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X-Linked Dilated Cardiomyopathy Presenting as Acute Rhabdomyolysis and Presumed Epstein-Barr Virus-Induced Viral Myocarditis: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Male, 23
Final Diagnosis: Primary X-linked dilated cardiomyopathy
Symptoms: Rhabdomyolysis
Medication: —
Clinical Procedure: —
Specialty: General and Intrenal Medicine
Objective: Rare co-existance of disease or pathology

Background: Rhabdomyolysis and primary dilated cardiomyopathies without skeletal muscle weakness are rare features of X-linked dystrophinopathies. We report a rare case of an X-linked dilated cardiomyopathy (XLDCM) presenting with acute rhabdomyolysis and myocarditis. We illustrate the confounding diagnostic influence of a reactivated, persistent EBV myocarditis as the presumed cause for this patient's XLDCM.

Case Report: A 23-year-old Australian man presented with acute rhabdomyolysis and elevated creatine kinase (CK) levels. He was managed conservatively with intravenous hydration and developed acute pulmonary edema. Cardiac MRI and transthoracic echocardiogram revealed a dilated cardiomyopathy and viral myocarditis. Extensive serological investigations identified reactivation of EBV, which was presumed to account for his viral myocarditis. The patient recovered and was discharged with down-trending CK levels. Follow-up transthoracic echocardiograms and cardiac MRI showed a persisting dilated cardiomyopathy. His CK continued to remain elevated and his EBV IgM serology remained positive. An inflammatory polymyositis with either a primary autoimmune pathophysiology or secondary to a chronic EBV infection was considered. Oral corticosteroids were trialed and reduced his CK significantly until therapy was ceased. Massively parallel sequencing eventually identified a two-exon deletion targeting Xp21 consistent with the diagnosis of a rare XLDCM.

Conclusions: Rhabdomyolysis and co-existing primary dilated cardiomyopathies are rare diagnostic manifestations in a minority of X-linked dystrophinopathies. Chronic viral infections and their reactivation may complicate the diagnostic process and incorrectly attribute an inherited cardiomyopathy to an acquired infective etiology. EBV reactivation rarely induces myocarditis. Therefore, primary and unresolving dilated cardiomyopathy with persistently elevated CK must prompt consideration of an underlying dystrophinopathy.

MeSH Keywords: Cardiomyopathy, Dilated • Epstein-Barr Virus Infections • Muscular Dystrophies • Rhabdomyolysis

Abbreviations: **ACE-I** – angiotensin-converting enzyme inhibitor; **ANA** – antinuclear antibodies; **ANCA** – anti-neutrophil cytoplasmic antibodies; **BMD** – Becker's muscular dystrophy; **C3/C4** – complement C3, C4; **CMV** – cytomegalovirus; ***DMD*** – dystrophin gene; **EBNA** – Epstein-Barr nuclear antigen; **EBV** – Epstein-Barr virus; **GBM** – anti-glomerular basement membrane; **HTLV** – human T cell lymphotropic virus; **IgG** – immunoglobulin G; **IgM** – immunoglobulin M; **m** – meter; **L** – liter; **min** – minute; **mL** – milliliter; **MRI** – magnetic resonance imaging; **U** – units; **XLDCM** – X-linked dilated cardiomyopathy; **µg** – microgram; **µmol** – micromoles; **%** – percent

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Background

X-linked dystrophinopathies encompass a heterogenous group of skeletal muscle disorders (e.g., Duchenne's muscular dystrophy and Becker's muscular dystrophy) with exon mutations targeting the dystrophin gene (*DMD*) on Xp21 [1,2]. Duchenne's muscular dystrophy is often more severe and presents in early childhood with profound disability [2]. In contrast, Becker's muscular dystrophy (BMD) typically presents in late childhood and early teenage years with mild skeletal muscle weakness and associated gait abnormalities [1,2]. A review of magnetic resonance imaging (MRI) investigations in a small cohort of BMD patients has shown that >80% of the thigh muscle groups usually appear abnormal with worsening atrophy [3]. This progresses towards abnormal pseudohypertrophy of the calves, increasing weakness, and immobility by the third decade of life [1,2,4]. The development of symptomatic hypertrophic and/or dilated cardiomyopathies with systolic dysfunction coincides with this period [1,2,5]. The incidence of cardiomyopathy in BMD following skeletal muscle involvement is estimated at 47.0–68.4% [3,6]. The clinical severity of BMD does not correlate with the underlying *DMD* lesion [1,7]. Similarly, the degree of symptomatic cardiomyopathy appears to be mutually exclusive of a patient's concurrent skeletal myopathy [1,2].

