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COMPARISON OF CLINICAL AND MORPHOLOGICAL CHANGES IN PERIHEMATOMAL BRAIN TISSUE IN HEMORRHAGIC STROKE

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Pathomorphological research changes in brain tissue in the perihematomal zone of 28 autopsies of patients with hemorrhagic stroke was examinated. It has been established that the combination of acute and chronic lesions of the vascular wall is the cause of premature death in stage I. Coagulation necrosis is a common variant of neuronal death. Hemistocytic astrocytes is significantly predominate in stages II and III. The intensity of regeneration is less pronounced and significantly increases among patients who died after 7 days after the disease. The demarcation inflammation zone in most researches reveals itself in period of early and late subacute hematoma, with the gliomesodermal capsule that forms over time.

Key words: hemorrhagic stroke, demarcation region, "red" neurons, neurons "shadows", hemistocytes, gliomesodermal scar.

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ПАТОМОРФОЛОГІЧНИХ ЗМІН ПЕРИГЕМАТОМНОЇ ТКАНИНИ МОЗКУ ПРИ ГЕМОРАГІЧНИХ ІНСУЛЬТАХ

Досліджено патоморфологічні зміни тканини мозку у перигематомній зоні 28 автопсій пацієнтів з геморагічним інсультом. Встановлено, що поєднання гострих та хронічних уражень судинної стінки є причиною передчасного настання смерті у I стадії. Найчастішим варіантом загибелі нейронів є коагуляційний некроз. Гемістоцитарні астроцити суттєво переважають у II і III стадії. Інтенсивність регенерації менш виражена та істотно зростає у пацієнтів, які померли після 7 дня з моменту захворювання. Зона демаркаційного запалення у більшості досліджень виявляється в період ранньої та пізньої підгострої гематоми, з якої з часом формується гліомезодермальна капсула.

Ключові слова: геморагічний інсульт, демаркаційна зона, «червоні» нейрони, «тіні» нейронів, гемістоцити, гліомезодермальна капсула.

The work is a fragment of the research project "Research of pathomorphological features in diseases of thyroid gland, cardiovascular, digestive, urinary and reproductive systems and perinatal period in order to improve their morphological diagnosis", state registration No. 0118U000100.

Non-intracerebral hemorrhage (NIH) accounts for 10-15% of all acute cerebral circulation disorders [11].

First of all, in early stages (within 24-72 hours), a neurological deficit is expressed and patients serious condition is associated with primary damage to the brain tissue by hematoma, expansion of its area, an increase in perihematoma edema and the spread of blood into the ventricular system [8, 13]. Secondary, brain damage occurs as a result of exposure to thrombin and erythrocyte breakdown (hemoglobin, heme, iron), which have a toxic effect on brain tissue and contribute to the development of inflammation [12,13]. At a later date, in the subacute stage of disease (second - third week), the deterioration of general condition of patients is associated with the formation of an encapsulated hematoma [8]. Therefore, a more detailed morphological research of the perihematomal zone will contribute to the improvement of modern diagnostic methods, as well as surgical and medical treatment of patients with hemorrhagic stroke (HS) in order to prevent mortality and reduce functional deficits in general [4].

The purpose of the study was to establish the features of pathomorphological changes in brain tissue in the perihematomal zone at different stages of the course beginning from the onset of hemorrhagic stroke.

Materials and methods. Based on materials of Lviv Regional Pathological Bureau, 28 lethal cases of patients aged 32 to 85 years old were analyzed and diagnosed with hemorrhagic stroke. These patients were treated in specialized hospitals (neurological departments of Lviv Regional Clinical Hospital and Clinical Emergency Hospital in Lviv). In all cases, medical histories were analyzed with the study of clinical data, the disease course, concomitant and background diseases.

The work was approved by the Commission on Bioethics (extract from Protocol No. 2 of February 26, 2017), all moral, ethical and professional requirements and standards were observed while examining cadaveric material in accordance with the principles of Helsinki Declaration of Human Rights, Council of Europe – Convention on Human Rights and Biomedicine and relevant laws of Ukraine.

The macroscopic examination of the brain determined the magnitude and localization of intracerebral hemorrhage (IUD), the spread of blood into the ventricular system, subarachnoid spaces, with the development of dislocation of brain structures and compression of the brain stem.

