

Application of ophthalmoscopy in forensic medicine

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Abstract

Postmortem examination of ocular fundus structures has historically relied on direct ophthalmoscopy and evisceration of the eye. The biggest limitation of direct ophthalmoscopy is coronal clouding. However, with the use of endoscopy, we can bypass this obstacle. This narrative review article with observations from our Department of Forensic Medicine explains the use of postmortem ophthalmoscopy in the following cases: asphyxia, post-mortem interval evaluation, carbon monoxide poisoning, postmortem position evaluation, shaken baby syndrome, subarachnoid hemorrhage, Terson syndrome, pediatric infections, retinal hemorrhages in cerebral malaria, acquired immunodeficiency syndrome and retinal air embolism. In addition, we presented the proprietary methodology we use to examine the fundus using postmortem ophthalmoscopy. This review of literature is based on a selective literature search of the PubMed and Web of Science databases from 2009 to 2023 using the search terms “ophthalmoscopy”, “forensic medicine” and “retina”.

Introduction

Ophthalmoscopy is a very useful examination in living patients. Examining the retina can answer many clinical questions and help with making an accurate diagnosis and implementing appropriate treatment. Often, the first signs of disease appear on the retina before they become visible in more noticeable places. Can postmortem retinal examination be useful? What are the advantages and disadvantages of this method? What is the specificity of detecting the cause of death or diseases that may have led to death? These are the main questions we asked ourselves before starting this overview, and we will try to answer them in this article.

Over the past two centuries, pathologists have drawn attention to postmortem ocular findings. In the 1950s and 1960s, Kevorkian and colleagues focused their studies on postmortem ocular changes, particularly of the retina and optic disk [1].

The examination of the ocular fundus after death has traditionally relied on direct ophthalmoscopy or ocular evisceration. However, recently, new techniques have been introduced, such as postmortem ophthalmic endoscopy and monocular indirect ophthalmoscopy [2–5]. Direct ophthalmoscopy (Figure 1) has been used to estimate the time since death and identify retinal abnormalities.

Unfortunately, its utility is limited due to the post-mortem corneal clouding (Figure 2), a restricted field

of view, the inability to visualize the peripheral retina, and a lack of depth perception (stereopsis) [6].

Postmortem monocular indirect ophthalmoscopy consists of a light source attached to a headband along with a hand-held lens (Figure 3).

This permits a wide view of the fundus after death. The restrictions of this technique are the same as for direct ophthalmoscopy due to corneal clouding [7].

For medical examiners and coroners, ocular enucleation is not a routine part of the autopsy procedure unless there is suspicion of child abuse. Consequently, this leads to inherent observational bias when reporting the prevalence of specific fundal findings, such as retinal hemorrhages (RH) (Figure 4).

The introduction of postmortem endoscopic fundoscopy (Figure 5) has enabled the visualization and documentation of retinal abnormalities, addressing some of the limitations of previous methods.

The endoscopic procedure described by Amberg *et al.* [2] involved the use of a rigid hand-held endoscope with a diameter of 4 mm and a 0° Hopkins-lens, providing a wide-angle view of 120°. The endoscope was inserted into the vitreous humor through the pars plana region by creating a sclerotomy 3.5 mm behind the limbus.

For photographic documentation, a reflex camera system with a special zoom lens and a switch for the endoscope was employed. This setup allowed for capturing detailed images during the procedure.



Figure 1. Application of direct ophthalmoscopy (own graphics)

To achieve a comprehensive examination, the endoscope was maneuvered appropriately to obtain a complete overview of the retina, pars plana, pars plicata of the ciliary body, and the posterior surface of the lens (Figure 6).

This comprehensive visualization was made possible through precise movements of the endoscope within the eye's vitreous cavity [8].

Asphyxia

Asphyxia from a forensic perspective can be broadly defined as an external action leading to cerebral hypoxia within the organism. Prolonged cerebral hypoxia can result in death. Forensic medicine deals with violent asphyxiation, which is non-natural in contrast to cerebral hypoxia from natural or disease-related causes, which represents the terminal phase of all deaths. The categorization of asphyxia and the delineation of its subtypes lack consistency,

