Journal of Cytology & Histology
ISSN: 2157-7099

Tomaszewski et al., J Cytol Histol 2017, 8:4
DOI: 10.4172/2157-7099.1000472

Research Article OMICS International

Histological Evaluation in Autologous Growth Plate Chondrocyte Grafting in Rabbits

Ryszard Tomaszewski, Artur Gap and Lukasz Wiktor*

Department of Paediatric Surgery, Trauma and Orthopaedic Ward, Upper Silesian Children's Health Centre, Katowice, Poland

Abstract

Objective: The aim of our study was to evaluate the usefulness of the histological assessment of the growth plate after transplantation of autologous cultured chondrocytes in New Zealand white rabbits in order to treat the injured growth plate.

Results: Based on the results we have found satisfying graft morphology, satisfactory basal integration of the graft, lack of inflammatory response, fairly good growth plate architecture.

Conclusions: Autologous chondrocytes transplantation is a good method of treating growth plate damage. The histological assessment of the growth plate cartilage grafts needs the multiple parameters.

Keywords: Growth plate; Chondrocyte; Trauma; Histology

Introduction

The purpose of this paper is to report the usefulness of the histological assessment of the growth plate after transplantation of autologous cultured chondrocytes in order to treat the injured growth plate. There are many classification systems which assess the histology of the cartilage. Most of them have been created for the evaluation of the articular cartilage in osteoarthritis [1]. Due to the constantly developing techniques for the treatment of articular cartilage defects, some systems are also used to assess the quality of the transplanted cartilage. The histological assessment of the cartilage is an important part of staging of osteoarthritis and the evaluation of therapeutic effects [1]. Histological-Histochemical Grading System was created in 1971 by Mankin but MODS were the first scale rating evaluating the quality of the cartilage [1]. ICRS I in 2003 proposes the cartilage classification based on 6 parameters but the last ICRS II classification contains the 14 parameters concerning the cartilage [1-5]. Our literature review did not reveal a system evaluating the growth plate. The growth plate, as the hyaline cartilage covering the articular surfaces, has a layered structure [3,6]. In both cases, the assessment is based on the arrangement of chondrocytes and their morphology. The growth plate is situated at the ends of the long bones and is responsible for the lengthening of the long bones [6,7]. The growth plates are present during the bones growth and they close after the end of the growth. The longitudinal growth depends on proliferation, hypertrophy and ossification at the growth plate cells [3]. There are resting zone, proliferating, hypertrophic and primary bone formation at the grown growth plate [3]. Chondrocytes are organised in columns of cells, chondrocytes undergo differentiation, then hypertrophy and in the end apoptosis [6]. Calcified cartilage with vascular ingrowth leads to bone formation made by osteoblasts and osteoclasts. The damage of the growth plate could lead to angular bones deformity with their possible shortening [3,6-9]. Autologous chondrocyte grafting is one potential repair of this problem [3,10]. The aim of our work was to evaluate the histological assessment of the growth plate after autologous chondrocytes grafting in New Zealand white rabbits.

Material and Methods

The histological evaluation included 24 samples obtained from the proximal tibial growth plate from 14 New Zealand white rabbits. The 1-14 samples (R) taken from the right lower limb constituted grafts after autologous chondrocyte transplant. The 1-14 samples (L) taken from the left lower limb constituted growth plate preparations without autologous chondrocyte transplantation. The scheme of the experiment is shown in Table 1 (detailed description was included below). The study group consisted of 14 New Zealand white rabbits aged 5 weeks numerated from 1 to 14. One day the surgery was performed in the right lower limb (22.08), while on another day it was done in the left lower limb (23.08). Each treatment was held in intravenous anaesthesia under the control of breathing and heart rate. After a typical preparation of the surgical field, we made a 1.5 cm cut on the medial side of the proximal tibia, then we incised the soft tissue and reached the growth plate. We collected the ½ medial width of the cartilage using a trepanning needle. The biopsy specimen contained the material for chondrocyte proliferation. The same procedure was carried out in the left leg. A site after the collected material was filled with a drain in rabbits 1-14 (R) and in rabbits 1-7 (L). A defect was left without filling in rabbits 8-11 (L). Rabbits 12-14 (L) constituted a control group (no material was taken). The mean duration of surgery was 25 minutes (12-37 minutes). Each time, we microscopically confirmed a radical removal of the growth plate. The material collected from the growth plate was inserted in a F12 Ham's tube with the addition of 50 g/ml of Gentamicin and 2 g/ml of Amphotericin B. The secured samples were sent to the Cell Laboratory. The chondrocyte proliferation process lasted 22 days (22.08-12.09). We confirmed separately the presence of chondrocytes for each of the 14 right samples (haematoxylin and eosin staining and by detection of S-100 molecules; (Figure 1). Proliferated autologous chondrocytes were suspended in fibrin glue and in this form they were ready for transplant. Each pre-implantation graft was controlled by surgeons in order to maintain accuracy of the numbering of the graft

