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Abstract

Foot drop following colonoscopy under general anesthesia is rare in children and is generally unexplained. We report two children who developed foot drop within 12 hours following colonoscopy under general anesthesia and recovered fully within 6 months following physiotherapy. Extensive investigation revealed no etiology. However, we observe that both children had a low BMI, a clinical feature recognized to be associated with common peroneal injury in adults following surgical procedures, but not unique to colonoscopy alone.

Keywords: Foot drop; children; peroneal nerve injury; colonoscopy; anesthesia; hyponatremia.

INTRODUCTION

Foot drop, possibly due to common peroneal nerve injury has been reported following colonoscopy in children [1]. The reported incidence is 1:250 [2] but causes are illusive. We present bilateral foot drop in a 9-year-old child and a unilateral foot drop in a 12year-old child, both following colonoscopy under general anesthesia. We aim to discuss possible etiologies and predisposing factors for this uncommon complication.

CASE REPORT 1

A 9-year-old boy presented as a day case for an elective esophago-gastro-duodenoscopy (OGD) and a colonoscopy as part of an ongoing investigation of chronic abdominal pain. He had no previous neurological or developmental concerns. Past medical history was unremarkable aside from a previous episode of vitamin D deficiency, with adherence to a vegetarian diet.

His preoperative assessment was unremarkable; ASA

grade 1[3], 25.3 kg body weight (BMI 12.8 kg/m²), BP 96/57, pulse 104/min, SpO, 100% breathing room air and apyrexial. He underwent general anesthesia for approximately 1 hour following a gaseous induction with nitrous oxide, oxygen, sevoflurane and received fentanyl 1mcg/kg, Hartmann's solution 20mls/ kg, and atracurium 0.6mg/kg. He was intubated with a 5.0mm cuffed endotracheal tube and was ventilated with 50% N_2O/O_2 and sevoflurane. He was hemodynamically stable throughout the general anesthetic and postoperatively. No adverse events were recorded during procedure carried out in supine 'frog leg' position. He had no regional or local anesthesia. Endoscopic findings of both upper and lower gastrointestinal tract including terminal ileum were macroscopically and histologically normal.

He was discharged home 2 hours following his procedure in the evening. He was fully conscious, had no pain and was able to sit up but complained that he could not walk. When assisted, he seemed to drag his feet. The discharging nurse noted a slightly decreased

circulation in the right leg, but considered it was trivial. Still, he had to be carried from hospital. At home, dad noted that his right foot had poor sensation and made a self-referral back to their local hospital A&E on the same evening. He had no sensation on his right foot, but circulation was intact. He was resident and was referred to a tertiary children's neurology unit on the 3rd day. Examination at this time demonstrated Medical Research Council (MRC) grades [4] 4/5 power in right hip flexion, 2/5 in left ankle dorsiflexion and 1/5 in right ankle dorsiflexion, planter flexion, inversion and eversion. Power in all other muscle groups, including upper limbs, was 5/5. Independent ambulation was possible with supervision, with a bilateral high stepping gait. Sensation was reduced in L4-S1 distribution in the right lower leg, and in L5 distribution on the left. Deep tendon reflexes where present and normal in character in the upper and lower limbs, except for an absent right ankle jerk. Investigations were undertaken to exclude a compressive or inflammatory pathology. An MRI of the brain and spine with contrast was unremarkable. Cerebrospinal fluid (CSF) studies demonstrated acellular CSF, normal protein, glucose and lactate levels. Virology studies, blood and urine investigations were unremarkable. His plasma CK was 87 IU/L and was negative for Mycoplasma Pneumonia IgM and Borrelia burgdorferi antibodies, and HSV serology. Vitamin A, Vitamin E, Vitamin B12 and Thiamine levels were all within normal range. HBA₁C was unremarkable at 5.4%, lead levels were undetectable, and a urinary porphyrin screen was normal.