Clinical features suggestive of an underlying BMD include persistently elevated creatinine kinase (CK) levels, exertional myalgia, intermittent and self-resolving myoglobinuria, and calf hypertrophy [1,2,4]. Skeletal muscle involvement and atrophy are considered primary clinical features, while cardiomyopathy is viewed as a progressive complication of the disease [1–6]. A minority of BMD patients present with rhabdomyolysis or a primary dilated cardiomyopathy without skeletal muscle involvement [8–16]. However, such cases are extremely rare. The latter is often classified as an X-linked dilated cardiomyopathy (XLDCM) and represents a rare subgroup of BMD [1,2]. These manifestations contribute to the clinical variability and diagnostic challenges of the dystrophinopathies.

We report the first Australian case of a 23-year-old man, who presented with acute exercise-induced rhabdomyolysis complicated by a presumed Epstein-Barr virus (EBV) myocarditis. He was eventually diagnosed with a rare XLDCM. We highlight the diagnostic processes and pitfalls in this case, with specific focus directed towards the confounding influences of an initial rhabdomyolysis presentation and dilated cardiomyopathy presumed to be secondary to chronic EBV reactivation. Ongoing therapeutic management and multidisciplinary recommendations are provided in the setting of a rare and primary XLDCM.

Case Report

A 23-year-old Caucasian man, previously fit and well, presented with a history of acute and worsening lower abdominal and bilateral loin pain, severe bilateral calf cramping and edema, and dark-brown-colored urine. He had completed a 10-kilometer, military style obstacle course the day before and had collapsed from exhaustion after the event. Collateral history obtained from his family noted that he had been lethargic during the week leading up to the event and appeared fatigued while running downhill for the first kilometer of the course. He had no active medical issues, nor a personal or family history of autoimmune or underlying muscle disease. He denied taking any regular medications. He undertook daily muscle building exercises prior to this admission and ran five kilometers several times per week. His vital signs were within normal limits, but he appeared clinically dehydrated. Systems examination elicited generalized tenderness across both loins on bimanual palpation and tender calves with profound left calf swelling. He showed no signs of muscular atrophy and appeared toned. His echocardiograph showed a regular sinus bradycardia but was otherwise unremarkable. His chest X-ray was essentially normal (Figure 1A). His urine appeared dark and urinalysis identified myoglobinuria, hematuria, and proteinuria. Initial blood investigations revealed an impressively elevated CK (199 390 U/L), high creatinine (146 µmol/L), and low estimated glomerular filtration rate (58 mL/min/1.73 m^2). A diagnosis of exercise-induced rhabdomyolysis with acute kidney injury was made.

His calf edema was reviewed by an orthopedic surgeon and compartment syndrome was excluded. He was aggressively resuscitated with intravenous fluids to maintain a minimum urine output of 200 mL/h. A renal tract ultrasound showed no hydronephrosis or obstruction. He subsequently developed acute pulmonary edema and type I respiratory failure, necessitating intensive care admission and bilevel positive airway pressure support. He became febrile and his chest X-ray showed extensive fluid overload (Figure 1B). Serum troponin-I was elevated (0.41 µg/L) and a transthoracic echocardiogram showed that the left ventricle was severely dilated, with moderate global systolic impairment (ejection fraction 38%). Concurrent moderate mitral regurgitation, left atrial dilatation, and mild pulmonary hypertension were also noted. All abnormal cardiac features were presumed to be secondary to excess fluid overload at this time. A cardiac MRI was performed, which showed features consistent with viral myocarditis (Figure 1C, 1D). Extensive serological investigations for infective etiologies (EBV, CMV, Q-fever, *Leptospira* species, *Legionella* species, *Mycoplasma* species, influenza A, influenza B, parainfluenza 1, parainfluenza 2, parainfluenza 3, adenovirus, respiratory syncytial virus, and human metapneumovirus) and autoimmune screen (ANA, ANCA, C3/C4 levels, and anti-GBM antibody) were performed. EBV IgM levels were positive in addition to detected EBV IgG