***Corresponding author:** Wiktor L, Department of Paediatric Surgery, Trauma and Orthopaedic Ward, Upper Silesian Children's Health Centre, Katowice, Poland, Tel: +48 606357016; E-mail: wiktor.ort@gmail.com

Received August 10, 2017; **Accepted** September 10, 2017; **Published** September 18, 2017

Citation: Tomaszewski R, Gap A, Wiktor L (2017) Histological Evaluation in Autologous Growth Plate Chondrocyte Grafting in Rabbits. J Cytol Histol 8: 472. doi: 10.4172/2157-7099.1000472

”	Excision of growth plate	Drainage tube in physis	Chondrocyte culture	Transplantation of autologous chondrocytes in physis
1 “L”	+	+	-	-
2 “L”	+	+	-	-
3 “L”	+	+	-	-
4 “L”	+	+	-	-
5 “L”	+	+	-	-
6 “L”	+	+	-	-
7 “L”	+	+	-	-
8 “L”	+	-	-	-
9 “L”	+	-	-	-
10 “L”	+	-	-	-
11 “L”	+	-	-	-
12 “L”	-	-	-	-
13 “L”	-	-	-	-
14 “L”	-	-	-	-
	Excision of growth plate	**Drainage tube in physis**	**Chondrocyte culture**	**Transplantation of autologous chondrocytes in physis**
1 “R”	+	+	+	+
2 “R”	+	+	+	+
3 “R”	+	+	+	+
4 “R”	+	+	+	+
5 “R”	+	+	+	+
6 “R”	+	+	+	+
7 “R”	+	+	+	+
8 “R”	+	+	+	+
9 “R”	+	+	+	+
10 “R”	+	+	+	+
11 “R”	+	+	+	+
12 “R”	+	+	+	+
13 “R”	+	+	+	+
14 “R”	+	+	+	+

/+/ indicates rabbits subjected to the procedure.

/-/ indicates rabbits not subjected to the procedure.

Table 1: Scheme of the experiment.

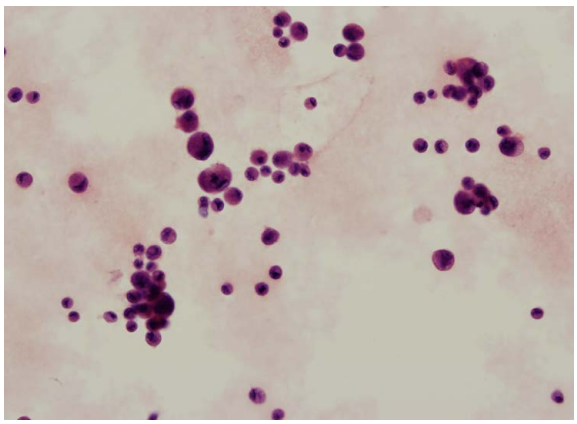

Figure 1: Microscopic view of a tibia after grafting of an autologous cultured growth (chondrocyte H&E staining).

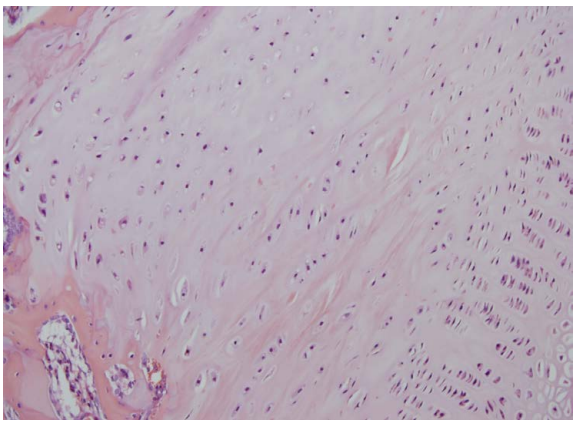

Figure 2: Histological examination of cultured -chondrocytes (H&E staining).

and the animal. After the same preparation, we performed a surgery involving the removal of a drain from the operated area of the right tibia and implanted proliferated autologous cells of the growth plate. The graft was shaped in order to fit it to the defect of the growth plate, and then, without fixation of the graft gentle periosteum suture was made using PDS 4-0 (Ethicon). Each graft was controlled for stability of fixation before the skin was sutured. The next day, we performed the procedure in all rabbits on the left side. We removed drains in rabbits 1-7 (L). After the surgery, the rabbits were allowed to walk freely in their cages. After 60 days of observation, the rabbits were killed, and the collected material was used to prepare histological samples that had been assessed. The samples were stained with haematoxylin and eosin and Safranin O after decalcification of the material using the TBD2 preparation and making 5 μm thick cuts (Figure 2).