At discharge from the ward, hip flexion had normalized, with improved dorsiflexion of the left ankle to grade 2/5, and right ankle to 2/5. Nerve conduction studies performed 6 days after the presentation were reported normal. Genetic testing including PMP22 gene analysis [5] and extended Bristol peripheral neuropathy panel (Bristol Genetics Laboratory https://www.nbt.nhs. uk/genetics) revealed a heterozygote variant in exon 19 of the SCN9A gene (c.2404A>G) which was felt to be of no clinical significance. His parents reported that at 3 weeks his right foot was showing progressive improvement, but not on the left.

At 6 weeks following the presentation, reduced dorsiflexion of the left ankle remained at grade 2/5 with an improvement in all other muscle groups to grade 4/5 in the right leg. He continued to receive physiotherapy and hydrotherapy. By 12 weeks

symptoms had entirely resolved and gait returned to normal. Physical examination 9 months after the episode was unremarkable.

CASE REPORT 2

A 12-year-old girl with a diagnosis of Crohn's disease for 2 years was reassessed with an OGD and colonoscopy under general anesthesia as she was found to have a distal ileal stricture on a routine bowel MRI scan. She was receiving maintenance therapy, prednisolone 5mg weekly and azathioprine 75mg daily. Her body weight was 41.3 kg (BMI 15.1 kg/m²). Her general anesthetic was uneventful.

Following the procedure, she experienced numbness localized to the lower left leg and a unilateral foot drop on the same side. She was unable to walk properly on the following day. Some sensation returned as early as 12 hours into her recovery period but exhibited markedly reduced plantar flexion needing assistance with a splint, orthotics and physiotherapy. She empirically received vitamin B12 and vitamin D therapy following a private consultation two week following her onset of foot drop, but these were subsequently stopped as blood levels of both Vit D and B12 were noted to be high. Her Crohn's disease therapeutic regimen was changed with the addition of an induction course of infliximab. No residual sensory or motor deficits were noted at 6 months follow up.

Discussion

Peripheral nerve injuries can occur during the perioperative period and their etiology is often obscure. Surgical, anesthetic or mechanical factors may contribute to the pathogenesis of this complication [6]. Motor neuropathy of a lower extremity is well-recognized as a complication of lithotomy position in adults. The recognized risk factors of this complication include lithotomy position for 4 h or longer, a body mass index of 20 kg/m² or less, and a history of smoking within 30 days of the procedure [7]. 'Foot drop' resulting from a peripheral neuropathy is also a recognized complication of colonoscopy in adults, and often fully recoverable [1]. The precise underlying pathological mechanism of this complication is unclear, but the 'frog leg' position used during colonoscopy is considered contributory. Regional anesthetic techniques have not been found to be associated with an increased risk of neuropathy [7]. A prospective evaluation of

lower extremity neuropathies amongst 991 adult patients undergoing general anesthetics and surgical procedures while positioned in lithotomy reported that lower extremity neuropathies were infrequent complications (1 per 3068 cases) and none resulted in prolonged disability [7]. The longer the patients were positioned in lithotomy position, the greater the chance of development of a neuropathy[8].

Common peroneal nerve injury leading to foot dropis also noted to occur after cardiothoracic surgery [9]. These patients were older, subnormal in body weight, and had considerable comorbidity such as peripheral arteriosclerotic disease, diabetes mellitus, and arrhythmias. Here too, prognosis was mostly good, with the provision of early physiotherapeutic treatment [9]. Common peroneal neuropathy can also be independently related to weight loss [10]. Entrapment neuropathy of the common peroneal nerve caused by compression at the fibula head region and sciatic nerve lesions can mimic a common peroneal nerve palsy [11]. Its incidence following post-gynecological surgery is 1.1 to 1.9% and alluded to lithotomy positioning related direct nerve compression and neuropraxia [9].