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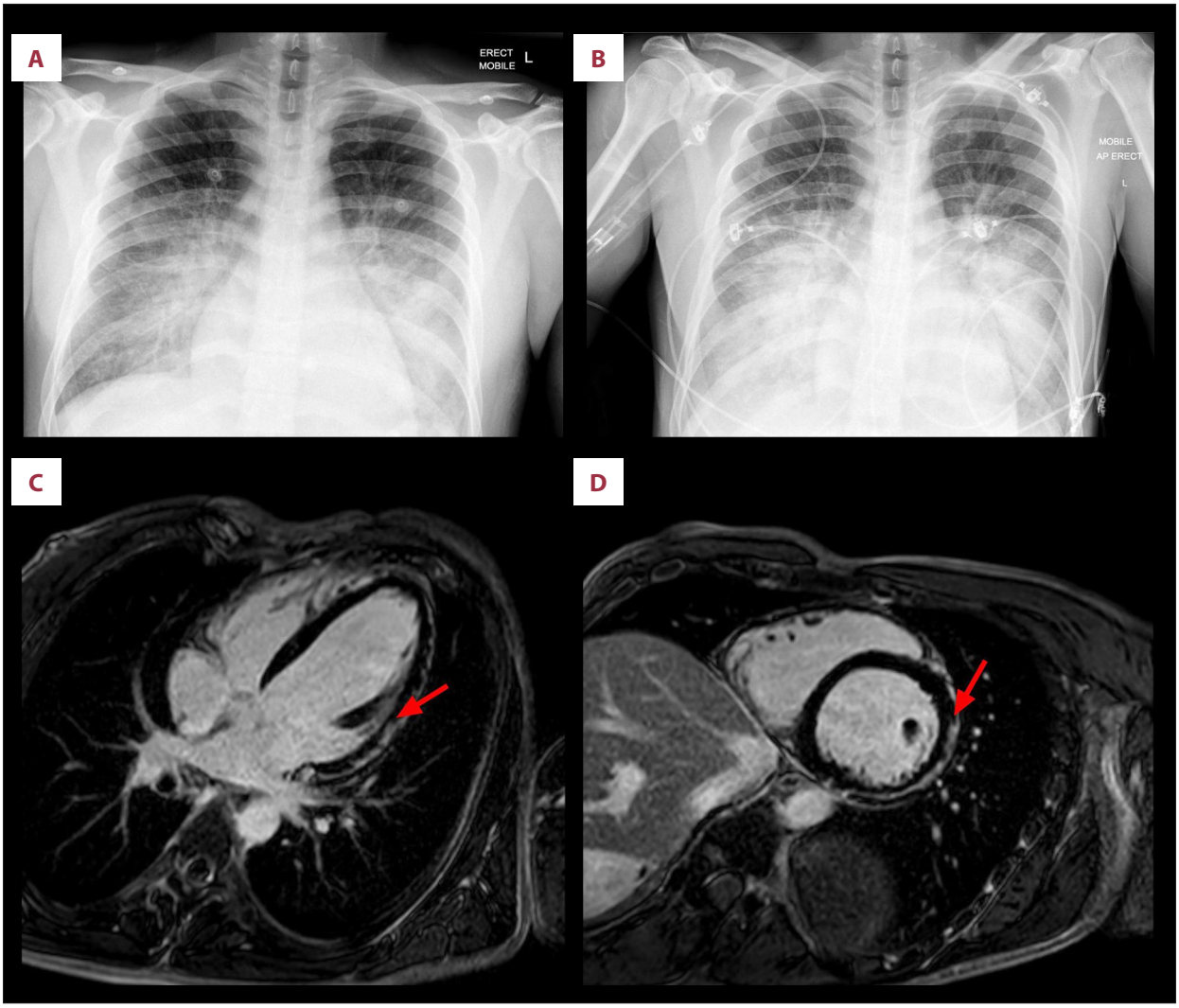

