Surgical treatment of pediatric moyamoya disease: own experience and review of the literature

Leczenie operacyjne choroby moyamoya u dzieci. Doświadczenia własne i przegląd piśmiennictwa

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STRESZCZENIE

Choroba moyamoya (*moyamoya disease* – MMD) to rzadka, uogólniona, w większości przypadków postępująca i nadal słabo poznana choroba naczyń krwionośnych mózgu, prowadząca do ich niedrożności i rozwoju charakterystycznego krążenia obocznego, widocznego w angiografii mózgowej. MMD jest jedną z niewielu chorób naczyń mózgu, gdzie interwencja chirurgiczna może zmienić przebieg kliniczny choroby, zapobiec jej progresji i znacznie poprawić rokowanie. **Celem pracy** była prezentacja własnych doświadczeń odnośnie chirurgicznego leczenia MMD u dzieci i przegląd piśmiennictwa. **Materiał i metoda**. Analiza obejmowała 2 dziewczynki i 2 chłopców w wieku od 5 do 11 lat w chwili przyjęcia (średnia wieku 7,7 roku), leczonych w latach 2003–2011 z rozpoznaniem MMD (średni okres obserwacji 6,2 roku). Dane uzyskano metodą analizy retrospektywnej dokumentacji medycznej. Zastosowano leczenie operacyjne metodą trepanacji wielootworkowej synangiozy okostnowo-mózgowej umożliwiającej zaopatrzenie w krew mózgu przez naczynia okostnej u 3 dzieci, podczas gdy 1 było leczone zachowawczo. **Wyniki**. Wszystkie dzieci z grupy badanej żyją, nie wystąpiła u nich progresja choroby ani nie pojawiły się żadne nowe ubytkowe objawy neurologiczne. **Wnioski**: 1) Leczenie MMD musi być zindywidualizowane z powodu nieprzewidywalnego przebiegu klinicznego. 2) Leczenie operacyjne MMD metodą trepanacji wielootworkowej i synangiozy okostnowo-mózgowej jest relatywnie bezpieczne i skuteczne. 3) Leczenie MMD powinno być wdrożone przed wystąpieniem ciężkiego i nieodwracalnego uszkodzenia mózgu. 4) Pacjenci z MMD wymagają ścisłego monitorowania ciśnienia tętniczego podczas znieczulenia dla uniknięcia jego spadków, grożących niedostateczną perfuzją mózgu. 5) Podwyższona leukocytoza i trombocytoza we krwi obwodowej naszych pacjentów może być wskazówką wyjaśniającą patomechanizm MMD – stan zapalny i nadkrzepliwość.

Słowa kluczowe: moyamoya, leczenie operacyjne, dzieci, synangioza okostnowo-mózgowa

ABSTRACT

Background. Moyamoya disease (MMD) is a rare, generalized, mostly progressive and still poorly understood disease of brain vessels, resulting in their occlusion and development of characteristic tangles of collateral circulation visualized by classic cerebral angiography. MMD is one of the few diseases of brain vessels where surgical intervention may change the clinical course, prevent progression and significantly improve prognosis **Aim of paper:** presentation of own experience concerning surgical treatment of MMD and review of pertinent literature. **Material and method**. Study population consisted of 4 children with MMD (2 boys and 2 girls), aged 5–11 years at the time of admission (mean age 7.7 y), treated since 2003 thru 2011 (mean follow-up: 6.2 y). Data were obtained based on the analysis of medical records. **Results**. All three children operated on and one patient treated conservatively are alive and well and did not experience disease progression nor development of new deficits. **Conclusions:** (1) Treatment of MMD must be individualized due to imprevisible clinical course; (2) Surgical treatment of MMD by multi-burr hole encephalo-duro-periosteal synangiosis is relatively safe and effective. (3) Effective treatment of MMD should be instituted prior to the development of severe and irreversible brain damage; (4) MMD patients require careful monitoring of blood pressure during and after surgery in order to maintain adequate cerebral blood perfusion. (5)Elevated leukocytosis and thrombocytosis in the peripheral blood may suggest possible pathogenetic mechanism – inflammation and hypercoagulation.