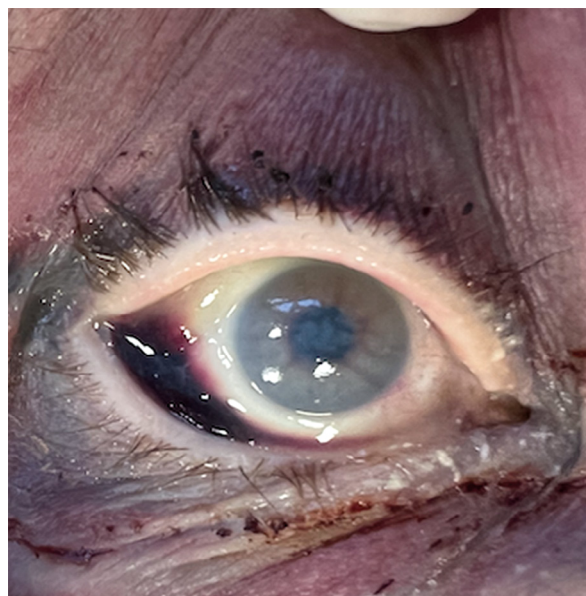


Figure 2. Postmortem corneal clouding (own graphics)

displaying considerable variation across different textbooks and from one research paper to another. Sauvageau and Boghossian suggested a forensic classification of asphyxia into four primary categories: suffocation, strangulation, mechanical asphyxia, and drowning. Suffocation is further broken down into smothering, choking, and confinement in spaces with compromised air quality. Strangulation comprises three distinct forms: ligature strangulation, hanging, and manual strangulation. Regarding mechanical asphyxia, it encompasses positional asphyxia and traumatic asphyxia [9].

Based on statistics from the Institute of Legal Medicine in Hamburg, Germany, one-third of suicides,

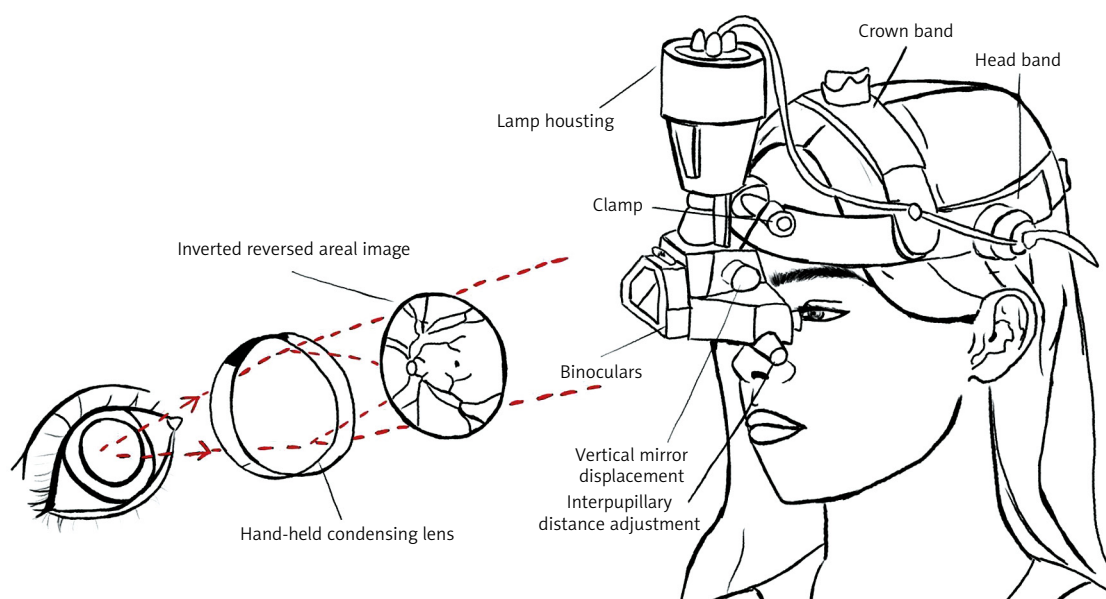


Figure 3. Application of indirect ophthalmoscopy (own graphics)



Figure 4. Dark, irregular hemorrhages beneath the retinal layers (own graphics)

one-fourth of homicides, and the majority of fatal accidents are directly related to asphyxia [10]. Azmak during a 21-year observation, found that 15.7% of autopsies were related to asphyxia [11]. Asphyxia is associated with impaired delivery of oxygen to the body's cells along with impaired removal of carbon dioxide [12–15]. The main postmortem signs associated with asphyxia, visible during an examination of the eye fundus, are RH. This is likely related to asphyxia, where there is an increase of intracranial pressure (ICP) involving mechanical obstruction of the upper airway (strangulation, hanging, smothering, drowning, or suffocation) and chest compression, which led to oxygen deprivation rather than the hypoxia of the retina itself [16–18].