The research material was grouped and based on clinical and radiological classification of hematoma organization stages according to Bradley W. [2]. In this research, patients in the acute and chronic stages were not observed, since the lethal end from HS occurred during the acute, early and late subacute stages of hematoma formation.

For microscopic examination, in each case, pieces of brain tissue (2.0 x 2.0 cm) were taken in the adjacent area of hematoma, then the tissue was fixed in a 10% solution of neutral formalin and stench in alcohols of increasing concentration according to standard method, embedded in paraffin and saparated sections were stained with hematoxylin and eosin. The histological sections were examined under a light-optical microscope “Zeiss Primo Star” (Germany), photomicrographs were performed using the microscope Leica DM 750/4 (Germany) with digital camera Leica DFC 420 (Germany) and software Leica Application Suit Version 3.8) was also used. The qualitative characteristics frequency in groups was compared using the two-tailed Fisher's exact test with the usage of package STATISTICA for WINDOWS 6.0 (StatSoft, USA) [5]. The difference between the groups was considered significant at $p \leq 0.05$. In cases of marginally significancy ($p > 0.05$ but <0.01) were used results of more sensitive one-tailed Fisher's exact test [5].

Results of the study and their discussion. As a result, among 28 sectional cases, 20 (71.4%) were composed from men and 8 (28.6%) women. The average age of men was 53 years (from 32 to 85 years, the mid-quartile interval is 38-59.5 years), for women – 56.5 years (from 45 to 79 years, the mid-quartile interval is 49-64 years).

Hypertensive disease was diagnosed in 26 cases (92.85%), cerebral atherosclerosis was observed in 14 researches (50%), ischemic heart disease and diabetes mellitus was detected in 9 sectional cases (32.14%). In 9 researches (32.14%), congenital and acquired cerebral vascular pathology was diagnosed. In addition to the main risk factors for the development of the disease, 12 researches (42.85%) observed concomitant pathology, that included micronodular cirrhosis of liver, chronic hepatitis, gastric ulcer and other diseases. The dominant clinical picture was a cerebral coma, which was documented in 16 cases (57.14%), tetra and hemiparesis, according to clinical data, was observed in 8 patients (28.57%), aphasia was diagnosed in 5 patients (17.85%).

Important to mention that in first three days from the moment of illness begining, 14 patients (50%) died during 4 to 7 days, 7 patients (25%) during 8 days – and also 7 patients (25%).

The macroscopic examination of brain was guided by the recommendations of Pedachenko E.G. and others [4]. According to these recommendations, 15 patients (53.57%) had large hematomas (≥100 cm3), 10 cases (35.71%) were medium-sized hemorrhages (from 51 to 100 cm3), and 3 researches (up to 50 cm3) showed macroscopically small hematomas.

In 19 autopsies (67.85%), hematomas were localized supratentorially. Among these, 4 cases (21.05%) were located laterally, 5 (26.31%) medially, mixed hematomas were observed in 10 cases (52.63%). In 9 researches (32.14%), IMHs were localized subtentorially: 7 (77.77%) – in pons and medulla oblongata, 2 (22.23%) – in cerebellar hemispheres. In 15 cases (53.57%), blood ended up in the ventricular system, and in 13 researches (46.42%), blood spreaded to subarachnoid spaces of cerebral hemispheres and cerebellum. The histological changes in the perihematomal brain tissue in hemorrhagic stroke are presented in table 1.

Table 1

Morphological manifestations characteristics of perihematomic zone in hemorrhagic stroke depending on disease duration