Key words: moyamoya disease, surgical treatment, encephalo-duro-periosteal synangiosis, children

INTRODUCTION

Moya-moya disease („puff of smoke" in Japanese; MMD) is a rare, generalized, usually progressive and still poorly understood disease of large and medium-sized brain vessels. MMD results in their progressive occlusion with development of characteristic "puffy" tangles of collateral circulation visualized by classic cerebral angiography. At the same time, normal arteries may be absent. The disease manifests

itself by recurrent ischemic or hemorrhagic strokes. Their neurological sequels accumulate, resulting in progressive and irreversible disability. MMD usually develops in young people, in an obvious and highly unfavorable way affecting their health status and prognosis.

At the same time, MMD is one of the few generalized diseases of brain vessels where surgical intervention may change the clinical course, prevent progression and significantly improve prognosis. Thus the importance of diagnostic vigilance of pediatricians and neurologists, who are usually the first-line medical professionals confronted with young people with stroke. Therefore the key issue is timely and effective intervention before the development of irreversible and extensive brain damage.

AIM OF PAPER

The aim of this paper is to present the experience of the Dept. of Neurosurgery, The Children's Memorial Health Institute concerning surgical treatment of MMD and to review pertinent literature dealing with this subject.

MATERIAL AND METHODS

Study population consisted of 4 children (2 boys and 2 girls), aged 5 to 11 years at the time of admission (mean age: 7.7 y) treated at our facility with the diagnosis of CMM since 2003 thru 2011 (mean follow-up: 6.2 y). The study is based on the analysis of medical records of the treated patients.

Three children underwent encephalo-duro-periosteal synangiosis m. Suzuki, while one was treated conservatively. Basic demographic and clinical information concerning the study population is summarized in Table I.

Surgical treatment was performed under general anesthesia and consisted of bicoronal skin incision, elevation of anterior and posterior scalp flaps, incision of *periosteum* in the form of multiple "V" (9 on each side, overlying the sensorimotor cortex), drilling of a burr hole at the base of periosteal "V", cruciate incision of the *dura* and placement of periosteal pedicled flaps directly over the cerebral cortex. Scalp incision was closed in the standard fashion in layers over a subcutaneous gravitational drain. Drainage was removed after 2-3 days and sutures were removed on the 10-th or12-th postoperative day.

RESULTS

Results obtained from the 2 to 10 years (mean: 5.7 y) follow-up are satisfactory. All children operated on and the patient treated conservatively are alive and well and did not experience disease progression nor development of new deficits. Perioperative complication in one patient (KB) in the form of brain infarct resulting from transient drop of blood pressure during anesthesia did not lead to any new permanent neurological deficit.

Table I. Basic clinical data of MMD patients treated at the Children's Memorial Health Institute

Basic demographic data	Medical history, co-morbidities, presenting symptoms.	Imaging studies	Laboratory findings	Treatement	Outcome
MS, boy l.5	Headaches, no neurological deficits.	Multifocal stenosis of brain vessels with collateral circulation, no ischemic lesions Hypoperfusion of right hemisphere.	BG: B(+) L 16-18 K Plts. 440-513 K B ↑	Conservative	Follow-up 4 y. Neurologically stable and intact, no disease relapses.
JG, girl 7y	Recurrent stokes, permanent rightsided hemiparesis.	Post-ischemic foci in the left frontal and central region. Left ICA and MCA occlusion. Collateral circulation.	BG: O (+) L 8-14 K Plts. 230-315 K M ↑	Surgical. No hypotension at surgery.	Follow-up: 10y No disease relapses after surgery.
KB, girl 8y	Recurrent TIAs; w 5 r.ż. stroke with rightsided hemiparesis and aphasia at age 5.	Bilateral post-ischemic foci, mainly in the left fronto-temporal area. Bilateral ICA and PCA stenosis, collateral circulation.	BG: O (+) L 8-15 K Plts 266 K PMNL ↑	Surgical. Hypotension at surgery. Post-op bilateral ischemic foci in frontal lobes.	Follow-up 8 y. No disease relapses after surgery.
MK, boy 11y	Hemophilia A. TIA at age 10 (transient aphasia and right-sided hemiparesis); later complete stroke with left-sided hemiparesis.	Ischemic foci in the right parietal region. Bilateral ICA and MCA stenosis, collateral circulation.	BG: A (+) L 6-10 K Plts 154-309 K B ↑	Surgical. No hypotension at surgery. Perioperative substitution of factor VIII..	Follow-up 2 y. No disease relapse after surgery.

