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New Onset Focal Weakness in Children With Down Syndrome

Gordon Worley^{1,*}, Rolla Shbarou³, Amy N. Heffner⁴, Karin M. Belsito⁴, George T. Capone⁴, and Priya S. Kishnani²

¹*Department of Pediatrics, Program in Neurodevelopmental Pediatrics, Duke University Medical Center, Durham, North Carolina* ²*Department of Pediatrics, Division of Genetics and Metabolism, Duke University Medical Center, Durham, North Carolina* ³*Department of Medicine (Neurology), Duke University Medical Center, Durham, North Carolina* ⁴*Kennedy Krieger Institute of the Johns Hopkins Medical Institutions, Baltimore, Maryland*

Abstract

New onset focal weakness is relatively common in patients with Down syndrome (DS), and has broad differential diagnosis. Ten cases of new onset focal weakness in patients with DS were encountered or are currently being followed in two DS clinics, with a combined population of patients of approximately 850, for a clinic population prevalence of 1.2%. The median age at presentation was 4 years old (range 1 month–44 years). The causes of new onset focal weakness were: stroke from Moyamoya disease (two patients); stroke from vaso occlusive disease (one patient); stroke from venous sinus thrombosis (one patient); traumatic subdural hematoma (one patient); brain abscess (one patient); spinal cord injury (SCI) from cervical spinal stenosis (two patients); SCI from atlantoaxial instability (AAI) (one patient); and brachial plexus injury (one patient). Of the 10 patients with focal weakness, 8 had potentially treatable conditions, and 5 had surgery. The differential diagnosis of new onset focal weakness in DS is broad, with diseases reported involving all levels of the nervous system from brain to muscle. For some diagnoses, expeditious diagnosis may improve outcome.

Keywords

Down syndrome; hemiparesis; Moyamoya; spinal cord injury; stroke; brain abscess; bacterial endocarditis

INTRODUCTION

Specialized clinics that care for patients with Down syndrome (DS) at tertiary care centers follow larger numbers of such patients and more patients with unusual problems than are followed in other settings. Encountering a relatively large number of patients with focal weakness in our clinics, we were struck by the condition's relative prevalence and by its many different etiologies. DS has many associated (secondary) conditions that can predispose to the development of focal weakness, making its differential diagnoses in DS different from and broader than that in children in the general population.

*Correspondence to: Gordon Worley, M.D., Box 3120, Duke University Medical Center, Durham, North Carolina 27710. E-mail: worle001@mc.duke.edu

METHODS

Cases of new onset focal weakness from DS Clinics at two institutions, Duke University Medical Center (DUMC) and the Kennedy Krieger Institute (KKI) of Johns Hopkins Medical Institutions, were retrospectively ascertained and patient records reviewed, to determine clinical presentation, diagnosis, and outcome and to estimate the prevalence of new onset focal weakness in our clinic populations.

RESULTS

Illustrative Case Presentations

Case one—A 17-month old boy with trisomy 21 DS presented with acute right gaze paresis and left hypotonic hemiparesis. Computerized tomography (CT) imaging of the brain revealed patchy, low density areas in the distribution of the right middle cerebral artery (MCA), involving both the cortex and basal ganglia. A cerebral angiogram showed decreased blood flow to areas of ischemic infarction, but not an embolic occlusion, aneurysm, or arteriovenous malformation. The “puff of smoke” angiographic finding of Moyamoya disease was not present. One month later, the patient developed left-sided facial seizures and was started on phenobarbital. At the age of 32 months, the patient presented again with acute weakness, this time involving the right side, following a brief right-sided focal seizure. He was now densely hemiparetic on the right side, with his arm being more involved than his leg and his arm weaker distally than proximally. Aspirin prophylaxis was initiated. A cerebral arteriogram now revealed marked stenoses in many vessels, dilatation of the lenticulostriate arteries bilaterally, and marked collateral circulation, considered diagnostic for Moyamoya disease. Nine months later, at 43 months of age, he underwent right MCA territory revascularization, by an encephalo-arteriodural synangiosis utilizing the right superficial temporal artery. He has had no further strokes since his surgery 12 years ago.

