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A case report of cerebral and cerebellar atrophy with cardiomyopathy and depression in a 29 years aged male

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Abstract
An unusual case of cardiomyopathy, cerebral and cerebellar atrophy with depression was presented in a 29 years male patient. The patient came to physiotherapy department with complaints of imbalance, in coordination, slurred speech and difficulty in walking. His last echocardiogram suggested left ventricular ejection fraction of 40% with moderate left ventricular systolic dysfunction, mild aortic regurgitation and dilated cardiomyopathy. MRI findings suggested diffuse cerebral and cerebellar atrophy with gliotic areas in bilateral frontal, parietal and right posterior medial temporal cortex. Patient scored low in six minute walk test and short term memory deficit was apparent on examination.

Keywords: cardiomyopathy, cerebral atrophy, cerebellar atrophy, depression

Introduction

Cardiomyopathy refers to diseases of the heart muscle. These diseases have many causes, signs and symptoms, and treatments. The heart muscle becomes enlarged, thick or rigid in cardiomyopathy, and in rare cases the muscle tissue is replaced with scar tissue [1].

Cerebral and cerebellar atrophy has been linked to disorders of the brain usually following stroke or traumatic brain injury.

Dilated cardiomyopathy is a heart muscle disorder defined by the presence of a dilated and poorly functioning left ventricle in the absence of abnormal loading conditions (hypertension, valve disease or ischemic heart disease) sufficient to cause global systolic impairment. A large number of cardiac and systemic diseases can cause systolic impairment and left ventricular dilatation, but in the majority of patients no identifiable cause is found—hence the term "idiopathic" dilated cardiomyopathy (IDC). There are experimental and clinical data in animals and humans suggesting that genetic, viral, and immune factors contribute to the pathophysiology of IDC [2]. Reinhold Schmidt *et al*. (1990) performed a study on brain magnetic resonance imaging and Neuropsychological Evaluation of Patients with Idiopathic Dilated Cardiomyopathy and found out that patients exhibited a significantly higher rate of cerebral infarcts and cortical and ventricular atrophy than controls also cognitive test performance was significantly worse in patients than controls [3]. Scott W. Sharkey *et al*. (2005) performed a study in where they found out 22 patients with acute and reversible cardiomyopathy which was seen in older women and characterized by left ventricular systolic dysfunction but had a favorable outcome with appropriate medical therapy [4].

Another study done by Chuwa TEI *et al*. on normal and dilated cardiomyopathy subjects concluded that (isometric contraction time + isometric relaxation time / ejection time) is a new, simple and reproducible Doppler index of combined systolic and diastolic myocardial performance in primary systolic dysfunction [5]. Also, cognitive deficits are often seen in patients with heart failure making their plan of care complicated so a conceptual model has been designed to help identify patients who are most likely to develop cognitive deficits [6].

Case Presentation

An unusual case of cardiomyopathy, cerebral and cerebellar atrophy with depression presented to the physiotherapy department of government physiotherapy college OPD on 1st February 2018. The patient was a 29 year old male, obese (Ht:169cm, Wt:91kgs, BMI:31.8) with chief complaints of difficulty in walking due to balance impairment, in coordination, short term memory deficits and decreased social interactions due to depression.

He had a past history of cardiomyopathy since 6th January 2016 when he presented to a general practitioner with complaints of cough and cold. When it did not resolve chest x-ray was done which reported enlarged cardiac shadow after which he was referred for echocardiogram that demonstrated dilated cardiomyopathy with left ejection fraction of 20-25% and severe left ventricular systolic dysfunction and biventricular failure. Medicines were given following which LVEF improved to 40% in Nov 2016. Also the patient was lethargic and had stopped interacting at home with family members and remained unusually silent following which they consulted a psychiatrist and medicines for depression were prescribed. In April 2017 as balance and memory started getting involved MRI was done that suggested cortical and subcortical gliotic areas in bilateral frontal, parietal and rt. Posterior medial temporal cortex. Small gliotic areas in bilateral thalamus, rt. Head of caudate nucleus and bilateral cerebellum. Cerebral and cerebellum atrophy were noted. Medicines were prescribed. As the condition progressed in

February 2018 PET CT was done which suggested moderate and focal hypo metabolism in bilateral frontal Lt>Rt predominantly in periventricular areas. Possibility of gliosis vs. demy lination and was advised for physiotherapy. The patient was currently on medications:

- Torvate chrono (sodium valporate)
- D. Venizep (serotonin norepinephrine reuptake inhibitor)
- Stilnox (hypnotic, sedative)

Discussion

A detailed physical examination of the patient was done to outline the best possible exercise regimen. On examination it was found out that the patient had cyanosis of fingertips and toes possibly as a result of decreased cardiac output (figure1) [7].

As the patient was not able to comprehend complex commands manual muscle testing was done grossly which revealed good strength in bilateral upper and lower extremity muscles. Passive range of motion was within normal limits measure with a goniometer. Tone was graded according to modified Ashworth Scale for Spasticity which was normal that is grade 5 for B/L upper and lower limbs. The deep tendon reflexes on the right side were exaggerated (table 1) [8].