Abbreviations: B – bazophiles; BG – blood group; ICA – internal carotid artery; L – leukocytes; M – monocytes; MCA – middle cerebral artery; PCA – posterior cerebral artery; Plts – platelets ; PMNL – polymorphonuclear leukocytes; TIA – transient ischemic attack.

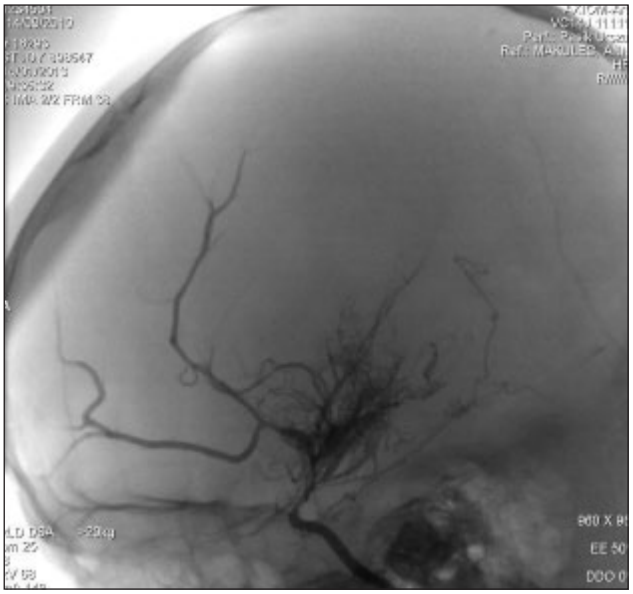


Fig. 1. Patient JG. Preoperative cerebral angiogram showing occlusion of major cerebral arteries and typical tangles of collateral circulation.

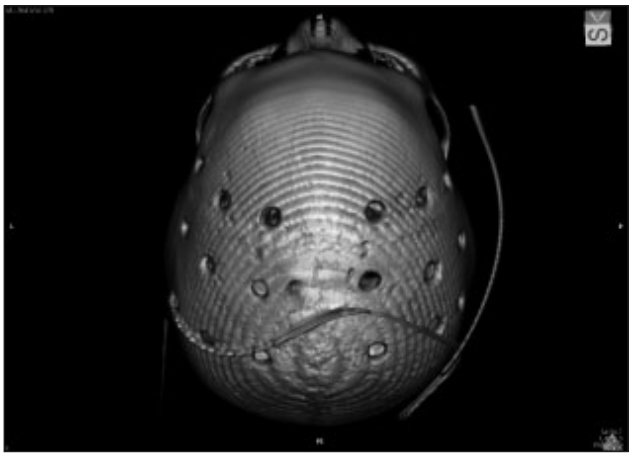


Fig. 2. Postoperative 3-D CT reconstruction of patient's calvarium showing multiple burr-holes and subcutaneous drain.

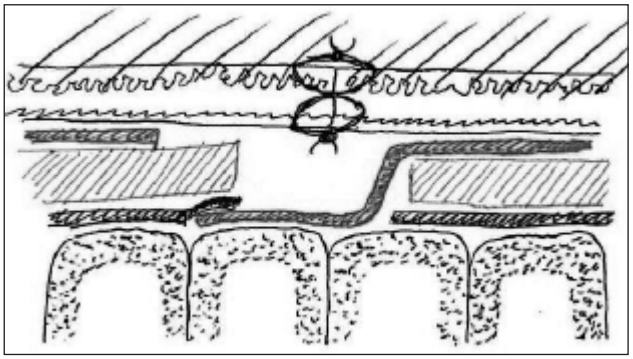
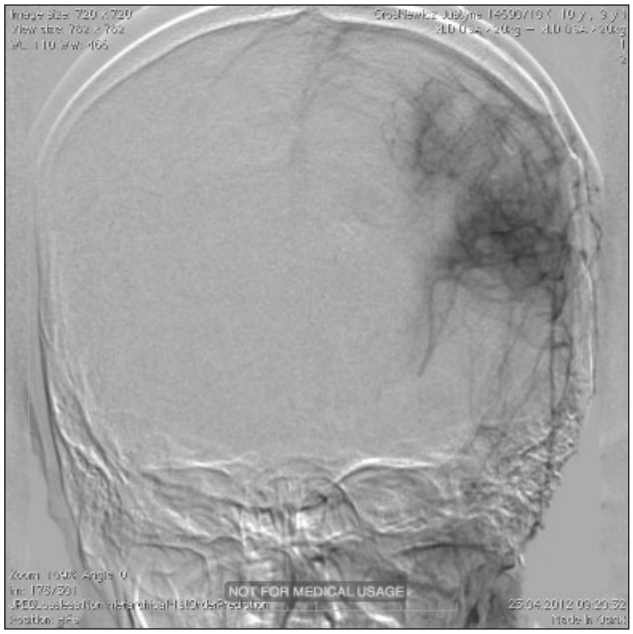


