone PCA (Demand 0.3mg, Lockout-8min, No basal, Hourly Max- 1.5 mg) as the patient complained of severe itching and nausea. The same night, the patient developed severe abdominal infarction pain, which continued into POD 2. Hydromorphone rescues were given and the PCA settings changed to include a basal infusion of 0.2mg/hr and the demand dose was increased to 0.4mg. On POD 3, the pain score (PS) was 0 (Numeric 0-10 scale) and therefore the basal was stopped and the patient was started on oxycodone 7.5mg PO 4-hourly PRN with continuation of the demand PCA. The following day the PS was increased to 6 and the basal infusion was restarted. The PS remained at 6 and on POD 6, oral methadone 20mg PO 8-hourly was started. The PCA was stopped on POD 9 and so was the oxycodone. The patient remained status quo until POD 12 when she developed severe abdominal pain, which was proven by abdominal CT to be due to a new infarct in the spleen. This was treated with a hydromorphone bolus and the hydromorphone PCA was resumed without a basal. The patient continued to have more infarctions on POD 14, 15 and 16, and was managed with hydromorphone rescues. The patient remained stable at PS score of 5-6 and started physiotherapy. It was observed that the patient timed her lockout intervals and consequently all the button pushes with the PCA resulted in a demand dose being delivered. As she started improving with physiotherapy, we decided to wean her from the PCA. The total dose of hydromorphone used in 24 hours was calculated and this was delivered as a continuous hydromorphone infusion. The infusion was tapered between POD 23 and 27. On POD 23 the methadone dose was increased to 30mg PO 8-hourly. The patient received occasional rescues prior to physiotherapy, but otherwise remained stable on the pain management regimen. On POD 28, the patient was discharged to home on methadone at 30mg PO 8-hourly with morphine sulphate rescues PO. The patient was uneventfully weaned from the methadone over a three-month period.

Discussion: PSE is becoming increasingly common as a technique to treat selected cases of hypersplenism. Kimura et al reported that all patients were discharged at approximately one week following PSE. (4) Although studies have recognized the need to manage pain after PSE, none have reported any difficulties in management of pain following PSE. (1,3) Pain following PSE causing delay in discharge has never been reported. In our patient, recurrent painful infarctions following the initial PSE was likely due to the large size of the spleen, and the sole cause of the delay in discharge. It was also a difficult situation for the family, as they had come with the expectation that the patient would be discharged within a week. We suggest that the need for a prolonged stay be kept in mind, particularly in cases of PSE for massive splenomegaly.

References:

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4. Kimura F. et al, AJR 2002.