There are many laboratory models that use isolated tissue preparations that demonstrate the vascular effects of hypoxia. However, many clinical animal studies have not shown the occurrence of RH caused by oxygen deprivation [19–21].

Therefore, the presence of intraocular hemorrhages cannot be used as a direct indicator of hypoxia because these are markers of an increase in central venous pressure as a result of strangulation, especially

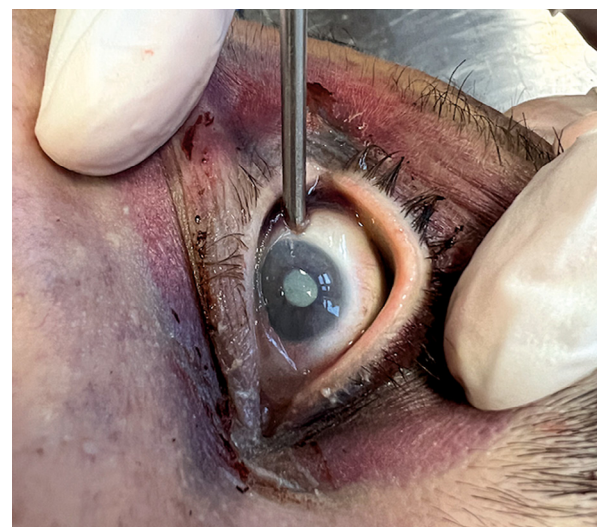


Figure 5. Insertion of the endoscope postmortem (own graphics)

those associated with ligature strangulation, hanging, and manual strangulation. However, such a finding should raise suspicions about the cause and circumstances of the oxygen deprivation.

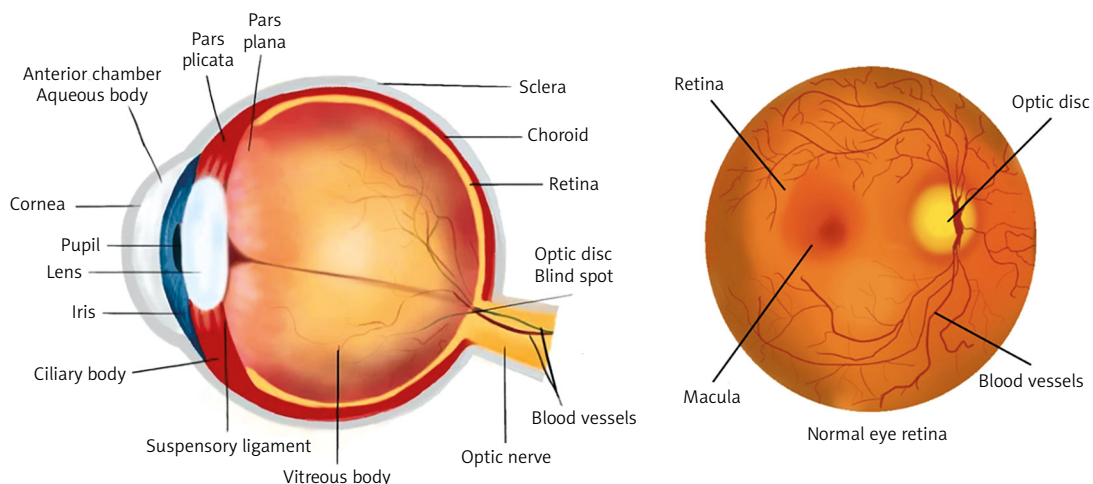


Figure 6. Anatomy of the eye, sagittal section, and fundus view (own graphics)

Post-mortem interval (PMI)

PMI refers to the period of time between the moment of physiological death and the subsequent examination of the deceased individual [20]. There are various methods to provide a PMI estimation based on physiological and environmental changes that take place after an unexplained death. In 1965, Aoki *et al.* suggested the importance of post-mortem ocular changes in determining the PMI [21]. Traditionally ocular changes and the PMI have primarily focused on time-related variations in the concentrations of certain metabolites and elements within the eye. The structural changes within the eye should be taken into consideration when measuring the PMI as there is significant evidence leading towards its aid in determining the time period that has passed since death. With the application of the ophthalmoscopy, the ocular parameters we can primarily focus on are the changes within the retina and corneal opacity.

The cornea is a transparent avascular tissue that covers the outer surface layer of the eye. In pathological conditions such as cholera, wasting diseases, iatrogenic trauma, or even graft rejections, we may observe the cornea opacity to become variably opaque.