The formulations were assessed independently 3 times by two

1.	Graft morphology compared to adjacent growth plate (viewed under polarised light).	0%: Fibrous tissue; 100%: Normal growth plate
2.	Matrix staining (metachromasia).	0%: No staining; 100%: Full metachromasia
3.	Cell morphology	0%: Elongated and spindle shaped cells; 100%: Typical cells.
4.	Chondrocyte clustering (4 or more grouped cells).	0%: presence of many clusters; 100%: Absence of chondrocyte clustering
5.	Growth plate architecture (laminar structure).	0%: Lack of laminar structure; 100%: Laminar structure
6.	Basal integration	0%: No integration; 100%: Complete integration
7.	Subchondral bone abnormalities	0%: Fibro vascular infiltration; 100%: Normal primary ossified bone lamella
8.	Inflammation	0%: Present; 100%: Absent
9.	Endochondral calcification abnormalities	0%: Absence of mineralization; 100%: Normal mineralization
10.	Vascularisation.	0%: Presence of vessels; 100%: Absence of vessels
11.	Reserve zone assessment	0%: Absent or chaotically located chondrocyte; 100%: Resting cartilage cells
12.	Proliferative zone assessment.	0%: Absent or chaotically located chondrocyte; 100%: chondrocytes well divided into longitudinal columns
13.	Hypertrophic and degeneration zone assessment.	0%: Absent or chaotically located chondrocyte; 100%: Chondrocytes with no intercellular junctions
14.	Overall assessment	0%: Bad (fibrous tissue) 100%: Good (epiphyseal growth plate)

Table 2: Scheme of the histological grafts evaluation.

pathologists (RT; AG); scheme of the histological grafts evaluation we enclosed in Table 2 using a microscope (Olympus type BX51, Olympus America Inc., Melville, New York). Graft morphology compared to adjacent growth plate (viewed under polarised light). Fibrous tissue indicating poor quality repair tissue (0%). Graft with normal/similar structure to adjacent growth plate (100%). Matrix staining (metachromasia) assessed using safranin O indicates the proteoglycan content in graft. Absence of any staining or metachromasia (0%) diffuse strong staining (100%). Cell morphology - elongated and spindle shaped cells associated with fibrocartilage/fibrous tissue (0%). Presence of typical cells for subsequent layers of normal growth plate (100%). In Chondrocyte clustering (4 or more grouped cells) we accepted the presence of many clusters throughout the repair growth plate, excluding the proliferative zone (0%). Absence of chondrocyte clustering (100%), growth plate architecture (laminar structure), lack of laminar structure (0%), Sandwich-like, multilayer structure divided into four well defined zones (100%), basal integration. No basal integration, irregular physeal shape with the lacunae, presence of fibrous tissue (0%) full basal integration through connection between the graft and the primary ossified bone (100%). Subchondral bone abnormalities. Fibrovascular infiltration the marrow space (0%), normal primary ossified bone lamella with small blood vessels and single osteoprogenitor cells compared to adjacent growth plate (100%). Inflammation: presence of lymphocytes or plasma cells, in graft tissue (0%). Absence of inflammation (100%): Endochondral calcification abnormalities. Irregular physeal shape with the lacunae, absence of mineralization (0%): Normal mineralization process of primary ossified bone, endochondral calcification (100%). Vascularisation, presence of vessels throughout the growth plate excluding the vessels in the region previously occupied by chondrocytes which underwent apoptosis (0%). Absence of vessels (100%), reserve zone assessment, absent or chaotically located chondrocyte with irregular shape (0%). Resting cartilage cells lying within the reserve zone are formed by small, uniform, compactly located chondrocytes that occur singly or in pairs and are rich in lipid and cytoplasmic vacuoles (100%). Proliferative zone assessment- Absent or chaotically located chondrocyte with irregular shape- Lack of column arrangement (0%). Flat chondrocytes well divided into longitudinal columns with mitotic activity found only in the base of the columns (100%). Hypertrophic and degeneration zone assessment- Absent or chaotically located chondrocyte with irregular shape. Lack of cytoplasmic volume increase (0%): lack of cellular division. Chondrocytes of hypertrophic zone, larger compared to the other zones. Chondrocytes lose intercellular junctions, located in special vesicles formed by graft (100%) Overall assessment - This parameter requires the observer to generate an overall assessment of the quality of the repaired tissue. Graft evaluation compared to the normal growth plate.