Fig. 3. Schematic drawing showing cross-section view of individual burr hole after surgery (pedicled periosteal flap overlying brain surface).



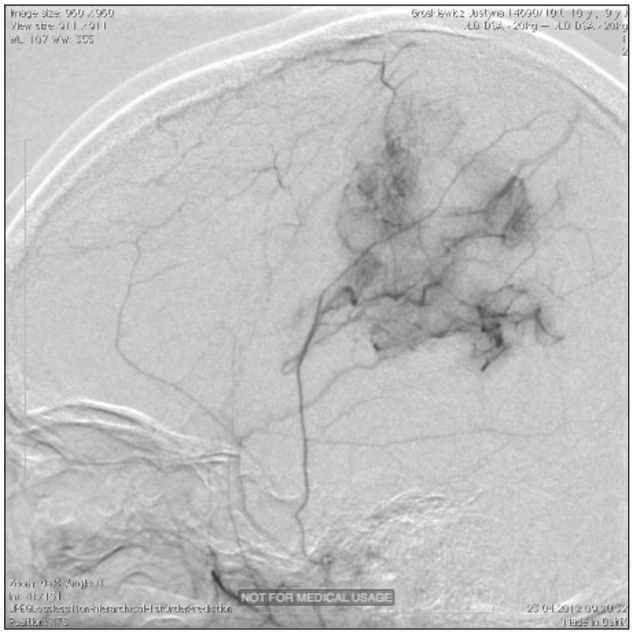


Fig. 4. Postoperative angiogram (A-AP view and B-lateral view) showing new vessels supplying the brain originating in branches of superficial temporal artery sprouting from multiple periosteal flaps overlying the cortex. Contrast medium administered to the external carotid artery visualizes superior sagittal sinus, thus proving passage through the brain tissue.

Discussion

In spite of its rarity, MMD benefits from a surprisingly large body of publications, concerning its pathogenesis (still poorly understood), epidemiology, attempts at conservative and surgical treatment and rehabilitation techniques. Web search at the time of preparing this manuscript (March 2014) yielded 56 publications concerning MMD appearing since the beginning of 2014 only. There are also several interesting papers to this respect in the Polish medical lit-

erature, so general information concerning MMD will be discussed only briefly [1–4].

A rare, generalized and progressive occlusion of brain vessels, clinically manifested by recurrent strokes resulting in the development of characteristic disseminated tangles of collateral circulation seen at angiography was first described by in 1957 [5]. The term "moya-moya" ("puff of smoke" in Japanese) first appeared in 1969 [6]. The disease consists of progressive thickening of the *intima* of medium-sized brain arteries as a result of an autoimmune reaction and edema, fibrosis and alteration of elastic *laminae* [7], leading to occlusion or rupture of the involved vessel(s) and hemorrhagic or ischemic brain infarcts or intraventricular hemorrhage [8]. According to a recently advanced hypothesis, MMD may be a 2-step process, i.e. primary obliterative vasculopathy and secondary proliferative response [9,10]. A possible etiological factors for MMD include alterations of vascular smooth muscle cells (SMC) response to platelet-derived growth factor (PDGF), systemic factors promoting migration and proliferations of SMC from the media to the *intima* [9]. Thrombophilia has been found in a considerable (1/3) proportion of MMD patients [10]. Genetic background of MMD (association with certains HLA alleles) is well established [11].

