Cardiac Findings in Congenital Muscular Dystrophies

abstract

Cardiac involvement (CI) in congenital muscular dystrophies (CMDs) has been only rarely investigated so far. By means of a systematic literature search we reviewed the literature about CI in CMD and found that CI is apparently absent in Ullrich CMD or CMD with integrin deficiency and only mild in Bethlem CMD. CI in merosin deficiency includes dilated cardiomyopathy and systolic dysfunction. CI in dystroglycanopathies seems most prevalent among all CMDs and includes dilated cardiomyopathy, systolic dysfunction, and myocardial fibrosis in Fukuyama CMD. Among the nonspecified dystroglycanopathies, CI manifests as dilated cardiomyopathy, hypertrophic cardiomyopathy (CMP) or systolic dysfunction. With CMD type 1C, as well as with limb-girdle muscular dystrophy 2I, up to half of the patients develop dilated cardiomyopathy. In rigid-spine syndrome, predominantly the right heart is affected secondary to thoracic deformity. In patients who carry *LMNA* mutations, CI may manifest as dilated cardiomyopathy, hypertrophic cardiomyopathy, or fatal ventricular arrhythmias. Overall, CI in patients with CMD varies considerably between the different CMD types from absent or mild CI to severe cardiac disease, particularly in merosin deficiency, dystroglycanopathies, and laminopathies. Patients with CMD with CI require regular cardiologic surveillance so that severe, treatable cardiac disease is not overlooked. *Pediatrics* 2010;126: 538–545

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KEY WORDS

congenital disease, neuromuscular disorder, genetics, cardiac disease, heart failure, arrhythmias, cardiomyopathy, congenital muscular dystrophy

ABBREVIATIONS

CMD—congenital muscular dystrophy
MEB—muscle-eye-brain disease
WWS—Walker-Warburg syndrome
MDC1C—congenital muscular dystrophy type 1C
RSS—rigid-spine syndrome
LGMD1B—limb-girdle muscular dystrophy type 1B
CI—cardiac involvement
ECG—electrocardiography
LGMD2I—limb-girdle muscular dystrophy 2I

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Congenital muscular dystrophies (CMDs) are rare genetic muscle disorders with preferentially autosomal recessive inheritance, which, per definition, manifest clinically at birth or early infancy. They are characterized by congenital hypotonia, delayed motor development, progressive muscle weakness, and dystrophic features on muscle biopsy.[1] CMDs are genetically and phenotypically heterogeneous and comprise disorders caused by (1) collagen VI mutations, which manifest as Ullrich or Bethlem CMD, (2) merosin mutations, which result in merosin-deficient CMD (MDC1A), (3) mutations in the *POMT1*, *POMT2*, *POMGnT1*, *FKRP*, *FKTN*, or *LARGE* genes, which manifest as nonspecified or syndromic conditions such as Fukuyama CMD, muscle-eye-brain disease (MEB), Walker-Warburg syndrome (WWS), or CMD type 1C (MDC1C), (4) mutations in the *SEPN1* gene, which manifest as rigid-spine syndrome (RSS), (5) lamin A/C mutations, which result in the congenital form of limb-girdle muscular dystrophy type 1B (LGMD1B), or (6) integrin $\alpha 7$ mutations, which manifest as CMD with integrin deficiency (Table 1).[1] Although some of the CMDs manifest also in organs other than the skeletal muscle, such as the cerebrum, the eyes, or the skeleton, and although neuromuscular disorders present frequently with cardiac disease, there is little known about the cardiac findings in patients with CMD. In this minireview of the Consensus Group of CMD Standard of Care, we provide an overview of cardiac involvement (CI) in CMDs, describe how CI in these patients can be managed, answer open questions, and outline future perspectives of CI in CMDs.

COLLAGEN VI CMDs

Ullrich CMD

Phenotype

Ullrich CMD is a rare congenital disorder that is clinically characterized by generalized muscle weakness, contractures of the proximal joints, and hyperextensibility of the distal joints and onset at birth or early infancy.[2] Onset is at birth or during the first year of life.[3–5] Only two-thirds of the patients acquire independent ambulation.[3] Decline of motor and respiratory functions is more pronounced within the first decade than in later years.[3] Approximately two-thirds of the patients are constant wheelchair users by 11 years of age.[3] Typically, the major palmar and plantar creases are absent.[2] Respiratory functions decline after 6 years of age.[3] Approximately two-thirds of the patients require noninvasive positive-pressure ventilation by 14 years of age.[3]

Genotype

Ullrich CMD is caused by mutations in the *COL6A1*, *COL6A2*, or *COL6A3* genes on chromosomes 21q22.3 and 2q37.