Case two—A 4 year 10 month old boy with trisomy 21 DS developed weakness in his right leg, followed by weakness in his right arm. He had no history of significant trauma. The weakness was slowly progressive over the next 7 weeks. Cervical spine lateral flexion and extension revealed no atlantoaxial instability (AAI) or atlanto-occipital dislocation (AOD). An MRI showed cervical spinal stenosis at the level of C1. He underwent decompression of the posterior arch of C1 with fusion of his spine from the occiput to C2, using a Luque rod and a bone graft from his posterior iliac crest and he was put in halo traction. Over the next 2 weeks in rehabilitation, his weakness began to improve and he became ambulatory with assistance. Subsequently, his recovery has been remarkable. When last seen 3 years after the surgery, he had good function in all extremities with no focal findings. He was discharged from Neurosurgical care.

Case three—An 8 year old boy with trisomy 21 DS and an endocardial cushion defect associated with Eisenmenger’s physiology presented 1 day before admission with nausea, vomiting, and diarrhea. Twenty-four hours later, he abruptly developed clonic seizures involving his right arm, lasting for 2 hr, not associated with loss of consciousness. He was aphasic during the seizures but could nod his head to answer questions. He was referred to DUMC. An MRI revealed a large brain abscess that was 4 centimeters in diameter located in the left frontal lobe just beneath the left coronal suture. Ten milliliters of purulent material, of a “chocolate brown” color was drained from the abscess, from which *Streptococcus milleri* was eventually cultured. He was treated with antibiotics to which the organism was sensitive and he was not allergic, and responded. Problems with his Eisenmenger’s physiology persisted, prolonging his hospitalization. He was evaluated by a Pediatric Dentist who found that he had 3 abscessed teeth and 11 carious teeth, with enamel erosion from gastroesophageal reflux.

Dental surgery was performed to remove his abscessed teeth and fill his carious ones, without complication. Transesophageal echocardiograms revealed no intra-cardiac vegetations. By 3 months after his admission, he was able to walk with a walker and could use his right arm as an anchor, but not for fine motor tasks. His aphasia was also resolving. He died 6 months later from septic shock, thought to be due to a central venous catheter infection.

Prevalence: Ten cases of new onset focal weakness were encountered in two DS clinics, with a combined population of patients of approximately 850, for a clinic population prevalence of 1.2%.

Patient description: The median age at presentation was 4 years old (range 1 month-44 years). Seven males (70%) and three females (30%) were encountered. All patients had a phenotype consistent with DS. Of the 10 patients, 6 (60%) had trisomy 21, 1 (10%) had translocation 14; 21; and the karyotype of 3 (30%) was unknown. In Table I, the 10 cases of focal weakness are presented by: age at onset; presenting symptom or sign; final diagnosis; treatment; and outcome.

Treatment: Five of the six patients with focal weakness from a brain abnormality required some treatment. Both of the patients with Moyamoya disease underwent synangiosis. Neither child has had a subsequent stroke. One patient had drainage of a traumatic subdural hematoma. One patient with a brain abscess causing acute hemiparesis responded to surgical drainage of the abscess and antibiotic treatment, and one child with Type IV hyperlipoproteinemia was started on a lipid-lowering agent and has had no subsequent strokes.

Of the three patients with a spinal cord injury (SCI) as the cause of their acute focal weakness, two required treatment. The patient with hemiparesis from cervical spinal stenosis underwent laminectomy of C1 and spine fusion from the occiput to C2. He has recovered completely. Surgical decompression was also performed for the patient with degenerative arthritis involving the cervical vertebrae, causing spinal stenosis and myelomalacia. The patient with AAI and spinal cord compression developed acute arm weakness when 13 years old. She was first seen by us 8 years later.

DISCUSSION

New onset focal weakness is relatively frequently encountered in patients with DS who are followed in specialized clinics that provide comprehensive care, such as those at our two institutions. The prevalence of new onset weakness was 1.2% in our combined clinic populations, a much greater prevalence than for hemiparesis from all causes in children in the general population.

To our knowledge, no series of new onset focal weakness in patients with DS has been compiled previously. We were prompted to do so by the unusual variety of diagnoses of our patients and by our recognition of the potential importance of prompt treatment for some of these conditions.

Table II summarizes the diagnoses causing focal weakness in DS, reported by others or encountered by us, categorized by the level of the nervous system involved: disorders or diseases of the brain; spinal cord; spinal roots; and peripheral nerves. The reported diagnoses causing acute focal weakness in children and adults with DS are different from those usually encountered in the general population [Riela and Roach, 1993]. Several deserve specific comment.