Symptoms	Hemorrhagic stroke			p
	1st stage (n=14)	2 st stage (n=7)	3st stage (n=7)	

	abs.	%	abs.	%	abs.	%	
Red neurons	13	92.85	6	85.71	6	85.71	>0.05
Neuron shadows	3	21.42	2	28.57	3	42.85	>0.05
Incl. combination of red and shadows	2	14.28	1	14.28	2	28.57	>0.05
Neurons with chronic changes	4	28.57	6	85.71	6	85.71	p_{1-2}=0.029* p_{1-3}=0.029*
Focal loss of neurons	2	14.28	2	28.57	2	28.57	>0.05
Hemistocytic astrocytes	2	14.28	6	85.71	6	85.71	p_{1-2}=0.0032** p_{1-3}=0.0032**
Myelin bodies	4	28.57	3	42,85	3	42.85	>0.05
Neutrophilic infiltrate	9	64.28	5	71.42	2	28.57	>0.05
Lympho-macrophage infiltrate	4	28.57	5	71.42	6	85.71	p_{1-3}=0.029*
Regeneration	2	14.28	3	42.85	6	85.71	p_{1-3}=0.0032**
Gliosis	2	14.28	2	28.57	3	42.85	>0.05
Vascular sclerosis	2	14.28	3	42.85	2	28.57	>0.05
Hyalinosis vessels	10	71.42	4	57.14	4	57.14	>0.05
Vascular necrosis	2	14.28	0	0	1	14.28	>0.05
Hemorrhage in perivascular space	14	100	6	85.71	7	100	>0.05
Hemosiderin	2	14.28	3	42.85	6	85.71	p_{1-3}=0.0032**

Note: p_{1-2} – the difference between indicators of phase 1 and phase 2, p1-3 – the difference between indicators of phase 1 and phase 3; * one-tailed exact Fisher test; ** two-tailed exact Fisher test.

At the edge of demarcation filling appeared on border between hematoma and perihematoma tissue of brain (fig. 1), that began to form in stage I and was formed by inflammatory cells (in stage I to a greater extent by neutrophils, and in stages II and III with the attachment of lymphocytes and macrophages), activated microglia, hemolyzed by erythrocytes, hemosiderophages, and in some single cases (in stage I) again with capillaries. The largest number of researches on demarcation inflammation was observed in stage II and amounted to 5 cases (50%), and in stage III – 4 cases (40%). In stage I, these changes were manifested only in 1 research (10%).

Patients who died in first 3 days after the disease, besides the significant edema of neuropil, infiltration of perihematomal areas of brain with inflammatory cells occurred, alterative to change in developed neurons and was activated by astroglia.

In 13 cases (92.85%), neurons with coagulative necrosis (“red” neurons) were observed (Fig. 2). Significantly less frequently occurred the neurons “shadows”– in 3 cases (21.42%) (p = 0.0003, two-tailed exact Fisher test), which were observed predominantly simultaneously with “red” neurons (2 cases – 14.28%).

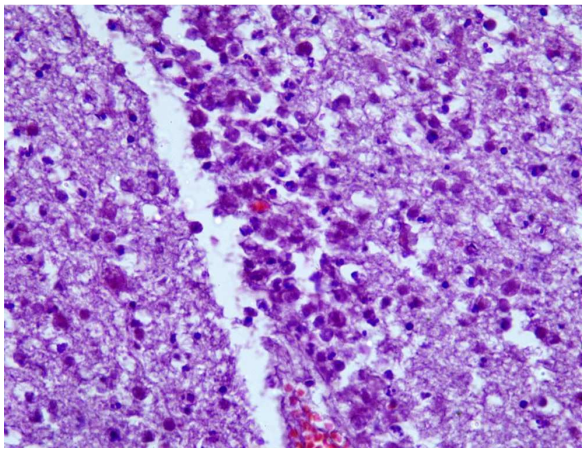

Fig. 1. Zone of demarcation inflammation formed by neutrophils, activated microglia, and single repeated capillaries. Magnification 10×40.

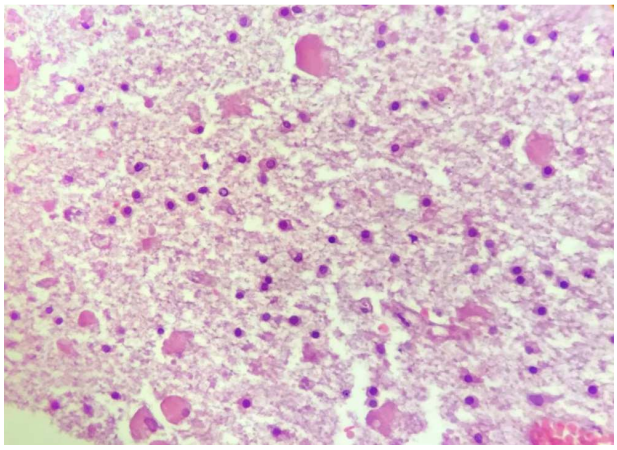

Fig. 2. “Red” neurons, single hemistocytic astrocytes and activated microglia in perihematomal zone. Magnification 10×40.