MMD has a double-peak incidence, usually manifested during the first and third-fourth decades, more often in the Asians and in the females (male:female ratio is as 1:1.8) [12]. In our material, the patient's mean age was 7.7 y, confirming the literature data, while gender distribution was symmetrical (2:2). Small size of our sample (n = 4) does not allow for any generalizations, though.

Annual incidence rate of MMD ranges from 3.5 per million (in Japan) to 0.8 per million (in the USA); noteworthy is a rare occurrence among Spanish-Americans (nearly half that seen among Caucasians) [13]. Based on the American data we may expect about 30 MMD cases in Poland, affecting mostly young people and necessitating effective and timely treatment [1]. Therefore, vigilance of first-line medical professionals confronted with a child or adolescent with recurrent strokes without any apparent cardiovascular risk factors, is paramount.

An important issue is to differentiate MMD and moya-moya syndrome (MMS). The latter consists in MMD-like cerebral vasculopathy combined with other conditions (atherosclerosis, renal disease, sickle cell anemia, leptospirosis, etc.) [14]. This has been comprehensively reviewed by Matsushima and Natori in 1997 [15, 16].

The cornerstone of MMD diagnosis is a classic cerebral angiography, visualizing the diffuse tangles of collateral circulation [16]. Pathognomonic is also the "re-build-up phenomenon" seen at the EEG, i.e. return of high-voltage waves 20-60 seconds after cessation of hyperventilation, not seen in any other pathological entity [17, 18]. SPECT and PET studies after acetazolamide challenge (administered in order to enhance diuresis, provoke a mild hypovolemia and arterial hypotension) may reveal areas of borderline cerebral perfusion at risk of ischemia prior to the development of reversible (or irreversible) clinical signs [19, 20]. These are valuable tools objectively assessing the effectiveness of the therapy. Unfortunately, limited access considerably delimits their practical role in everyday practice.

All our patients had their diagnosis confirmed by angiography. In the first two, follow-up angiography was obtained to confirm revascularization. In the remaining two, in view of excellent clinical outcome, we departed from invasive and purely confirmatory procedure.

Laboratory tests revealed elevated levels of leukocytes and thrombocytes in the peripheral blood. This may be a cue to a component of MMD or MMS pathogenesis – systemic inflammatory reaction directed against own cerebral blood vessels with secondary hypercoagulation. Such a suggestion may be also found in the literature [10].

Statistics indicate that MMD is progressive in 50–60% of the cases, while in the remaining 40–50% it is stable, calling for highly individualized therapeutic approach [21]. In our material, stable clinical course was seen in one patient, but again, small sample size precludes any generalization. Natural course of the disease during the pre-clinical phase is poorly understood, i.e. we do not know whether these patients were born with normal cerebral vasculature and MMD-related alterations develop later or else their vessels are abnormal from the very beginning and collateral circulation develops early, in fact as an anatomical variant, not ensuring adequate blood perfusion, though [5].

Limited effectiveness of conservative treatment (which is beyond the scope of this paper) encourages a more aggressive approach. There are two types of situations necessitating surgical intervention: life-threatening intracranial hematoma (intracerebral or subarachnoidal) which requires emergency evacuation or acute ischemic brain edema, which requires decompression by fronto-temporo-parietal craniectomy, or recurrent ischemic episodes, resulting in progressive neurological deficits, which may require re-vascularization procedures in a scheduled setting.

Emergency procedures (type 1) are performed according to generally accepted standards and will not be discussed here. Scheduled re-vascularization procedures (type 2) address cerebral ischemia itself and we shall discuss them here.

The basic concept is to improve cerebral perfusion, either directly by creating extracranial-intracranial arterial anastomoses or indirectly, by placing well vascularized tissue in direct contact with ischemic brain. In the latter setting, angiogenetic factors secreted by hypoperfused tissue will promote neo-angiogenesis and in-growth of new vessels into the brain, thus improving it's perfusion the metabolic status [23]. In currently employed re-vascularization techniques, blood supply is derived from scalp vessels, *galea* and *periosteum, dura mater, temporalis* muscle, greater *omentum* and combinations thereof [23–26].