Statistical Analysis

Statistical analysis was done using STATISTICA 12.5 system (Table 3). For statistical analysis we assumed graduation of the results: poor result - VAS 0-20; average result - VAS 20-40; fairly good result - VAS 40-60; good result - VAS 60-80; very good result - VAS 80-100. For all analysis we assumed significance level equal to 0,05.

Results

Based on the results we found: very good graft morphology (median equal 86,50; (Figure 3), very good basal integration (median equal 86,50; (Figure 4), lack of inflammatory response (median equal 90,00), fairly good growth plate architecture (median equal 65,00).

Based on Spearman's rank correlation coefficient and Mann - Whitney U test we showed that our system is reliable and gives similar results. The statistical significances and p values of our results are presented in Table 3.

Histological findings

Right tibia (samples 1-14 "R"): In samples 1-6, 11, 13 we found a

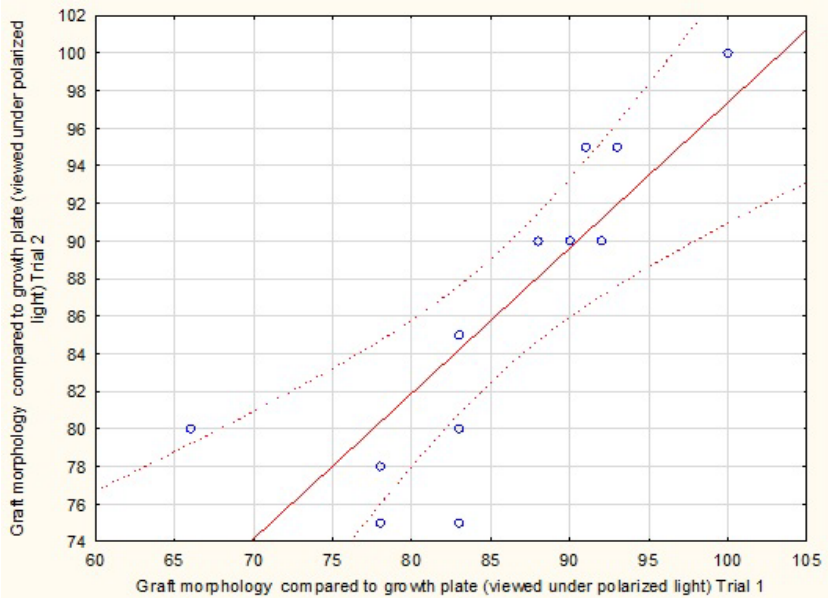

Figure 3: Very good graft morphology (median equal 86,50).

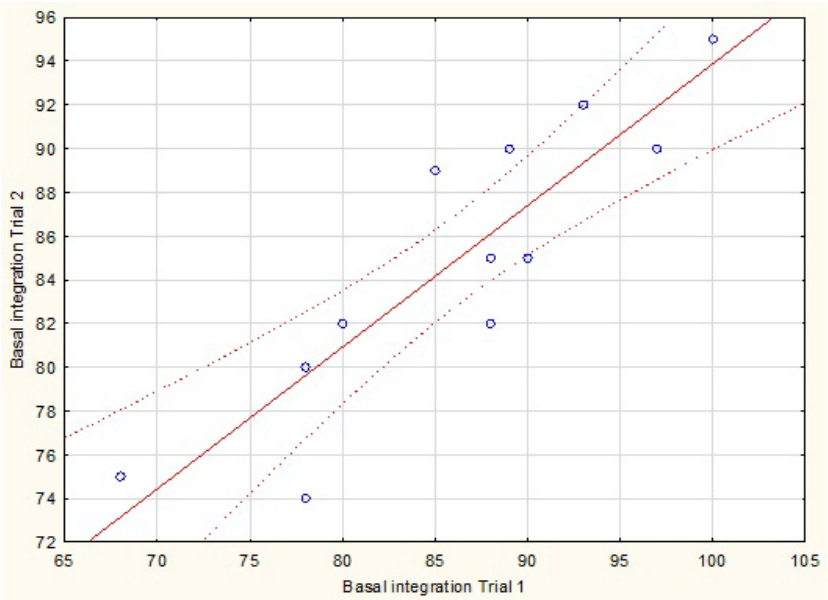

Figure 4: Lack of inflammatory response (median equal 90,00), fairly good growth plate architecture (median equal 65,00).

growth plate with regular shape and laminar structure. We observed an arrangement of columns in proliferative zone and hypertrophic chondrocytes below. In degenerative zone chondrocytes in various stages of apoptosis and single blood vessels were present.