The ligamentous laxity of DS may predispose to SCI. We encountered one case of SCI resulting in hemiparesis from AAI and spinal cord compression. Dzenitis [1966] first reported SCI in

DS from AAI. When C1 attains a relatively anterior luxation on C2 due to ligamentous laxity, the spinal cord can be compressed between the odontoid process anteriorly and the posterior arch of C1 posteriorly, if the instability is severe enough. AAI can be diagnosed by obtaining cervical spine X-rays in the flexion, extension, and neutral positions of the spine and measuring the distance from the anterior aspect of the odontoid to the posterior aspect of C1, with the neck in flexion [Hreidarsson et al., 1982]. The presence of other cervical spine abnormalities that are common in people with DS, such as odontoid dysplasia, may also predispose to AAI [Pueschel et al., 1990]. Asymptomatic AAI is found in 13.1% of persons with DS, but symptomatic AAI is much less common, occurring in only 1.5% at most [Pueschel and Scola, 1987]. Manifestations of cord compression from AAI include: quadriparesis; hemiparesis; incontinence; balance problems; leg weakness; and neck pain. The onset is usually insidious but can also be sudden, presenting with paralysis. Imaging of the cord by MRI can be helpful in diagnosis. The prognosis depends upon the severity of SCI, the presence of associated bony abnormalities [Chaudhry et al., 1987], and perhaps prompt treatment (see below). Ligamentous laxity might also predispose to the development of AOD, which can also result in SCI [Hungerford et al., 1981; Martich et al., 1992]. A hypoplastic posterior arch of C1 is found in 25% of patients with DS, causing cervical spinal stenosis and placing them in “double jeopardy” for SCI, if present in association with either AAI or AOD, or both [Martich et al., 1992]. One of our patients had an SCI from cervical spinal stenosis.

One adult patient in our series developed weakness from cervical stenosis and radiculomyelopathy. Cervical spine degenerative joint disease (DJD) develops early [Bosma et al., 1999] and frequently [Maclachlan et al., 1993] in patients with DS, usually associated with spur formation, narrowing of the foramina, and narrowing of the disc inner space. Frost et al. [1999] reported a 49-year old with severe DJD of the cervical spine that led to cervical stenosis. After a fall, she developed acute triplegia, from spinal cord compression. She subsequently died. Autopsy findings were presented.

The baby in our series with arm weakness had a brachial plexus injury, also a possible consequence of ligamentous laxity. Her delivery was unremarkable. The cause of her brachial plexus injury was not determined. Her arm weakness resolved without treatment by 6 months of age.

Moyamoya disease is much more common in patients with DS than in the general population [Fukushima et al., 1986]. It is a disease of the internal carotid arteries and the vessels of the circle of Willis, in which stenoses of involved vessels progress to occlusion. As stenoses worsen, collateral vessels develop distal to the stenoses in order to supply the under-perfused parenchyma in intervascular “water shed” zones [Suzuki and Takaku, 1969; Schrager et al., 1977; Suzuki and Kodama, 1983; Farrugia et al., 1997]. Children with Moyamoya disease have ischemic strokes, but adults have hemorrhagic ones, in both cases in collateral vessels. Patients with stroke usually initially present with hemiparesis and/or seizures. Other neurological deficits can also occur but are less common. Substantial recovery of acute neurological deficits over time is common in children [Cramer et al., 1996] but the course is usually eventually progressive resulting in severe motor deterioration and intellectual impairment [Suzuki and Kodama, 1983]. Three neurosurgical procedures are used to prevent subsequent strokes in children (they are not effective in adults): anastomosis of the superficial temporal artery to the MCA; encephalomyosynangiosis; and encephaloduro-arterio synangiosis [Ueki et al., 1994; Adelson and Scott, 1995]. Exuberant angiogenesis after anastomosis results in improved regional brain perfusion. Low dose aspirin may also be helpful to prevent ischemic strokes.

One patient with acute hemiparesis was found to have had a MCA occlusion on MR angiography, without evidence of Moyamoya disease. He had an elevated fasting triglyceride concentration of 398 mg/dl, an elevated cholesterol concentration of 217 mg/dl, and a low high-

density lipoprotein concentration of 27 mg/dl. His protein electrophoresis was diagnostic of Type IV hyperlipoproteinemia.