As the time since death increases, one of the notable patterns we can generally observe is the increasing opacity in the eye. This opacity is primarily caused by the gradual drying out of the cornea, and its onset may be delayed for up to 2 hours if the eyelids are closed after death.

The correlation between corneal transparency and PMI was established in 1965. Aoki found that all corneas remained transparent for 8–12 hours after death. Opacity was found in 15% of corneas after 12–18 hours. Over 18–24 hours, 25% were opaque. For 24–36 hours after death, the percentage rises to 75% [21]. In a study by Suzutani *et al.* the corneal turbidity on cadavers with closed eyelids is observed within a time frame of 6 hours to 3 days for weak turbidity, 12 hours to 2 weeks for moderate turbidity, and over 18 hours for significant turbidity. The study also states that within 12 hours after death, corneal turbidity varied solely due to eyelids open or closed in cadavers [22]. In 1970, Wróblewski and Ellis conducted a study on postmortem ocular alterations, specifically focusing on the removal of organs for transplantation. Out of the 96 cases analyzed, they observed that within 2 hours following death, there was minimal opacity observed in the cornea. However, after 2 hours, corneal opacity was seen in 74% of the instances. The lack of cornea opacity suggested recent death, specifically within 1–2 hours. If opacities are detected, it indicates that the time of death is likely to be more than 2 hours [23]. The gray scale method can be employed to estimate the PMI by comparing the level of corneal opacity to the gray scale. An inherent drawback lies in the subjective nature of the examiner's evaluation

[24]. However, it is not necessary to extract the eye, which would alter the deceased's appearance. Conversely, alternative techniques like light transmittance and estimation of the state of DNA degradation in the corneal cell measurement necessitate specialized procedures, equipment, and a significant time commitment [25, 26]. Thus, utilization of ophthalmoscopy would enable the forensic pathologist to directly determine a precise time interval since death at the crime scene.

Kumar *et al.* observed the postmortem corneal turbidity at different PMI with variations depending on weather conditions. The corneal opacity changes occurred more often in moist conditions compared to dry conditions. The study also explores the impact of temperature, discovering that it is observed more quickly in conditions described as warm than in cold settings [27]. Therefore, the cornea is an important physical structure that shows described changes after the time of death and can be analyzed using non-invasive ophthalmic methods and provides data for estimating the PMI. However, there are many limitations and factors that should be further evaluated such as the effects of temperature, humidity, and individual variability.

Retinal changes are observed immediately after death. With the non-invasive approach of ophthalmoscopy, we can observe changes in the retinal color, folding, and vessels. These retinal variations are related to the PMI. The color of the retina starts to change after death. As the time interval increases, we can observe that the retina tends to become grayer. However, studies have been limited to the color variation of the retina as it is subjectively observed and described by the examiner.

Right after the moment of death, a process known as retinal vessel segmentation, or “trucking” (also referred to as the Kevorkian sign), occurs. During this phenomenon, the unbroken blood column within the retinal blood vessels fragments into smaller segments, resulting in their collision with one another. As the PMI increases, the retina progressively loses its color, and the outline of the optic disc becomes less distinct after a few hours [28]. Segmentation of the retinal vessels starts to occur 10–15 minutes after death. Within the first 3 hours post-mortem, segmentation can be stimulated by exerting pressure on the ocular globes, head rotations or even exerting pressure onto the thorax. With the use of ophthalmoscopy, it has been able to assist in the identification of RH, but the utilization of post-mortem fundoscopy still requires further investigation [29].

The PMI can be estimated by analyzing ocular changes, particularly corneal opacity and retinal variations. Ophthalmoscopy, a non-invasive method, allows forensic pathologists to directly assess these changes, providing valuable insights into the time elapsed since death.