Therewith the acute changes in neurons, 4 researches (28.57%) showed neurons with minor chronic changes, and 2 researches (14.28%) observed areas of neuronal prolapse and small glial scars.

In 2 researches (14.28%), this group showed single hemistocytes. Myelin globes were observed in 4 researches (28.57%).

In the acute stage, migration of neutrophils and their infiltration of the perihematomal zone occurred, were present in more than half of the cases (9 researches – 64.28%) (fig. 3).

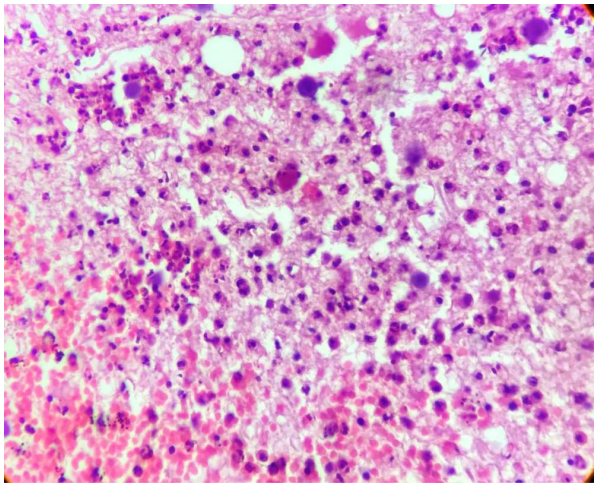

Fig. 3. Significant neutrophil infiltration is in the perihematomal zone and fresh erythrocytes and solitary myelin balls in the neuropil. Magnification 10×40.

On the other hand, in a smaller number of researches at this stage, single lymphocytes and macrophages were observed (4 researches – 28.58%, p = 0.06, one-sided Fisher's test), since such infiltration develops at later stages of the disease progress.

Early formation that was made with proliferating capillaries was found in 2 researches (14.28%).

The vascular wall sclerosis was observed in 2 cases (16.6%), vascular necrosis was present in 2 cases (16.6%), and however, arteriole hyalinosis appeared as a very common phenomenon – in 10 researches (83.33%).

In all studied histological preparations of the perihematomal brain tissue around the altered arterioles, multiple hemorrhages were observed, and in 2 cases (16.6%) macrophages with a yellow-brown pigment (hemosiderin) were detected.

Among patients who died while 4 to 7 days, the perihematomal brain tissue contained areas with revealed necrosis of all structural elements of brain and blood imbibition; at the same time, there were areas of incomplete necrosis of perifocal zone. The perihematoma neuropil became more swollen and resembled dribnoacuole spongiosis.

As in the acute stage, the predominant variant of neuronal necrosis was coagulation, which manifested itself in 6 cases (85.71%), neurons with characteristics of colliquation necrosis were observed in 2 cases (28.57%), including in 1 case (14.2 %) in combination with coagulation necrosis.

The neurons with chronic changes were present in 6 cases (85.71%), that was significantly more frequent than in acute stage (p = 0.05). In addition to it, in 2 cases (28.57%), an area with a loss of neurons was observed in examinated brain tissue.

Subsequently, in most researches, neutrophils were shown in critical zone (5 cases – 71.42%). At the same time, a significant increase in frequency of lymphocytic-macrophage infiltration was evidenced (5 cases – 71.42%, p = 0.05).

In a significant part of cases, 6 (85.71%) had hemistocytic astrocytes, that was significantly more frequent than in the previous stage (p = 0.00032). The amyloid bodies and a thin-walled non-tight capillary liaison were observed in 3 cases (42.85%).

Other six researches (85.41%) showed perivascular annular or massive confluent hemorrhages. In 3 cases (42.85%) hemosiderophages were microscopically present.