Cardiac Findings

In a study of 9 patients with Ullrich CMD, none showed any cardiac abnormalities on clinical examination, electrocardiography (ECG), or echocardiography.[2] In a study of 15 patients from 11 families, no cardiac abnormalities were found with ECG (10 patients) or echocardiography (5 patients).[4] Jimenez-Mallebrera et al[5] investigated 14 patients but did not find cardiac abnormalities on echocardiography in any of them. In the majority of the published cases, however, no cardiac examinations were conducted at all.[6] Overall, patients with Ullrich CMD do not seem to develop CI, but more systematic investigations are required to finally assess this point.

Bethlem CMD

Phenotype

Bethlem CMD is a rare congenital muscle disease that is clinically characterized by progressive muscle weakness and wasting, respiratory compromise, joint contractures, and distal laxity.[7]

TABLE 1 Cardiac Abnormalities in CMDs

Type	Mutated Gene	Cardiac Abnormality	Reference No.
Ullrich CMD	*COL6A1, COL6A2, COL6A3*	None	2 and 6
Bethlem CMD	*COL6A1, COL6A2, COL6A3*	None or mild (ECGab, hCMP)	7–12
Merosin-deficient CMD	*LAMA2, 1q42?*	None or ECGab, dCMP, SYSTDF, HF, MF	15 and 17–20
Fukuyama CMD	*FKTN, FKRP, LARGE*	dCMP, MF, SYSTDF, HF, ECGab	22–26
MEB	*POMGnT1, POMT2, FKRP, POMT1*	UK	32
WWS	*POMT1, POMT2, POMGnT1, FKTN, FKRP, LARGE*	None	34
MDC1C	*FKRP*	hCMP, SYSTDF, ECGab	35–37
RSS	*SEPN1*	ECGab, right HF, PH, HF, MPS coarctation	46–49
Lamin A/C	*LMNA*	None, MPS, PH, dCMP, hCMP, ECGab	51 and 53
CMD with integrin defect	*ITGA7*	UK	54

ECGab indicates ECG abnormalities; hCMP, hypertrophic cardiomyopathy; dCMP, dilated cardiomyopathy; SYSTDF, systolic dysfunction; HF, heart failure; MF, myocardial fibrosis; UK, unknown; PH, pulmonary hypertension; MPS, mitral prolapse syndrome.

Genotype

Bethlem CMD is allelic to Ullrich CMD and, thus, is also caused by mutations in the *COL6A1*, *COL6A2*, or *COL6A3* genes on chromosomes 21q22.3 and 2q37.

Cardiac Findings

In a study of 50 patients with Bethlem myopathy, several cardiac abnormalities were found on clinical examination, ECG, and echocardiography, but the abnormalities were not attributed to CI by the muscle disease.[8] In a study of 27 patients with Bethlem CMD, only 1 presented with asymmetric septal hypertrophic cardiomyopathy.[9] In a study of 33 members of a French-Canadian kindred, none showed any abnormality on cardiac examination and none of 2 on autopsy.[10] In a study of 3 patients with Bethlem CMD from the same family, cardiac investigations did not reveal any compromise.[11] Also, in 2 Japanese patients with Bethlem CMD, CI was absent.[12] Respiratory compromise in patients with Bethlem CMD is attributed to involvement of the respiratory muscles rather than to CI.[7] Overall, the heart seems to be only mildly affected in some patients with Bethlem CMD, possibly because of other comorbidities.

MEROSIN-DEFICIENT CMD (MDC1A)

Phenotype

Merosin-deficient CMD is the most common form of CMD and represents ~40% of them.[13,14] The clinical presentation is characterized by severe, progressive muscle weakness and wasting, inability to achieve independent ambulation, severe restrictive respiratory insufficiency that requires mechanical ventilation, macroglossia, occasionally mental retardation, seizures, markedly raised creatine kinase levels, and characteristic white matter hyperintensities on cerebral MRI.[15,16]

Genotype

Merosin-deficient CMD is caused by mutations in the *LAMA2* gene on chromosome 6q22-23 (Table 1).[13] In one family it was linked to a yet-unknown gene on chromosome 1q42 (*MDC1B*) and showed a more severe phenotype than those with classical merosin-deficient CMD but has not been reported again since then.[14]