Figure 1. Chest X-ray and cardiac MRI investigations performed during the patient's hospital admission. (**A**) PA chest X-ray shows no evidence of cardiomegaly. A small, non-specific right lower lobe pulmonary infiltrate was noted. (**B**) Progressive AP chest X-ray shows extensive infiltrates in the middle and lower zones consistent with acute pulmonary edema. (**C, D**) Cardiac MRI shows low-grade, subepicardial enhancement at the lateral aspect of the lateral ventricular wall, which was more pronounced on the basal to middle portions of the left ventricle (arrows). These features were suggestive of a viral myocarditis. AP – anterior-posterior; PA – posterior-anterior.

and EBNA IgG. All other investigations were negative. His viral myocarditis was presumed to be caused by the reactivation of EBV, although he had no coryzal symptoms, lymphadenopathy, or abdominal organomegaly. An endomyocardial biopsy was not performed, as the result was unlikely to alter his current management and the procedure was deemed to be associated with significant risk. His creatinine peaked at 689 µmol/L and returned to normal prior to his discharge, without the need for dialysis. His CK level reduced to 7477 U/L. His acute pulmonary edema resolved and he remained asymptomatic in terms of his viral cardiomyopathy. Bisoprolol 1.25 mg once daily was commenced and he was discharged 20 days following his admission, with specialist follow-up.

Cardiological review with repeat transthoracic echocardiogram at one month following his discharge from hospital (50 days after admission) identified a persisting dilated cardiomyopathy with mild improvement in systolic function (ejection fraction 45%). It was thought at this time that the slow improvement in his cardiac function was related to an underlying, chronic EBV myocarditis. Pulmonary pressures had normalized and his repeat troponin-I serology was negative. Ramipril 1.25 mg once daily was commenced in addition to the continuation of bisoprolol to provide further cardiac protection given his persisting dilated cardiomyopathy.

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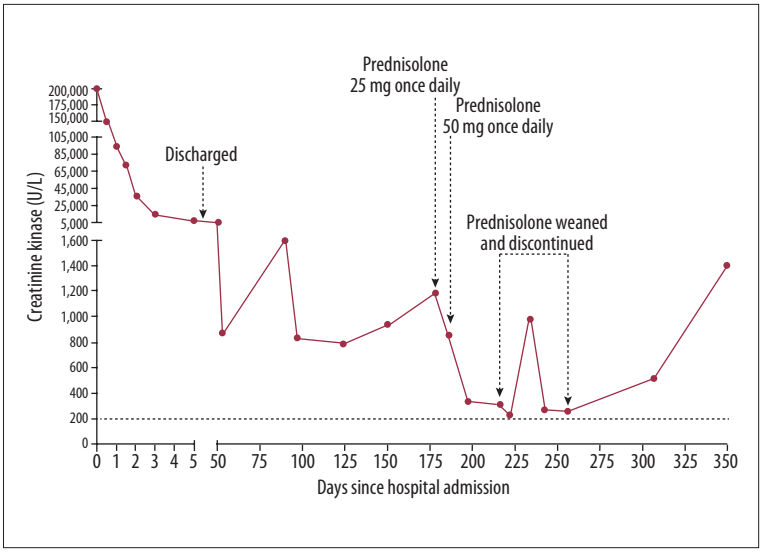

Figure 2. Creatine kinase (CK) levels during the patient's admission and outpatient follow-up after discharge from hospital. CK levels were massively elevated during the patient's admission (peak 199370 U/L), which was consistent with a diagnosis of rhabdomyolysis. CK levels reduced but remained elevated following the patient's discharge from hospital. A trial of prednisolone 25 mg once daily for one week followed by a subsequent weaning schedule of prednisolone 50 mg once daily significantly reduced CK levels from 1181 U/L to 229 U/L. Discontinuation of corticosteroid therapy resulted in CK levels increasing again to a maximum level of 1400 U/L prior to the patient's diagnosis of an XLDCM. Dotted horizontal line represents the upper reference limit for CK levels (200 U/L). CK – creatine kinase; L – liter; mg – milligrams; U – units.