On each level of graft the proper ratio of the cellular elements to extracellular matrix was maintained. Samples 7-9 presented an irregular physeal shape with the lacunae but with normal morphological changes in the process of cartilage differentiation and no bone bridge formation. Sample 14 presented bone bridge formation at the operated medial side of approximately 15% of the growth plate width, however, the remaining grafted cartilage on another 10% of the growth plate width showed an irregular growth plate with the lacunae. An inflammatory reaction on graft was not observed in none of the samples.

Left tibia (samples 1-14 "L"): In samples 1-11 we observed fibrous and bone bridge formation over growth plate resection between the epiphysis and the metaphysis. There was no tendency for the remaining physeal cartilage to grow laterally into the defect.

However, in samples 12-14 (with no surgery) no changes in growth plate were found (growth plate with typical histological structure). The difference between the mean values of the number of cells in both the right tibias and the left ones was 475.17. In the right tibia there were 66% more cells in 1 mm 2 of the plate than there were in the left tibia.

	Mean	Standard deviation	Reference point	t	df	p
Graft morphology compared to growth plate (viewed under polarized light)	85,75	8,51	80,00	3,31	23	0,003
Matrix staining (metachromasia)	74,75	12,95	60,00	5,58	23	0,00001
Cell morphology	66,08	16,78	60,00	1,78	23	0,089
Chondrocyte clustering (4 or more grouped cells)	79,38	9,08	60,00	10,46	23	0,000001
Growth plate architecture (laminar structure)	65,54	14,04	60,00	2,43	23	0,066
Basal integration	85,54	7,73	80,00	3,51	23	0,002
Subchondral bone abnormalities	75,83	13,87	60,00	5,59	23	0,00001
Inflammation	89,04	4,97	80,00	8,91	23	0,000001
Endochondral calcification abnormalities.	85,88	9,28	80,00	3,10	23	0,005
Vascularization	93,88	6,15	80,00	11,06	23	0,000001
Reserev zone assessment	88,67	7,56	80,00	5,61	23	0,00001
Proliferative zone assessment	82,13	6,64	60,00	16,34	23	0,0000001
Hypertropic zone/degeneration zone assessment	76,08	15,32	60,00	5,14	23	0,00003
Overall assessment	79,96	4,75	60,00	20,58	23	0,000001
Mean	80,61	3,10	60,00	32,59	23	0,000001

t: Student's t-test; df: Degrees of Freedom; p: Probability Value.

Table 3: Statistical analysis.

Discussion

Damage to the growth plate can lead to the bone bridge formation between epiphysis and metaphysis, with the consequent disorders of the linear growth and angular deformities. There are many causes of growth plate damage, but injuries are the most common [8,11-14]. When partial physeal arrest is located peripherally, the remainder of the physis usually continues to grow, producing an angular deformity. Partial physeal arrest is particularly apt to produce progressive angular deformation when a bridge of sclerotic bone forms eccentrically between the epiphyseal ossification center and the metaphyseal bone, replacing a segment of the physis and the zone of Ranvier. There is no tendency for the remaining physeal cartilage to grow laterally into the defect [6,8,15,16]. A variety of treatment techniques were developed for the growth plate damage. The Langenskiöld technique involves resection of the bone bridge and filling the defect with the artificial material to prevent the emergence of a new bridge joining the epiphysis and the metaphysis [6,13,17,18]. Interpolating material may be: biological (fat, autologous rib, cartilage of the hip) or artificial (silicone, rubber, methyl methacrylate). Distraction epiphysiolysis is a surgical procedure used to break the bone bridge of bone [14]. Stem cells were also used as a material filling defects in the growth plate [7,11,19]. Literature contains works describing auto or allogeneic transplants of chondrocytes [15,16,19,20]. Our work used autologous chondrocytes which prevent an immune response of the recipient, but require a relatively long process of multiplying of a small number of cells taken from the healthy growth plate.