Cardiac Findings

In a study of 5 patients with merosin-deficient CMD, none presented with cardiac abnormalities.[15] In a literature search on 248 reported cases, however, ECG or echocardiography results were abnormal in 35% of the cases.[15] In more than half of these cases, CI was subclinical.[15] Cardiac abnormalities most frequently reported were right bundle branch block and dilated cardiomyopathy.[15] In a study of 6 patients, a reduced ejection fraction (ejection fraction $<$ 40%) was found on echocardiography in 2 patients.[17] Among 4 patients with merosin-deficient CMD, only 1 developed heart failure and dilated cardiomyopathy.[18] According to a report on 2 patients, a girl died suddenly from ventricular fibrillation at 5 years of age in the absence of structural cardiac abnormalities, and her 9-year-old brother presented with abnormal depolarization and prolonged QT interval.[19] Both were assumed to have had myocarditis. At autopsy merosin may be absent in cardiac muscle without clinical manifestations.[20] CI may be also completely absent in this disorder.[15]

DYSTROGLYCANOPATHIES

Dystroglycanopathies are CMDs with autosomal recessive inheritance characterized by abnormal glycosylation of α-dystroglycan.[21] Dystroglycanopathies are caused by mutations in 6 different genes (*POMT1*, *POMT2*, *POMGnT1*, *Fukutin* [*FKTN*, *FCMD*], *FKRP*, or *LARGE*) and are clinically heterogeneous, including different phenotypes such as Fukuyama CMD, MEB, WWS, or MDC1C.[21]

Fukuyama CMD

Phenotype

Autosomal recessive Fukuyama CMD is clinically characterized by severe progressive weakness and wasting, structural central nervous system abnormalities (cortical dysgenesis), mental retardation, ocular involvement, and skull deformities.[22,23]

Genotype

The disease is genetically heterogeneous caused by mutations in the *FKTN*, *FKRP*, or *LARGE* genes, respectively. However, the vast majority of patients carries the Japanese ancestral retrotransposal insertion of tandemly repeated sequences in the *FKTN* gene. Mutations in the *FKRP* and *LARGE* genes produce Fukuyama CMD–like phenotypes.

Cardiac Findings

CI in Fukuyama-type CMD seems to be more prevalent than in other CMDs. Among 4 families with the disease, dilated cardiomyopathy was reported in 6 individuals.[23] All of them had reduced systolic function and increased left ventricular end-diastolic diameter. One patient presented with right bundle branch block, 1 presented with premature ectopic beats, and 1 died from progressive heart failure at 12 years of age.[23] In a 17-year-old boy with the disease, myocardial fibrosis in the absence of coronary heart disease was detected and interpreted as CI.[22] In a study of 34 patients aged 6 months to 30 years, systolic dysfunction was observed in 83% of the patients older than 15 years.[24] Fractional shortening at $<$28% or reduced mean velocity of circumferential fiber shortening was observed in 47% of the patients. Left ventricular fractional shortening decreased with age but was normal in

most of the patients older than 10 years of age. Five patients died of heart failure or respiratory problems.[24] Autopsy revealed myocardial fibrosis in all of them. It is interesting to note that none of the patients showed myocardial thickening or electrocardiographic abnormalities.[24] Indication for CI in the disease is also a selective deficiency of highly glycosylated α-dystroglycan not only in the skeletal muscle but also in the myocardium.[25] Absence of CI has been only rarely described.[26]

Muscle-Eye-Brain Disease

Phenotype

The phenotype is characterized by severe diffuse muscle weakness, structural brain malformation, microcephaly, severe mental retardation, eye abnormalities (congenital blindness, retinal detachment, microphthalmia), and marked scoliosis with hyperextension of the head.[21,27] Cerebral imaging may show cortical atrophy, focal white matter lesions, partial corpus callosum hypoplasia, or cerebellar vermis hypoplasia.[21] MRI may also show cortical migration defects, such as lissencephaly, pachygyria, polymicrogyria, or focal cortical dysplasia. In some cases eye involvement may be absent or mild.[21]

Genotype

The syndrome is genetically heterogeneous because of mutations in the *POMT1*, *POMT2*, *POMGnT1*, or *FKRP* genes.[21,27] Among these mutations, the most common ones occur in the *POMGnT1* gene.[28,29]