Interestingly, his repeat CK remained significantly elevated at 6425 U/L (Figure 2) and only later reduced to 830 U/L at 97 days after his hospital admission. Comprehensive specialist general physician review elicited no previous history or peripheral stigmata of autoimmune disease (e.g., oral ulcers, alopecia, arthralgia, myalgia), muscle disease (e.g., atrophy, fasciculations, Gottron's papules, nailfold erythema, heliotrope rashes, dysphonia, dysphagia), or congestive cardiac failure. The patient had made a full recovery and was undertaking regular toning exercises at a gym and cycling 12 kilometers to work daily without myalgia or recurrent rhabdomyolysis. However, his muscle bulk was noted to be reduced in comparison to his admission. A suspected primary diagnosis of an underlying inflammatory myopathy was considered [17]. Differential diagnoses included polymyositis, connective tissue disease, metabolic myopathies (e.g., carnitine palmityl phosphotransferase myopathy), dystrophinopathies, and idiopathic chronic myositis, which are diseases that all present with persistently elevated CK levels. Additional investigations, including a repeat autoimmune screen, myositis antibodies (Ro52, EJ, OJ, PL-12, PL-7, SRP, Jo-1, PM-SCl 75, PM-Scl 100, Ku, and Mi-2), and HTLV-I/II viral serology, were negative. Repeat EBV serology testing on two occasions showed a persisting positive EBV IgM and EBNA IgG. CK levels remained elevated up to 178 days after admission (Figure 2). An MRI of his thighs was unremarkable (Figure 3), so muscle biopsy was considered to be of low diagnostic yield, as almost all cases of dystrophinopathies show some degree of thigh muscle atrophy [3]. Muscle biopsies are also not required during the routine diagnostic workup for dystrophinopathies [2].

A trial of prednisolone 25 mg once daily over one week reduced his CK levels to 858 U/L. Further reductions to 229 U/L were achieved with a follow-up course of prednisolone 50 mg once daily for two weeks with progressive weaning. Subsequent corticosteroid cessation resulted in his CK increasing again to 522 U/L and 1400 U/L (Figure 2). The differential diagnosis was revised to conform to an underlying inflammatory or congenital pathology. An atypical inflammatory polymyositis with associated EBV reactivation which had responded to corticosteroid therapy was suspected. An isolated case series of atypical polymyositis caused by chronic EBV infection has been previously published [18]. However, these patients had shown no response to corticosteroids [18]. In addition, BMD and McArdle's disease were also considered given his persisting dilated cardiomyopathy and precipitating exercise-induced rhabdomyolysis. However, these were thought to be unlikely, as the latter does not typically respond to corticosteroids [19]. Moreover, only a small percentage of BMD patients with skeletal muscle involvement have benefitted from corticosteroid therapy [20,21]. In the interim, ongoing cardiological review with repeat transthoracic echocardiogram had revealed no further improvement in the patient's dilated cardiomyopathy and enduring systolic impairment (ejection fraction 45%). Repeat cardiac MRI nine months following his hospital admission showed no changes, with subepicardial hyperenhancement remaining at the lateral aspects of the lateral ventricular wall. Ramipril was increased to 2.5 mg once daily and bisoprolol was discontinued. The patient remained asymptomatic and showed no signs of congestive cardiac failure.

Neurogenetic opinion was sought and, given his ongoing dilated cardiomyopathy and persistently raised CK, a peripheral blood sample was collected to screen for congenital muscular dystrophies. Massively parallel sequencing identified