Fatal carbon monoxide poisoning

Carbon monoxide (CO) is a colorless, odorless, tasteless, non-irritating gas that is present in the environment. It can even be present in situations where there is no fire or smoke but in normal environmental settings. According to estimates, 50,000 people in the United States of America are affected by CO poisoning each year, with non-fire-related smoke inhalation accounting for the majority of cases. Approximately 1000 to 1300 of these cases are fatal [30]. Compared to oxygen, CO has a strong affinity for hemoglobin that is more than 200 times greater, therefore leading to the formation of carboxyhemoglobin which causes tissue hypoxia by shifting the oxyhemoglobin dissociation curve. This interferes with oxygen delivery to the tissues and oxygen consumption in the mitochondria [31]. As a consequence, the hypoxia of the retina after CO poisoning can make the vessel system of the retina itself produce transient or continuous spasms, change the permeability of the vessel wall, then aggravate the edema of the retina, and even cause RH [32]. CO poisoning can manifest unique and identifiable signs, some of which can be visually detected in living individuals. Notably, one of the most striking indicators is the appearance of a distinctive cherry red coloration in the skin, typically when the concentrations of CO-Hb are exceeding 30%, this finding is usually observed [15]. The classical triad includes cherry red lips, along with two characteristic clinical features: cyanosis and RH. In a study by Kelley, findings show that the retinal nerve fiber layer of the individuals who had been exposed for longer than 12 hours, were hemorrhages [33]. Furthermore, a study on ocular dysfunction after CO poisoning has revealed a high incidence of ocular complications including keratopathy, damage to retinal nerve cells, optic nerve impairment, retinal vascular disease, macular disease, and damage to the occipital visual center [32]. Drawing on the insights from non-fatal CO poisoning cases, researchers can potentially apply the current findings to improve post-mortem ocular examinations. However, it is vital to acknowledge that further research is necessary to expand our understanding of the ocular implications in post-mortem cases as the findings are currently limited.

Post-mortem positioning

Endoscopic examination of the postmortem retina allows for the accurate determination of the position in which the body lies after death. In an example presented by Davis *et al.* the deceased was testified to be lying on his side with his partner in an embrace, resting on his left side. The only vessels in both eyes that were engorged, according to endoscopic retinal images acquired just before the autopsy, were the ones that would be most vulnerable to being filled by

gravitational forces if the deceased were to spend a lot of time lying on his left side after passing away. In this case, the blood in the left retina would have a tendency to rest in the vessels that are more lateral, whereas the blood in the right retina would rest in the vessels that are more medial [3]. However, there is an absence of in-depth studies on the effect of body position after death on the filling of the retina vessels.

Shaken baby syndrome (SBS)

The primary factor in childhood death globally is trauma, with inflicted head injury consistently being the most common cause of traumatic death [34–37]. SBS is generally diagnosed in infants under 1 year of age, with boys representing 65% of the victims and most perpetrators of abuse being male. SBS is characterized by a combination of findings: RH, intracranial injury (most often subdural hemorrhage (SDH), subarachnoid hemorrhage (SAH), cerebral edema) and bone fractures [35]. The fractures are found in 25% of post-mortem examinations, most commonly of ribs, long bones, occipital bone, and posterior parietal bone which stems from the specificity of abuse; in SBS the infant is held by its thoracic region and shaken, generating rapid and repetitive acceleration/deceleration movement of the cranium in respect to the torso or by pounding the baby's head against a surface [34]. The baby is often shaken in a fit of frustration by the caretaker, in a desperate attempt to quiet the child who is unable to be comforted. In 30% of cases, it ends in a fatality in a span of a few days and with the survivors suffering severe impairments – blindness, cognitive impairment, and behavioral disorders [36].

Due to the possibility that the baby may not show obvious indicators of maltreatment, diagnosis can be challenging. An ophthalmoscopic exam should be carried out in order to find retinal bleeding because the symptoms of SBS are nonspecific and range from vomiting, apathy, lethargy, irregular breathing, to seizures. Unfortunately, RH cannot be precisely dated although indirect ophthalmoscopy can be used up to 3 days after death, or ocular endoscopy can be utilized during an autopsy with the risk of retinal detachment as a post-mortem artifact. Since a greater extent of RH is correlated with more serious damage, particularly if the hemorrhages are multilayered and numerous, a swift and easy ophthalmoscopic exam may shed light on the severity of the brain injury. RHs are thought to occur as a result of the blockage of veins in the retina due to elevated pressure within the skull caused by cerebral edema and SDH [37]. RH is only present in 3% of cases of accidental head trauma, while it is present in 85% of cases of non-accidental head trauma and even higher in autopsy investigations [36]. RH is regarded as a defining characteristic of SBS, however, medical professionals should be aware that close to 40% of vaginally deliv-