Cardiac Findings

In a patient with congenital blindness, bilateral retinal detachment, and microphthalmia, muscle biopsy revealed α-dystroglycan deficiency caused by a novel homozygous *POMGnT1* mutation.[27] No cardiac investigations were conducted for this patient. In 2 studies of 19 patients,[30] 9 of whom had MEB,[31] no cardiac abnormalities were detected. In a mouse model of the disease, however, the expression of the dystrophin-associated glycoprotein complex proteins was reduced not only in the skeletal muscle but also in cardiomyocytes.[32] In an overlay assay, binding of laminin by α-dystroglycan was absent in the skeletal muscle, myocardium, and brain.[24] At the moment it is unclear if there is CI in human MEB.

Walker-Warburg Syndrome

Phenotype

WWS is the most severe form of the dystroglycanopathies and is clinically characterized by CMD coupled with severe ocular and brain malformations (cerebellar dysgenesis, lissencephaly).[33]

Genotype

In the majority of patients with WWS, mutations in the *POMT1* and *POMT2* genes are responsible for the phenotype.[33] Less frequently, mutations in the *POMGnT1*, *FKTN*, *FKRP*, or *LARGE* genes have been identified (Table 1).

Cardiac Findings

Cardiac abnormalities seem to be absent or rare in WWS. In 2 patients with the disease, neither subclinical nor clinically manifesting CI was reported.[34] One patient with WWS had coarctation of the aorta.

CMD Type 1C (MDC1C)

Phenotype

MDC1C represents a severe form of CMD that affects the skeletal muscle and the cerebrum. Most patients present with severe general muscle weakness since the first weeks of life, muscle hypotonia, macroglossia, and calf hypertrophy and do not gain the ability to walk.[14,35,36] Single patients may develop scoliosis.[35] In severe cases, respiratory muscles may be affected, and mechanical ventilation may be necessary.[35] Some patients may develop microcephaly, mental retardation, cerebral atrophy, cortical dysplasia, white matter changes, pontine and cerebellar atrophy, or cerebellar cysts that can be seen on cranial MRI.[35,36] There may be marked elevation of serum creatine kinase levels (hyper-CK-emia), and muscle biopsy may show dystrophic changes with secondary deficiency of laminin α2 expression and marked decrease in immunostaining of α-dystroglycan but also β-dystroglycan.[14,36,37]

Genotype

MDC1C is caused by mutations in the *FKRP* gene on chromosome 19q13.3 and, thus, allelic to limb-girdle muscular dystrophy 2I (LGMD2I) and other dystroglycanopathies. *FKRP* is a member of the glycosyltransferases that mediates *O*-linked glycosylation, and the primary target is thought to be dystroglycan.[37]

Cardiac Findings

In a study of 7 patients with congenital muscle weakness, inability to walk, and calf hypertrophy, 2 presented with cardiac dysfunction. One patient had left ventricular myocardial thickening, and the other had reduced fractional shortening but normal ejection fraction on echocardiography.[35] In 2 patients from Mexico, 1 presented with nonspecific ST- and T-wave abnormalities, whereas the other had normal ECG and echocardiography results at 3 years of age.[37] In the other patients reported thus far, cardiac investigations were not mentioned.[14,36]

Although LGMD2I is allelic to MDC1C, it is clinically distinct from and more frequent than MDC1C. LGMD2I is not congenital (thus, not CMD) and typically presents with proximal muscle weakness and wasting. Because LGMD2I is more frequent than MDC1C, cardiac investigations have received more em-

phasis. In a study of 4 siblings with LGMD2I, CI was mild in 3 of them but severe in the other. The latter developed fatal cardiomyopathy.[38] In 2 patients with early-onset LGMD2I, dilated cardiomyopathy was diagnosed at 7 months and 17 years of age, respectively.[39] A single patient with LGMD2I developed heart failure and dilated cardiomyopathy at 8 years of age and required heart transplantation a few months later.[40] From a retrospective multicenter analysis of 38 patients, it turned out that 55% had developed CI.[41] Heart failure developed in 42% of the patients by 38 years of age. Fifty-eight percent of the patients had ECG abnormalities.[41] CI occurred earlier in patients who were heterozygous, rather than homozygous, for *FKRP* mutations.[41] Two thirds (4.4%) of the heterozygotes (homozygotes) were predicted to develop CI by 20 years of age and 100% by 39 years of age (58 years).[41] In a prospective trial with 38 patients with LGMD2I, 6 developed dilated cardiomyopathy.[42] In an investigation of 23 patients with LGMD2I for CI, mean systolic function was reduced compared with that in controls, and 60% of the patients had reduced ejection fraction.[43] Among a series of 3 patients, all of them presented with dilated cardiomyopathy.[44]