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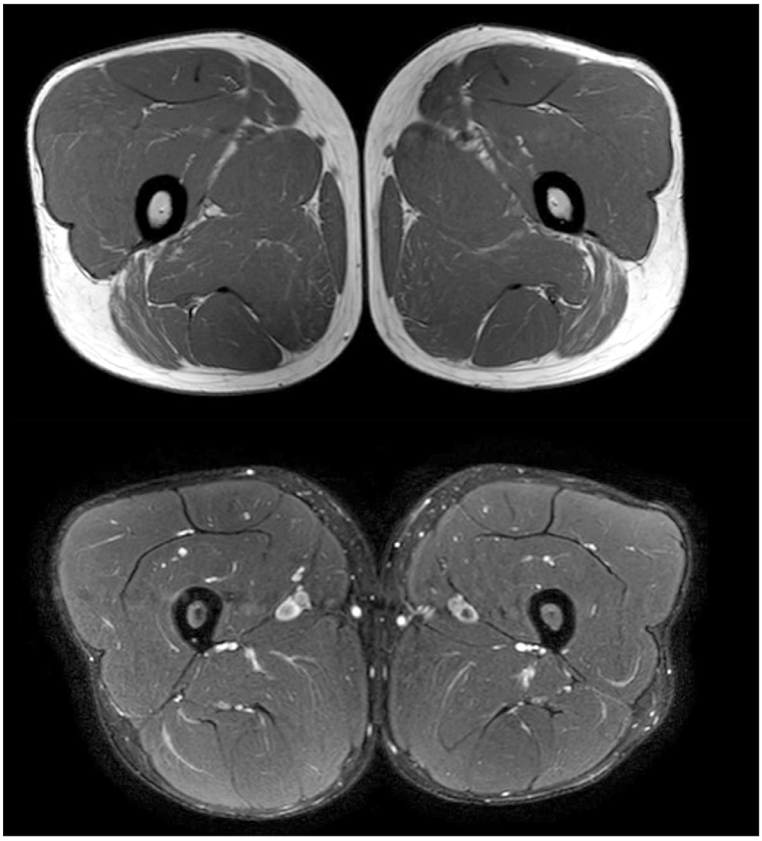

Figure 3. MRI of the bilateral thighs. All muscles and intermuscular fascia showed no evidence of edema or an inflammatory myopathy.

an in-frame deletion spanning exons 48 and 49 of the *DMD*; c.(6912+1_6913-1)_(7200+1_7201-1)del. A *DMD* deletion involving exons 48 and 49 has been previously described in BMD [11, 22, 23]. A diagnosis of a rare primary XLDCM was subsequently made.

Discussion

The dystrophinopathies represent a heterogenous cluster of congenital muscle disorders which typically present with progressive proximal skeletal muscle weakness of the upper and lower limbs [1,2]. Few cases have been described in which rhabdomyolysis and manifesting dilated cardiomyopathy are the primary diagnostic features of a dystrophinopathy, although these are known complications in BMD [1,2,8–16]. To the best of our knowledge, we have reported the first Australian case of a rare XLDCM in a 23-year-old who presented with exercise-induced rhabdomyolysis and a presumed, reactivated EBV myocarditis.

Rhabdomyolysis is an infrequent clinical presentation associated with BMD, let alone in the subgroup of patients with a primary XLDCM [14–16]. A previously published case report of an 18-year-old American man with undiagnosed BMD who presented with rhabdomyolysis had similar clinical features to that observed in our patient [16]. Following the resolution of his rhabdomyolysis, his CK levels remain elevated at 600–36000 U/L (median 1200 U/L) and reflects a similar fluctuating trend to that of our patient (Figure *2*). The authors noted that recurrent exercise-induced rhabdomyolysis and persistently elevated CK levels may be a feature associated with BMD and should form part of the differential when such clinical presentations arise [16].

Our case also represents a unique diagnostic challenge confounded by a presumed chronic EBV reactivation and associated viral myocarditis. Endomyocardial biopsy analyzes have shown that EBV reservoirs do reside within the myocardium (incidence: 1–6%) and may facilitate the development of a dilated cardiomyopathy with associated left ventricular systolic dysfunction [24–27]. However, EBV-related cardiomyopathies are uncommon phenomena and have never been reported in dystrophinopathies. Two cases with EBV myocarditis in immunocompetent adults have been previously described [27,28]. Of these, one patient with dilated cardiomyopathy was identified to have a chronic EBV infection isolated from his endomyocardial biopsy over a 31-month follow-up period [27]. Interesting, a murine model has shown that viral infections contribute to the appearance of an inflammatory cardiomyopathy in mice lacking dystrophin [29], but this has yet to be reported in human XLDCM. Concurrent EBV IgM and EBNA IgG positivity in immunocompetent individuals has

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been associated with late primary infectious mononucleosis and subclinical EBV reactivation [30,31]. Such clinical scenarios can only be distinguished by high-avidity EBV IgG testing [30]. Given our patient's EBV IgM, EBNA IgG, and EBV IgG positivity, and lack of classical symptoms bar fever during his hospital admission, we entertained a diagnosis of EBV myocarditis as a cause for his dilated cardiomyopathy. This is unusual, as EBV IgM levels usually decline after two to six months [31] and may therefore represent a subclinical EBV myocarditis with chronicity. An endomyocardial biopsy would be required to confirm the presence of an EBV reservoir, but this was not indicated and would not alter the longitudinal management of our patient's asymptomatic XLDCM. Nevertheless, the confounding influence of a presumed chronic EBV reactivation represented an unavoidable pitfall in the diagnostic workup of our patient as a case of viral myocarditis and atypical polymyositis.