Among patients who died after 7 days because of the hemorrhage onset, the brain neuropil and due to severe edema and formation of large cavities that was filled with fluid.

Those patients who died at earlier stages, in a significant part of cases (6 - 85.41%), their “red” neurons were observed in perihematomal area, less often - neurons “shadows” (3 cases – 42.85%) that were mainly and simultaneously obsereved with coagulation necrosis neurons (2 cases – 28.57%).

Later, more than 3/4 of researches (6 autopsies – 85.71%) showed neurons with revealed chronic changes. The areas of loss of these cells were founded in 2 cases (28.57%).

In a significant part of microscopic researches, hemistocytes were revealed (6 autopsies – 85.71%), that were intensively located in the perihematomal tissue of brain. The myelin balls were observed in 3 cases (42.85%).

The frequency of neutrophil infiltration of perihematomal zone was decreased (2 cases – 28.57%). In most researches, infiltration formed with lymphocytes and macrophages prevailed (6 researches – 85.71%), that was much more frequent in stage I (p = 0.0029).

In comparison to patients who died in the acute stages, in this study group, angiogenesis was represented by a more reveal liaison of proliferating capillaries that appeared in 6 cases (85.71%).

The walls sclerosis of small vessels in perihematomal area was observed in 2 cases (28.57%), hyalinosis of arterioles – in 4 (57.14%), vascular necrosis – in 1 case (14.28%). In 6 cases (85.71%) hemosiderophages were manifested, that was significantly higher than in stage I (p = 0.0032) HS.

So, the early death of patients with hemorrhagic stroke was influenced by the size, hematoma localization, as well as the spread of blood into ventricular system and subarachnoid spaces, and caused to development of dislocation syndrome [2]. Such changes were clinically revealed in onset of deep cerebral

coma, impaired vital functions and / or significant motor deficits. Also, the presence of background and concomitant diseases in patients complicated clinical picture of underlying disease, burdened its course and accelerated the lethal completion [8].

During the research about brain changes that was caused by hemorrhage according to our data, and according to the data of other authors [6, 9] neurons experienced coagulation and colliquation necrosis. However, in all three stages, neurons often died by coagulation, and such changes could be associated with both the pressure of hematoma and the influence of decay products of erythrocytes on the adjacent brain tissue. Both variants of neuronal death in one case appeared in single research, but were present in all intervals. In response to acute brain injury, reactive inflammation developed with infiltration of the perihematomal zone by neutrophils. [11]. These cells were observed more intensively in first 3 days from the disease onset. With the progression of hemorrhagic stroke in stage II and III, neutrophils were isolated and in fewer researches the infiltrates were perihematoma. The lymphocytes and macrophages were stimulated by thrombin, hemoglobin, Gemini iron and neutrophils, were eliminated the breakdown products of erythrocytes and destroyed brain tissue, in some cases were founded in the most acute stage and grew at early and late subacute stages of hemorrhagic stroke [12]. Single hemistocytes appeared from the first days of the disease, and with the duration of this disease their number increased and in a significant part of cases these cells were presented in stage III of this disease [10]. The low-graded formation appeared in some cases in first stage. In the second stage, a more active angiogenesis was observed, that at the third stage occurred in a significant proportion of cases and was represented by a dense capillary liaison filled with fresh erythrocytes. At all stages, myelin balls, neurons with chronic damage, and glial scars were found in perihematomal zone. These morphological manifestations can be interpreted as changes that were associated with long-term damage to the vessels of macrocirculatory and microcirculatory layer, that developped into hemorrhagic stroke [1].

Conclusion

1. The combination of acute and chronic lesions of the vascular wall in form of revealed hyalinosis and necrosis became the cause of vascular decompensation and premature death in phase I.

2. In all three stages of hemorrhagic stroke, neurons in a greater number of cases died because of coagulation necrosis.

3. As a result of the mechanical effect of hematoma and decay progress of erythrocytes, the intensity of regeneration was less pronounced, and more often manifested in patients that died after the seventh day because of the disease.

4. The zone of demarcation inflammation was more often founded after the third day of hemorrhage development, during period of early and late subacute hematoma. This zone was observed not along the entire perimeter of the hematoma, but in separate areas, and probably became the basis for the further formation of the gliomesodermal capsule.