Other Congenital Dystroglycanopathies

Other congenital dystroglycanopathies may present clinical similarities with the phenotypes already described, manifesting muscular dystrophy and central nervous system and ocular changes isolated or combined with variable degrees of intensity. For example, in a study of 31 patients with mutations in the *POMT2* ($n = 9$), *POMT1* ($n = 8$), *POMGnT1* ($n = 7$), *FKTN* ($n = 6$), or *LARGE* ($n = 1$) gene, CI in the form of a right bundle branch block was found in 1 of them.[29] In a study of 4 patients carrying *POMT2* mutations, only 1 presented with CI in the form of myocardial thickening.[21] In a study of mentally retarded patients with CMD carrying *POMT2* mutations, only mild cardiac abnormalities were reported.[21]

At present there is a field of research directly involved with the task of identifying other glycosyltransferase genes related with the pathogenesis of this type of CMD in addition to the 6 that are already known.[29]

RIGID-SPINE SYNDROME

Phenotype

RSS is a rare CMD characterized by early rigidity of the spine and respiratory insufficiency.[45,46]

Genotype

RSS is caused by mutations in the *SEPN1* gene on chromosome 1p35.13 (Table 1).[45]

Cardiac Findings

In a study of 10 patients with RSS, auscultation findings were abnormal in half of them. Three of 9 patients showed ECG abnormalities indicative of right heart disease.[46] Holter monitoring revealed paroxysmal supraventricular tachycardia in hypoxic or hypercapnic patients. Echocardiography revealed mitral valve prolapse in 5 and pulmonary hypertension in 3 of 9 patients.[46] There was no evidence of left ventricular involvement, but in 3 patients right heart involvement including cor pulmonale as a complication of restrictive respiratory failure, right-sided heart failure, and mild incomplete right bundle branch block, were detected. Cor pulmonale was attributed to the restrictive chest wall defect and weakness of the respiratory muscles.[46] In a study of 4 patients with the disease, 1 presented with coarctation of the aorta that was repaired with balloon angioplasty.[47] Two had borderline right ventricular conduction delay.[47] In a study of 6 patients only 1 presented with heart failure at 4 years of age.[48] In a study of 11 patients from 9 families, none of the investigated subjects presented with CI.[49] Although respiratory impairment was present in a study of 11 patients, CI was absent in 7 patients who were systematically investigated for cardiac disease.[50] Overall, there seems to be predominantly right heart involvement in RSS secondary to respiratory distress. Involvement of the left ventricular myocardium occurs only in some patients.

CONGENITAL FORM OF LGMD1B (LAMINOPATHY)

Phenotype

Congenital LGMD1B is clinically characterized by congenital diffuse weakness and wasting, particularly of the cervicoaxial muscles, and dropped-head syndrome. Limb involvement is predominantly proximal in the upper limbs and distal in the lower limbs. There is early development of talipes and rigid spine with thoracic lordosis. Proximal contractures appear later, most often in lower limbs, sparing the elbows. There is early involvement of the respiratory muscles, requiring ventilatory support, and death from respiratory impairment.[51,52]

Genotype

The disorder is caused by heterozygous mutations in the *LMNA* gene on chromosome 1.[51,52]

Cardiac Findings

In a study of 15 patients with congenital laminopathy, 5 presented with CI. Two had paroxysmal atrial tachycardia, 1 had AV delay with syncope, 1 had ventricular arrhythmias, and 1 died from sudden cardiac death.[52] In a family with consanguineous parents, 2 children developed congenital muscle disease caused by germinal *LMNA* mosaicism.[53] CI in these patients developed not earlier than at the ages of 15 and 11 years, respectively.[53]

CI is also a frequent finding in noncongenital LGMD1B. In a study of 4 patients, 2 with early-onset and 1 with adult-onset LGMD1B, 3 presented with severe cardiac problems. The 9-year-old patient had dilated cardiomyopathy, the 8-year-old patient had hypertrophic cardiomyopathy, and the 57-year-old patient had atrial fibrillation.[51] In 1 of the young patients, CI preceded the muscular manifestations by years.[51] In some patients, severe ventricular arrhythmias may be associated with sudden cardiac death and may necessitate implantation of an implantable cardioverter-defibrillator (Hans Keller, MD, verbal personal communication, 2009).