Table 1

Jerks	Right side	Left side
Bicep jerk	3+	2+
Tricep jerk	3+	2+
Brachioradialis	3+	2+
Knee	3+	3+
Ankle	2+	2+
Babinski sign	Flexor response	Flexor response

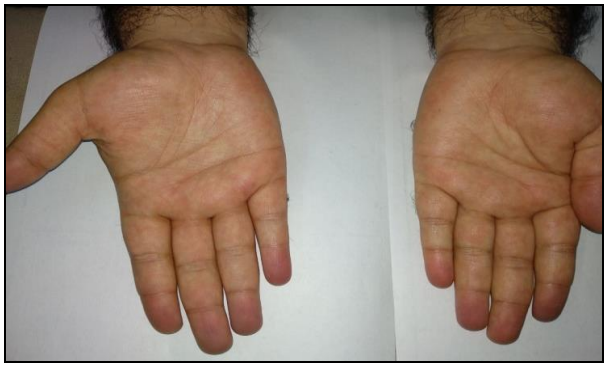

Fig 1: cyanosis in fingertips.

Balance examination was done using timed get up and go test. The normal value for adult, male is within 10sec [8]. the patient was instructed to get up from the chair, walk for 3m than turn come back to the chair, turn and sit. Time was recorded for completing the whole sequence. 3 attempts were given and average was taken. The time taken was 14.4sec on average which is more than normal.

Coordination was checked with various equilibrium and non equilibrium tests on which tandem standing, walking on toes, stair climbing without support was not possible. Mild swaying was observed on both standing with eyes open and eyes closed. Standing with eyes closed, walking sideways was affected more than the rest of them. Upper limb and lower limb coordination both were affected.

Memory was checked by asking questions and giving him things to remember. Immediate recall and long term memory are normal but patient demonstrated short term memory deficits. Perception and cognition deficits were also present like anosognosia (He maintains that there is nothing wrong with him.) and position in space (not able to place objects up, down, sideways). Left and right discrimination was present but patient took time to think and decide [8].

Gait analysis revealed slow and short steps with trunk bending on Rt. Side and taking more weight (Table 1: deep tendon reflexes) as well as wide base of support and Rt. Leg was externally rotated.

Cranial nerve examination and sensory examination

Revealed no abnormality.

An exercise protocol was prepared according to the assessment including:

- Frenkle's exercise in supine, sitting and standing [9].
- Coordination exercises including equilibrium and non equilibrium tests
- Balance exercises in front of mirror in parallel bar like tandem standing with eyes open than eyes closed.
- One leg standing and weight shifting in parallel bars.
- Fast walking
- Fine motor activities like peg board.
- Stair climbing without support
- Aerobic exercise for 10 minutes on stationary bicycle at mild to moderate intensity.

All exercises were done under the supervision of a physical therapist with proper verbal commands and progressed from fast to slow speed and increasing the complexity of commands. Appropriate rest periods were given in-between.

On 25/03/2018 patient was reassessed showing improvements in his walking speed, coordination and balance. Parents have noticed improvements in ADL's as well he has started responding to commands at home. Also six minute walk test distance was 262m but we could not compare the improvement as pre data was not present. He was able to climb stairs without support, maintain tandem stance with eyes closed for a few seconds. Overall improvements in his functional capacities were seen both by the therapist as well as the family members. Pulmonary function test was done on 4th April 2018 but no significant differences were observed except for PEFR which was only 46% of the predicted value. (Table 2) PFT results remain inconclusive as patient doesn't experience any respiratory symptoms.

Table 2: PFT (Pulmonary Function Test) values)

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Patient Information

ID	Name	Age	Height	Weight	Gender	Test Date
3	Narendrabhai israni	30 Yrs.	170 Cms.	91 Kgs	M	04-Apr-2018

Test Results

Parameter	Pred.	Pre	% Pre	Post	% Post	Imp
FVC	003.59	002.73	076	002.58	072	--05
FEV1	003.04	002.56	084	002.27	075	--11
FEV1/FVC	084.68	093.77	111	087.98	104	--06
FEF25-75	004.39	003.59	082	002.86	065	--20
PEFR	009.20	004.23	046	004.62	050	+09
FIVC	---	001.35	---	001.66	---	+23
FEV.5	---	001.98	---	001.81	---	--09
FEV3	003.48	002.73	078	002.58	074	--05
PIFR	---	004.34	---	003.68	---	--15
FEF75-85	---	001.52	---	000.91	---	--40
FEF.2-1.2	007.57	003.64	048	003.40	045	--07
FEF 25%	008.08	003.64	045	004.02	050	+10
FEF 50%	005.92	004.22	071	003.60	061	--15
FEF 75%	003.13	002.23	071	001.26	040	--43
FEV.5/FVC	---	072.53	---	070.16	---	--03
FEV3/FVC	096.94	100.00	103	100.00	103	+00
FET	---	001.51	---	002.52	---	
ExplTime	---	000.33	---	000.20	---	
Lung Age	030	035	117	000.20	127	+09
FEV6	003.59	---	---	---	---	
FIF25%	---	003.66	---	000.92	---	--75
FIF50%	---	002.35	---	000.17	---	--93
FIF75%	---	001.09	---	003.13	---	+187

Willem MA Verhoeven *et al*. studied a case report of ataxia with depression and cardiomyopathy and attributed the cause to a POLG mutation [10].

Also the clinical presentation led us to doubt that the patient was exhibiting akinetic mutism rather than depression. Akinetic mutism is characterized by profound apathy and a lack of verbal and motor output for action, despite preserved alertness [11]. The condition usually follows bilateral damage to the medial frontal subcortical circuits. The features relate to the case but evidence remains inconclusive and still needs to be investigated.

Conclusion

Therefore in patients with complex neuropsychiatric presentation extensive diagnostic evaluation is warranted including the search for mitochondriopathies to accurately determine the cause. Also, proper medical and physiotherapy management can help improve the quality of life.

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