The constantly developing treatment techniques, mainly concerning post-traumatic changes of the growth plate, including those using autologous chondrocytes, require the reliable assessment of the clinical, radiological and histological course of treatment in order to monitor and predict its effects but the histological assessment of the plate growth is one of the tools to evaluate the treatment results. There are a lot of histological classifications concerning the evaluation of therapeutic effects on articular cartilage, but we did not find the growth plate histological evaluation. The first system developed for the microscopic evaluation of the articular cartilage in osteoarthritis was Histological-Histochemical Grading System (HHGS) created by Mankin in 1971. MODS (modified O'Driscoll scale) was the first scale rating the quality of the transplanted cartilage. It was used to evaluate the histology of the transplanted cartilage in rabbits. The ICRS I scale was an example of the scale proposed in 2003 by the Histological Endpoint Committee for the assessment of the cartilage grafts in humans. This scale includes 6 categories (surface, matrix, cell distribution, cell population, subchondral bone, cartilage mineralization), each of which is evaluated in the system 0-3. In 2009 Histology Working Group of the ICRS proposed the ICRS II classification which has been used in a large prospective randomised study comparing the clinical and microscopic effects of the treatment of articular cartilage defects using the micro-fracture method and ACI (characterised chondrocyte implantation) [21]. The ICRS II scale includes 14 parameters assessing the articular cartilage [2,22]. Each parameter is evaluated using a 100 mm scale VAS (visual analogue scale), where 0 indices the poor quality of the cartilage and 100 denotes the high quality of the cartilage.

But the difference of the structure between the articular and growth plate cartilage did not permit the histological evaluation using the classifications created for hyaline cartilage. The articular cartilage has four layers: I – superficial containing small fibroblast-like cells and numerous collagen fibres parallel to the surface of the joint; II – intermediate, where the cells are spherical, and their arrangement is irregular; III – deep, which comprises chondrocytes arranged in groups perpendicular to the articular surface. The course of the collagen fibres in this layer is radiant; IV – a layer of the calcified cartilage which is in the direct contact with a layer of the subchondral bone [1,2,4]. The growth plate also called epiphyseal is a highly specialised layered structure responsible for an increase in the bone length, arising from mesenchymal cells, under the influence of the SOX family of gene expression [3,23]. The life cycle of chondrocytes in the growth plate area includes proliferation, maturation, hypertrophy, mineralization associated with cell apoptosis, then its ossification [3,23]. Resting zone (I) - is composed of small cells surrounded by the abundant extracellular matrix. Cells of this layer are characterised by slow divisions and the low rate of synthesis of proteoglycans and collagen II. Peripherally, this zone is surrounded by the Ranvier groove which is a form of a ring surrounding the growth plate and containing in its composition progenitor cells conditioning the transverse growth of the plate to the width. Concentrically, there is the LaCroix ring located on its edge, which is externally fused with the periosteum; it strengthens the growth plate from the side, acting as a framework for its lateral parts, preventing from the axial compressive and shear forces. Proliferating zone (II) is characterised by multiple proliferative cell divisions and dynamic metabolic processes. Chondrocytes are arranged there in

specific columns and enhance the synthesis of collagen type II and XI. In the hypertrophic zone (III) - the volume of the chondrocytes increases up to 10 times. Numerous extracellular matrix components are synthesised in this layer. The production of alkaline phosphatase is typical of this layer. The spaces of the cartilage matrix between the columns are quickly mineralised; the blood vessels growing into them depose hydroxyapatite crystals. Degeneration zone (IV) contains chondrocytes at different stages of apoptosis. Some cells, however, are inactivated by autophagocytosis. In the primary spongiosa zone (V), apoptotic chondrocytes are removed and they leave a place for the newly created blood vessels under the influence of VEGF. In this zone the matrix comprises both the products of synthesis of chondrocytes and osteoblasts in a form of the bone matrix. Deeper primary bone trabeculae are formed which over time are replaced by bone-forming secondary trabeculae creating the metaphysis [3]. The process of the growth plate cells differentiation is divided into 4 stages: mesenchymal precursor cells (MPCS); prechondrocytes; early chondroblasts; differentiated chondrocytes [23]. Our work is based on an animal model but this model allows to control the size of a lesion, the choice of a graft, the age of the specimens and in the end the timing of analysis with the possibilities of bones examination [4,5]. We have found in the literature that the rabbits' model is mostly used for the cell therapies for growth plate regeneration [9,24].

The aim of our work was not to create the new scale of histological assessment of growth plate grafting, but to evaluate the histological finding after autogenous chondrocytes plate growth graft in rabbits. The assessment contains parameters assessing the growth plate phenotype (cell morphology, matrix staining), a layered structure of the growth plate (growth plate architecture, reserve zone assessment, proliferative zone assessment, hypertrophic and degeneration zone assessment), integration (basal integration). The evaluation of vascularization and endochondral calcification abnormalities is an important element of the histological assessment having a direct impact on the radiographic and clinical effects [2,3,12].