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RESULTS OF FOOT PLANOVALGUS DEFORMITY SURGICAL TREATMENT IN CHILDREN WITH CEREBRAL PARALYSIS

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Issues of treatment feet planovalgus deformity in children with cerebral palsy remain unresolved due to various structural, functional and biomechanical changes in the foot, as well as the complex pathogenesis of the disease formation. The purpose of the study was to present the results of retrospective analysis of surgical treatment of feet planovalgus deformity in children with cerebral palsy. Retrospective analysis was performed on case histories of 39 feet planovalgus deformity patients, with spastic type of cerebral palsy, who underwent surgical treatment in the period from 2002 to 2019. In Group 1, combined soft tissue interventions were used to correct feet planovalgus deformity, and in Group 2, in addition to the surgeries, interventions were performed aimed at correcting alignment in the joints of the hindfoot and midfoot. In Group 3, all children underwent combined surgery. Surgical treatment of feet planovalgus deformity in children with cerebral palsy aged 7-11 years, using techniques that are singularly aimed to correct contractures and tendon-muscle balance, is accompanied by a significant recurrence rate (66.7%). An algorithm for differentiated choice of methods for feet planovalgus deformity surgical treatment in children with cerebral palsy has been developed.

Key words: cerebral palsy, planovalgus deformity of feet, algorithm.

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ХАРАКТЕРИСТИКА РЕЗУЛЬТАТІВ ХІРУРГІЧНОГО ЛІКУВАННЯ ПЛОСКОВАЛЬГУСНОЇ ДЕФОРМАЦІЇ СТОП У ДІТЕЙ ХВОРИХ НА ДЦП

Проблеми лікування плосковальгусної деформації стоп у дітей з дитячим церебральним паралічем залишаються не вирішеними через різноманітні структурно-функціональні і біомеханічні зміни в стопі, а також складного патогенезу формування. Мета роботи: представити результати ретроспективного аналізу результатів хірургічного лікування плосковальгусної деформації стоп у дітей, хворих на дитячий церебральний параліч. Матеріал: Ретроспективний аналіз історій хвороб 39 пацієнтів із плосковальгусною деформацією стоп, зі спастичним типом дитячого церебрального параліча, яким виконано хірургічне лікування у період із 2002 по 2019 рр. В Групі 1 для корекції плосковальгусної деформації стоп використані комбіновані втручання на м'яких тканинах, а в Групі 2 разом із хірургічними втручаннями, виконувалися втручання, спрямовані на корекцію взаємовідносин у суглобах заднього та середнього відділів стопи. В Групі 3 усім дітям проведено комбіноване хірургічне втручання. Хірургічне лікування плосковальгусної деформації стоп, у дітей, хворих на дитячий церебральний параліч, у віці 7-11 років, із використанням методик, що ізольовано спрямовані на корекцію контрактур та сухожилково-м'язового балансу, супроводжується значним відсотком рецидивів (66,7 %). Розроблено алгоритм диференційованого вибору методики хірургічного лікування плосковальгусної деформації стоп у дітей, хворих на дитячий церебральний параліч.

Ключові слова: дитячий церебральний параліч, плосковальгусна деформація стоп, алгоритм.

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One of the significant deviations in the musculoskeletal system of children with cerebral palsy is development of feet planovalgus deformity (FPVD), which accounts for 16 to 35% of all orthopedic pathology in this group of patients. The feature of foot development in children with cerebral palsy is that their feet have a normal shape from birth, and only later, during the child's growth against the background of soft tissue imbalance, there occurs deformation of the foot shape and function, especially during active growth. [4, 5, 8, 12]. Additionally, over time, plantar callosity is formed on the plantar-medial surface of the foot, pain syndrome and difficulties in choosing shoes occur.

Formation of FPVD in a child with cerebral palsy differs from the idiopathic form of FPVD in children without neurological pathology, therefore, the developed standard treatments can not be used in children with cerebral palsy [1, 6, 10].

In the treatment of FPVD in children with cerebral palsy, the following techniques are used: physical rehabilitation (exercise therapy and other techniques), orthotics, botulinum toxin treatment, soft tissue foot structures surgery, bones and joints, and their combination surgery [2, 11].