Common peroneal neuropathy post colonoscopy is recognized in adults. The pathogenesis is not clear [12] and speculative. It is unlikely to be via a mechanical injury alone associated with 'frog leg'position. This is because it has been described even after procedures such as liver surgery with legs in supine neutral position [13]. Although placing children in the 'frog leg' position increases the diameter of the femoral vein visualized, no flow compromise has been observed [14]. A normal nerve conduction study (NCS) was noted 1 week following the incident in one of our children but it does not necessarily exclude a conduction abnormality at inception. NCS can be normal in early GBS (<2 weeks from initial symptom onset) and not all patients can be electro diagnostically ascertained [15]. In a case series of 746 young people (aged 6m-24 years), common peroneal nerve palsy was recognized in 3 patients following colonoscopy under deep sedation or general anesthesia. All 3 patients were thin and malnourished adolescents with a low body mass index, two had systemic inflammatory conditions and

were receiving steroids. All patients were temporarily supine with the hips flexed and externally rotated and with the knees flexed and supported laterally ("frog leg position"), and temporarily in a left lateral decubitus position. All three nerve injuries resolved, within days in one patient, 4 weeks in the second, and more than 3 months in the third [1].

Other possibilities are inflammatory bowel syndrome related neurological presentations [16]or that is related to electrolyte abnormalities. Electrolyte abnormalities such as hyponatremia may occur following bowel preparation for colonoscopy, that can trigger neural demyelination. Bowel preparation leading to significant dehydration, aggravated by preoperative fasting is common as most children complain of thirst before the procedure. In general, children receive intravenous fluids under anesthesia, in the form of normal saline or Hartman's solution. It is known that rapid correction of hyponatremia may also be linked to an osmotic demyelination syndrome that may lead to pontine or extra pontine demyelination [17] [18]. A link to associated hypokalemia has also been hypothesized [18]. Children with chronic diarrhea, for example with inflammatory bowel disease may be vulnerable to electrolyte imbalances associated with bowel preparation. Oral bowel preparations can lead to severe hyponatremia in the vulnerable adult patient, particularly, with sodium picosulfate/magnesium citrate (PICOLAX ®) with two liters of water and two liters of tea, albeit within two hours leading to neurological manifestations [19].

A case of colonoscopy-induced hyponatremia encephalopathy preceded a study into hyponatremia as a potential risk of gastrointestinal endoscopy. This study investigated 40 patients prior to and following colonoscopy and found the incidence of hyponatremia of 7.5% post colonoscopy. Raised serum levels of anti-diuretic hormone found in these cases may form the physiological basis for the hyponatremia. Hyponatremia can trigger neurological injury [16]. Disorders of water balance, termed 'bowel prep hyponatremia" is a recently proposed risk factor [19].

Colonoscopy rarely requires sedation in adults. On the contrary, intravenous sedation or general anesthesia (GA) is frequently used when performing

colonoscopies on children. A prospective audit of pediatric colonoscopy under GA was conducted on a total of 250 colonoscopies in 215 children of median age 10.7 years-old. The audit aimed to present the safety of colonoscopy under GA and the advantages of using GA vs intravenous sedation. In this study, theonly major adverse event reported from colonoscopy performed under GA was unilateral peroneal nerve palsy in a16-year-old girl whose colonoscopy was reported normal [17]. It is believed that perioperative positioning was responsible for this and indeed other literature seems to agree that positioning can cause unilateral nerve damage, especially to the common peroneal nerve.

CONCLUSIONS

Although perioperative positioning, leading to direct nerve compression is the speculated etiology for motor neuropathy following colonoscopy, recognized risk factors such as low BMI, hyponatremia and prolonged lithotomy positionpoints to an elusive pathophysiology. Until the exact etiology is found, optimization or mitigation of the known risk factors such as hypovolemia, dehydration, electrolyte disturbances, prolonged lithotomy position and low BMI, may reduce the incidence of nerve injury following colonoscopy in children.

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