The first element which the authors histologically assessed was [2,23] morphology. Using the microscope with polarized light the graft morphology was compared to growth plate, especially the similar structure to adjacent growth plate. The presence of the fibrous tissue between the chondrocytes indicates the poor result of grafting, which concerns [2] the growth plate graft.

The cell morphology was examined to look for the presence of typical cells for subsequent layers of normal growth plate and subchondral bone abnormalities where normal primary ossified bone lamella with small blood vessels and single osteoprogenitor cells were compared to adjacent growth plate. Inflammation was examined for presence of the lymphocytes or plasma cells in graft tissue. And we have found no inflammation in a healthy growth plate cartilage [1,3,4].

Normal mineralization process of primary ossified bone was assumed by presence of endochondral calcification abnormalities. The significance of clusters in repair tissue is not yet known, the anchors of the scale were arbitrarily chosen. The significance of the chondrocyte clusters in repatriated cartilage is unclear [2]. Similarly to other authors we accepted the presence of many clusters throughout the repair growth plate, excluding the proliferative zone [4].

Basal integration of the graft was assessed to look for the integration between the graft and the primary ossified bone, The graft was prepared using the autologous growth cartilage and the bone formation provides the information as far as transplant agreement is concerned [3,5].

There are no vessels at the healthy growth plate, but it was looked for the presence of vessels throughout the growth plate excluding the vessels in the region previously occupied by chondrocytes which underwent apoptosis [2,7,23]. Invading capillary loops from the metaphysis break through the last traverse septum of mineralised cartilage to enter the hypertrophic chondrocyte lacuna and it is indispensable items in the regulation of endochondral ossification and is necessary for normal bone formation [3]. The inhibition of vascular invasion leads to profound disturbances in the structure of the growth plate and affects the longitudinal growth [9,23].

The zones are characteristic for the growth plate cartilage [3,23]. At the reserve zone assessment it was looked for if there are no present or chaotically located chondrocytes with irregular shape but if there are any resting cartilage cells lying within the reserve zone which are formed by small, uniform, compactly located chondrocytes that occur singly or in pairs and which are rich in lipid and cytoplasmic vacuoles. At the proliferative zone assessment the authors looked for flat chondrocytes well divided into longitudinal columns with mitotic activity found only in the base of the columns. The hypertrophic and degeneration zones were controlled for absent or chaotically located chondrocytes with irregular shape and the lack of cytoplasmic volume increase.

The final assessment concerns the overall assessment. This parameter requires the observer to generate an overall assessment of the quality of the repaired tissue. Graft evaluation was compared to the normal growth plate [2,23] because the injury repair could lead to the undesirable bone repair, namely the inflammatory, the fibrogenic, the osteogenic and remodelling period with eventually bone bridge formation [9]. Safranine O staining proves [2,5] the presence of the proteoglycan content in matrix. Many authors use the Safranine O staining in quantitive assessment with the scale from 0 to 5 [5], but VAS (visual analog scale) system for evaluation provides the proper results such as the ICRS II classification [2]. We have used the VAS scale results from 0 to 100%, and as in ICRS II classification the poor result was 0% and the improvement of the findings leads to score 100%. Some variability between the observers has been noted [2], but VAS scale helps to reduce differences in the assessment of individual parameters obtained by different pathologists. We agree with many authors to assess the histological results at least by two researchers [1,4]. We recognise the limits of our work, firstly our histological assessment concerns the rabbit's tissues, secondly we try to choose the histological parameters to assess the results of autologous plate growth graft, but we believe that there is a need to create the classification for the plate growth cartilage assessment such as ICRS II classification. And finally, the pathology of growth plate cartilage could frequently create the disorders of the limb growth, which could be perfectly shown using radiographic diagnostic. It seems there is a need for the histological classification with correlation with radiographic assessment since the longitudinal bone's growth is the best parameter of the efficient graft result.

Conclusion

1. The histological assessment of the growth plate cartilage grafts needs the multiple parameters.

2. The autologous growth plate cartilage grafting presents good histological results.

References

1. Rutgersy M, van Pelty MJP, Dhertyz WJA, Creemersy LB, Sarisy DBF (2010)

Evaluation of histological scoring systems for tissue-engineered, repaired and osteoarthritic cartilage. Osteoarthritis and Cartilage 18: 12e23.