The distinctiveness of our patient is that he developed a primary dilated cardiomyopathy without any skeletal muscle involvement, which is grossly atypical for dystrophinopathies. To the best of our knowledge, very few case reports have described patients with primary XLDCM preceding skeletal muscle abnormalities [8–13]. In two of these cases, patients required heart transplants for symptomatic systolic dysfunction and life-threatening heart failure sequelae [10,12]. Finsterer et al. (1999) demonstrated in their case that corticosteroid therapy following heart transplant exacerbated the elevation of their patient's CK levels [10], which is at odds with the observations in our patient. It is likely that this variation is related to different corticosteroid dosing schedules between our patients. Our patient was trialed on a short course of prednisolone while multiple, long-term immunosuppressive agents used by Finsterer and colleagues induced a toxic myopathy [10]. While cardiac involvement is a known complication of BMD, it rarely presents as the primary manifestation and has been associated with profound cardiac morbidity [8–13]. XLDCM patients with mutations targeting exons 48 and 49 have been previously described and are associated with elevated CK levels [9,23]. However, such *DMD* lesions are associated with symptomatic cardiomyopathy occurring in the third decade of life [22]. If cardiomyopathy presents during the early twenties, it is often more severe and conforms to a congestive cardiac failure phenotype [8]. These reports do not correlate with the asymptomatic cardiomyopathy observations seen in our patient's XLDCM. This provides further evidence for the phenotypic heterogeneity featured across the dystrophinopathies despite a common underlying genetic lesion.

Therapeutic options for patients with a primary XLDCM are not well described. The utilization of angiotensin-converting enzyme inhibitors (ACE-I) and diuretics has been cited to improve systolic and diastolic dysfunction in BMD with complicating dilated cardiomyopathies [1,2]. The former also abrogates sympathetic hyperactivity in the heart [1]. With progressive systolic heart failure and recurrent cardiac arrests, a heart transplant is indicated and has been previously described in isolated case reports, with positive prognostic effect [10,12]. Therefore, ongoing cardiac follow-up, routine echocardiograms, and ACE-I drugs are necessary to prevent cardiac failure and/or arrhythmias, and to avoid poor cardiac outcomes in this subgroup of patients. From the anesthetic perspective, the use of halothanes and succinylcholine during general anesthesia has resulted in catastrophic cardiac arrest, rhabdomyolysis, and the deaths of several children [14,15]. Thus, comprehensive anaesthesia review prior to any surgery is needed to ensure that depolarizing and volatile agents are excluded to avoid severe cardiac arrest and rhabdomyolysis in this subgroup of BMD patients with a primary XLDCM.

Conclusions

We have reported the first Australian case of an XLDCM who was complicated by acute exercise-induced rhabdomyolysis and presumed chronic EBV reactivation. We have highlighted the rarity and diagnostic challenges of a primary XLDCM. In addition, viral myocarditis caused by chronic EBV reactivation is an unusual phenomenon, although inflammatory myocardial changes in a dystrophin-deficient murine model secondary to concurrent viral infections have been reported [29]. This raises the possibility that our patient is the first XLDCM case to be described with complicating EBV myocarditis. Nevertheless, viral myocarditis in such settings likely presents a confounding clinical factor that delays and obscures the diagnosis of a primary muscle disorder. Patients presenting with enduring elevated CK levels and a persisting cardiomyopathy should prompt an investigation into an underlying dystrophinopathy. Ongoing cardiological surveillance and thorough anesthesia review prior to any intended general anesthesia is required to avoid cardiac morbidity and associated mortality in this minority of BMD patients with a primary XLDCM.

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Conflicts of interest

None.

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