CMD WITH INTEGRIN DEFICIENCY

Phenotype

In 1998, Hyashi et al[54] investigated 117 patients with unclassified congenital myopathy for integrin $\alpha 7$ expression in muscle biopsies. They found 3 patients with integrin $\alpha 7$ deficiency but normal laminin $\alpha 2$ chain expression.[54] Clinically, these patients presented with congenital myopathy with delayed motor milestones.

Genotype

All 3 patients carried mutations (splice-site mutations that cause an insertion and a deletion) in the integrin $\alpha 7$ (*ITGA7*) gene on chromosome 12q13.[54]

Cardiac Findings

To our knowledge, no cardiac investigations have been conducted on the patients reported by Hyashi et al.[54]

UNCLASSIFIED CMDs

In a study of 10 merosin-positive patients with CMD, 3 had reduced systolic function and 1 presented with dilatation of the left ventricle.[55] A Turkish group investigated 42 patients with merosin-positive CMD and found a lower mean systolic function in the patient group compared with controls, although only 3 patients had an ejection fraction of $<55\%$.[56]

CARDIAC FOLLOW-UP

Generally, all patients with CMD should systematically undergo cardiac investigations, including clinical investigation, ECG, Holter monitoring, and echocardiography, at diagnosis. Frequency of follow-up visits for monitoring CI should depend on the degree of CI. In patients at high risk for CI, such as those with merosin deficiency, dystroglycanopathies, and laminopathies, ECG, Holter monitoring, and echocardiography should be conducted yearly. In patients at high risk for arrhythmias, such as those with laminopathy or those with systolic dysfunction, Holter recordings should be conducted even more often to not overlook the indication for an implantable cardioverter-defibrillator.

CONCLUSIONS

CI in CMDs is quite variable, although it has to be admitted that most of the reported patients were not systematically investigated for cardiac disease, neither prospectively nor retrospectively. CI seems to be generally absent in Ullrich CMD and occasionally absent in Bethlem CMD, MDC1A, and in congenital LGMD1B. CI may be subclinical in up to half of the patients with merosin deficiency or mild in Bethlem CMD. CI seems to be most prevalent and most severe in patients with merosin deficiency, dystroglycanopathies, and laminopathy. For these patients the outcome may be fatal despite adequate cardiac therapy. Sudden cardiac death has been reported in some patients with laminopathy. Generally, involvement of the left ventricular myocardium seems to be more frequent than arrhythmias in patients with CMD. Concerning the onset of cardiac manifestations, CI may precede muscle involvement for years in some patients with laminopathy, but it is unknown if there are CMDs that exclusively manifest in the myocardium. CI is not at variance in patients with congenital onset and adult patients. Because no studies of CI in families with CMD have been reported, information about CI in other affected family members is scarce. Implantation of a cardioverter-defibrillator (Hans Keller, MD, verbal personal communication, 2009) or heart transplantation[23,40] may be the treatment of choice in some patients. Overall, before the frequency, severity, and therapeutic consequences of CI in CMD can be finally assessed, more data about cardiac abnormalities in these patients are required.

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Tougher to Get Into Than Medical School: *Getting a teaching job may be harder than getting into medical school. According to* The New York Times *(Winerip M, July 11, 2010), a record 46 359 applicants applied for 4500 Teach for America positions for an acceptance rate of only 9.7%. The American Association of Medical Colleges (www.aamc.org/data/facts/applicantmatriculant/table1-facts2009school-web.pdf. Accessed July 26, 2010) reports that in 2009 42 269 men and women applied to medical school with 18 390 matriculating for an acceptance rate of 43.5%. The popularity of Teach for America may stem not only from a desire to help poor children but also the promise of a steady job and livable salary for two years. Additionally, a stint with Teach for America looks good on the resume. That may explain why on many college campuses, including Dartmouth, Duke, Georgetown, University of North Carolina, and Yale, Teach for America, a program that provides teachers for low income communities, hired more seniors than any other employer. The long-term implications of a position with Teach for America are not so clear. While the vast majority of medical school applicants remain in medicine after graduation, only 15%–40% of Teach for America recruits stay beyond their two year commitment and few remain in the teaching profession beyond five years. Still, an interest in education and community service, even if brief, seems to be a good (and popular) idea.*

Noted by WVR, MD

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