2. Varlet PM, Van Damme B, Nesic D, Knutsen G, Kandel R, et al. (2010) A New Histology Scoring System for the Assessment of the Quality of Human Cartilage Repair: ICRS II. Am J Sports Med 38: 880.
3. Ballock RT, O'Keefe RJ (2003) The Biology of the Growth Plate. J Bone Joint Surg Am 85: 715-726.
4. Varlet MP, Aigner T, Brittberg M (2003) Histological assessment of cartilage repair: a report by the Histology Endpoint Committee of the International Cartilage Repair Society (ICRS). J Bone Joint Surg Am 85-A Suppl 2: 45-57.
5. Hoemann C, Kandel R, Roberts S (2011) International Cartilage Repair Society (ICRS) Recommended Guidelines for Histological Endpoints for Cartilage Repair Studies in Animal Models and Clinical Trial. Cartilage 2: 153-172.
6. Chung R, Foster BK, Xian CJ (2011) Preclinical studies on mesenchymal stem cell-based therapy for growth plate cartilage injury repair. Stem Cells Int 570125.
7. Broughton NS, Dickens DRY, Cole WG, Menelaus MB (1989) Epiphyseolysis for partial growth plate arrest: results after four years at maturity. J Bone Joint Surg [Br] 71-B: 13-16.
8. McCarty RC, Xian CJ, Gronthos S, Zannettino AC, Foster BK (2010) Application of Autologous Bone Marrow Derived Mesenchymal Stem Cells to an Ovine Model of Growth Plate Cartilage Injury. Open Orthop J 4: 204-210.
9. Chung R, Xian CJ (2014) Mechanisms for growth plate injury repair and potential cell-based therapies for regeneration. Journal of Molecular Endocrinology 53: T45-T61.
10. Foster BK, Hansen AL, Gibson GJ (1990) Reimplantation of growth plate chondrocytes into growth plate defects in sheep. J Orthop Res 8: 555-564.
11. Ogden JA (1987) The evaluation and treatment of partial physeal arrest. J Bone Joint Surg [Am] 69: 1297-1302.
12. Martiana K, Low CK, Tan SK, Pang MW (1996) Comparison of various interpositional materials in the prevention of transphyseal bone bridge formation. Clin Orthop Relat Res 325: 218-224.
13. Osterman K (1994) Healing of large surgical defects of the epiphysial plate: an experimental study. Clin Orthop Relat Res 300: 264-268.
14. Son SM, Park IH, Oh CW (2013) A histomorphometric study of cellular layers after hemiepiphyseal stapling on the physeal plate in rabbits. J Orthop Sci 8: 152-158.
15. Bright RW (1974) Operative correction of partial epiphyseal plate closure by osseousbridge resection and silicone-rubber implant. J Bone Joint Surg [Am] 56-A: 655-664.
16. Canadell J, de Pablos J (1992) Correction of angular deformities by physeal distraction. Clin Orthop Relat Res 283: 98-105.
17. Langenskiold A (1975) An operation for partial closure of an epiphysial plate in children, and its experimental basis J Bone Joint Surg [Br] 57-B: 325-330.
18. Gotfried Y, Yaremchuk MJ, Randolph MA, Weiland AJ (1987) Histological characteristics of acute rejection in vascularized allografts of bone. J Bone Joint Surg [Am]. 69-A: 410-425.
19. Campbell CJ, Grisolia A, Zanconato G (1959) The effects produced in the cartilaginous epiphyseal plate of immature dogs by experimental surgical traumata. J Bone Joint Surg [Am] 41-A: 1221-1242.
20. Lee EH, Chen F, Chan J, Bose K (1998) Treatment of growth arrest by transfer of cultured chondrocytes into physeal defects. J Pediatr Orthop 18: 155-160.
21. Saris DB, Vanlauwe J, Victor J, Haspl M, Bohnsack M, et al. (2008) Characterized chondrocyte implantation results in better structural repair when treating symptomatic cartilage defects of the knee in a randomized controlled trial versus microfracture. Am J Sports Med 36: 235e46.
22. Peterson L, Minas T, Brittberg M (2000) Two- to 9-year outcome after autologous chondrocyte transplantation of the knee. Clin Orthop Relat Res 374: 212-234.
23. Burdan F, Szumi J, Korobowicz A, Farooquee R, Patel S, et al. (2009) Morphology and physiology of the epiphyseal growth plate. Folia Histochem Cytobiol 47: 5-16.
24. Ohlsson C, Nilsson A, Isaksson O, Lindahl A (1992) Growth hormone induces multiplication of the slowly cycling germinal cells of the rat tibial growth plate. Proc Natl Acad Sci USA 89: 9826-9830.

Citation: Tomaszewski R, Gap A, Wiktor L (2017) Histological Evaluation in Autologous Growth Plate Chondrocyte Grafting in Rabbits. J Cytol Histol 8: 472. doi: 10.4172/2157-7099.1000472