Type IV hyperlipoproteinemia is characterized by an elevated plasma triglyceride concentration in patients and family members. Patients with the disorder are at increase risk for atherosclerosis and early coronary artery disease [Merck Manual, 2001]. Type IV hyperlipoproteinemia has not been reported in DS previously. It is possible our patient's hyperlipoproteinemia is related only by chance with his DS. Gorska et al. [1976] found no significant difference in concentrations of total serum lipids, triglycerides, total cholesterol, or phospholipids; and no characteristic electrophoretic pattern of alpha or beta lipoproteins in subjects with DS, compared to normal controls and Dorner et al. [1984] found that total cholesterol and triglyceride concentrations in DS subjects were not significantly different from mentally retarded controls. It is also possible there may be a relationship between his hyperlipoproteinemia and DS. Triglyceride concentration has been reported to be elevated in subjects with DS, compared to mentally retarded controls by Nishida et al. [1977] and compared to healthy controls by Zamorano et al. [1991] who also found that low density lipoprotein and total cholesterol concentrations were elevated, but high density lipoprotein cholesterol concentration was lower.

Other secondary conditions in DS may also cause hemiparesis, but associations have not yet been reported, to our knowledge. Thromboembolism associated with congenital heart disease (CHD) is the leading known cause of stroke in children in the general population [Lanska et al., 1991]. Since CHD associated Eisenmenger's physiology with a right to left intra-cardiac shunt occurs relatively frequently in DS, it is possible that strokes due to thromboembolism have occurred in patients with DS, even though not reported. Other potential causes of acute focal weakness that might be more likely in patients with DS, but which also have not to our knowledge been reported so far in this population, include: septic emboli to the brain from subacute bacterial endocarditis; ischemic stroke from the polycythemia of cyanotic CHD; and stroke associated with the leukemias.

Expeditious diagnosis of focal weakness is now imperative since effective treatments are available for many etiologies. Surgery was performed on 5 of 10 patients with focal weakness.

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TABLE 1
Causes of Focal Weaknesses in Children With Down Syndrome (DS)

Age at presentation	Presentation	Diagnosis	Treatment	Outcome
1 month	Left arm paresis	Brachial plexus injury	None	Resolved
10 months	Right hemiparesis	Moyamoya disease (CVA)	Synangiosis	No more strokes; no change in hemiparesis
17 months	Left hemiparesis	Moyamoya disease (CVA)	Synangiosis	No more strokes; improved hemiparesis
28 months	Left hemiparesis, seizures	Traumatic subdural hematoma	Surgical drainage	Seizure-free; "persistent left hemiparesis"
4 years	Right hemiparesis	Cervical spinal stenosis (SCI)	Laminectomy spine fusion	Resolved
8 years	Fever, seizures, right hemiparesis	Brain abscess	Surgical drainage; antibiotics	Abscess resolved; improved hemiparesis
11 years	Left hemiparesis	Venous sinus thrombosis (CVA)	None	Improved hemiparesis
13 years	Left arm weakness	AAI (SCI)	None	Persistent arm weakness
25 years	Right arm weakness	MCA CVA from hyperlipidemia Type IV	None	Resolved
44 years	Right hemiparesis	Degenerative arthritis of cervical spine; cervical myelopathy, myelomalacia (SCI)	Spine surgery	Improved hemiparesis

AAI, atlantoaxial instability; CVA, cervical vertebral body; MCA, middle cerebral artery; SCI, spinal cord injury.

TABLE II
Reported Causes of New Onset Focal Weakness in Patients With DS

Brain
Cerebral infarction
Moyamoya disease (see text)
Large vessel thrombosis [Pearson et al., 1985]
Embolism [Moore et al., 1997]
“Top of the basilar” artery stroke [Baram and Fishman, 1985]
Stroke from Type IV hyperlipoproteinemia (reported herein)
Cerebral amyloid angiopathy and intracerebral hemorrhage [Belza and Urich, 1986]
Fibrous hypertrophy of arterial intimal walls, distinct from Moyamoya disease [Nagasaka et al., 1996]
Venous sinus thrombosis (reported herein)
Subdural hematoma (reported herein)
Internal carotid hypoplasia [Gaggero et al., 1996]
Brain tumor
Germinoma [Matsumura et al., 1998]
Cerebral infection
Brain abscess [Pearson et al., 1985]
Meningitis [Pearson et al., 1985]
Septic emboli [Pearson et al., 1985]
Other
Bilateral globus pallidus calcification [Okano et al., 1992]
Spinal cord
AAI (see text)
AOD (see text)
Demyelinating polyradiculoneuropathy [Yamana et al., 1990]
Cervical spinal stenosis [Martich et al., 1992]
Spondylarthrosis myelopathy [MacLachlan et al., 1993]
Transverse myelitis [Nazer et al., 1993]
Peripheral nerve
Carpal tunnel syndrome [Christensen et al., 1998]
Brachial plexus injury (reported herein)