First attempts at direct brain revascularization in the treatment of MMD date back to the early 1970s (Yassargil: direct STA-MCA by-pass) [24]. STA-MCA by-pass is a technically demanding procedure and in the setting of pediatric MMD this technique does not yield satisfactory long term results. Apart from technical problems caused

by the small size of child's peripheral arteries, the effect is spatially limited to shunted vessel only, which tends to lose its patency with time [25]. Some authors recommend double STA-MCA anastomoses [26] or combined approach [22].

Indirect techniques include placement of pedicled flaps of *periosteum*, *temporalis* muscle or greater *omentum* on the pial membrane covering the brain [21]. Based on our experience we may state that the multi-burr-hole Suzuki technique (encephalo-galeo-pericranio-synangiosis) is technically simple, safe and effective, encompassing by re-vascularization a very wide brain convexity area. Contrariwise, single-flap techniques, including muscle--pial, omental-pial or pericranial-pial synangioses cover a limited area of the brain only, therefore their effect is by definition spatially limited. Furthermore, transposition of pedicled omental flap is an extensive procedure, requiring collaboration of general-surgical and neurosurgical teams, exposing pediatric patient to an unnecessary risk of a large--scale procedure. Nevertheless, late results of these procedure are generally satisfying in experienced hands [27].

Multi-burr hole technique proved effective both clinically (lack of recurrent ischemic episodes) and radiologically (angiography-proven formation of cerebral neo-vessels at trephination points, enabling flow of contrast medium from extra-cranial vessels, via brain parenchyma, to large dural sinuses) (Fig. 4). Favorable long-term outcomes of EDAS were confirmed in the general population of MMD/MMS patients by Ross and Scott [26, 29, 30] and in patients affected with sickle cell anemia by Kennedy and Hankinson [23, 30].

A promising, while still experimental approach in this setting, is the use of drug-eluting stent placed in supraclinoid ICA [31].

The mere number of re-vascularization techniques used currently indicates that none of them is considered optimal, while a given technique is being selected based mainly on surgeon's preference [21]. During surgery one of our patients (K.B.) developed a mild, transient and apparently clinically insignificant hypotensive episode, resulting in postoperative frontal lobe infarct, which fortunately did not lead to any clinically significant neurological deficit. In a MMD patient devoid of any functional brain perfusion reserve, even minor reduction of blood pressure may result in potentially devastating stroke. This issue has been addressed in the literature but the authors emphasize mainly the significance of the optimal depth of anesthesia to prevent surgical stress and circulatory instability associated therewith [31, 32]. In our opinion, the key factor is adequate brain perfusion during surgery. This is supported by Fujimura et al., who recommend administration of minocycline, a neuroprotective matrix metalloproteinase 9 (MMP-9) –blocking agent to stabilize the patients' perioperative course [33]. We recommend an extremely careful monitoring of blood pressure and brain perfusion, particularly in situations potentially at risk of hypotension (induction of general anesthesia, intra-operative bleeding and hypovolemia, intra-hospital transfer, change of patient's position, etc.). To sum up, surgical treatment of MMD patients requires experienced anesthesia staff and intensive monitoring.

Rarity of MMD in the general population precludes accumulation of adequate material at a single institution, let alone by a single surgeon. Development of reliable therapeutic guidelines would require multi-center trials and the exchange of information between centers diagnosing and treating cerebro-vascular diseases in children and adolescents .

CONCLUSIONS

1. Paucity of material does not allow formulation of reliable guidelines, but some general recommendations can nevertheless be made.
2. Treatment of MMD must be individualized due to imprevisible and often indolent clinical course.
3. Surgical treatment of MMD by multi-burr hole encephalo-duro-periosteal synangiosis is relatively safe and effective.
4. Effective treatment of MMD should be implemented prior to the development of severe and irreversible brain damage.
5. MMD patients require careful monitoring and maintenance of optimal blood pressure during and after surgery in order to maintain adequate cerebral blood perfusion.
6. Elevated leukocytosis and thrombocytosis in the peripheral blood may suggest possible pathogenetic mechanism of MMD – inflammation and hypercoagulation state.

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