ered neonates have RHs in the early days of their life but the condition observed after 6 weeks of age should not be traced back to birth [34]. Because of this, neither RH nor SDH or SAH are pathognomonic for SBS, and the only symptoms that may be used to diagnose SBS are the three symptoms listed above in combination with a comprehensive investigation of the patient's family history and current condition. Two-thirds of shaken babies will exhibit severe hemorrhagic retinopathy, which includes pre-, intra-, and subretinal hemorrhage as well as distribution throughout the retina to the ora serrata and hemorrhages that are too numerous to count. Nonetheless, some SBS patients may experience fewer hemorrhages that are restricted to the posterior pole's intraretinal distribution. The appearance of a RH depends on where it is located within the retina. A splinter- or flame-shaped hemorrhage will arise when bleeding occurs in the superficial nerve fiber layer. Dot or blot hemorrhages are the names for RH that are more deeply embedded in the retinal tissue and have a spherical or amorphous appearance. Preretinal hemorrhage, which occurs in front of the retina and is located on its surface, is distinguished by the concealment of deeper retinal vessels that pass through the outer layers of the retina. Similar to subretinal hemorrhage, it is characterized by arteries that run across the bleeding. Blood can also become caught between the vitreous and retina. Retinal detachment, choroidal, and vitreous hemorrhages frequently follow intraretinal and subhyaloid hemorrhages. RH can be used to assess the severity of brain trauma and be an indication of child abuse.

Spontaneous subarachnoid hemorrhage

Intracranial bleeding between the brain and the tissue covering it, the subarachnoid space, is known as subarachnoid hemorrhage (SAH). Spontaneous SAH is the third most common type of stroke and is frequently related to aneurysmal rupture. And it mostly affects people around 55 years of age. Its sequelae in the patients' neurology are severe and drastically impair the years of productive life and, therefore, its quality. Many population-based studies have proved that SAH incidence is around 7–9 cases per 100,000 people per year, excluding Finland and Japan, which have way higher rates. Mortality rates are high, around 50% of cases [38–40].

After postmortem examinations, it has been concluded that: 85% of underlying causes are due to the rupture of an aneurysm within the cranial cavity; the rebleeding of this is the most concerning danger [39] reporting a 20–60% in-hospital mortality rate [38]. The most common reasons why an aneurysm would rupture, causing bleeding, include hypertension, age, and aneurysm characteristics like larger size, location, and irregular shape.

In relation to ophthalmological postmortem examination, SAH presents as bleeding in either the vitreous hemorrhage (Terson syndrome) or, in a lower incidence ratio, RH. The Department of Legal Medicine at Gifu University Graduate School of Medicine conducted research where they performed endoscopic vitrectomies to access the posterior chambers of the eye. Trying to avoid the superficial vessels of the eye in order not to cause bleeding, an endoscope was introduced into the eyeball, and further images of the posterior aspect of the lens, ciliary body, vitreous body, retina, and optic disc were taken, and examined. Based on the results of the postmortem optic examination, they concluded that it is possible to have an approximation of the time that elapsed between the start of bleeding and the time of death [5]. Unfortunately, hypertension, cigarette and alcohol use, estrogen deficiencies, and family history contribute to the development of SAH. As for why aneurysms form in the brain is not known, therefore the cause of death cannot be determined [41].

Terson syndrome (TS)

TS is now described as intraocular hypertension (IOH) located in the vitreous, subhyaloid, and intraretinal compartments secondary to a subarachnoid or subdural bleeding [42]. It was first described by a French ophthalmologist, Albert Terson, in 1900. As of today, the mechanism of TS is understood as a drastic increase in the ICP that channels through the optic nerve sheath and causes venous IOH and a rupture of the retinal vessels. TS can go along with macular holes, retinal detachments, and optic neuropathy [43]. The hemorrhage is seen both unilaterally and bilaterally. Bilateral cases are usually asymmetric and are seen in 42–60% of the cases [43].

8–46% of patients who suffer from acute aneurysm SAH are seen to have TS, and it has been demonstrated to increase the mortality rate within these patients to 90% [42]. Patients who suffered from a coma are more likely to present with TS, possibly because this disease also presents a great, rapid ICP enhancement [44–46]. The most common aneurysm-induced SAH concerns the anterior communicating artery and the internal carotid artery [43]. How proximal, anatomically wise, to the orbit the aneurysm takes place has no correlation to the presence of this syndrome [45].

In the forensics world, TS is not really discussed, even though mortality rates have shown that around 50% of the cases present with intraocular hemorrhages [3]. The primary method for diagnosing the TS is through fundoscopy. A crescent-shaped hyperdensity/hyperintensity of the retina observed on CT and MRI scans has been documented to exhibit a diagnostic sensitivity of 66.7% [42].

A pars plana endoscopy provides illustrative and representative information of the posterior ocular segment during autopsies. This technique allows for

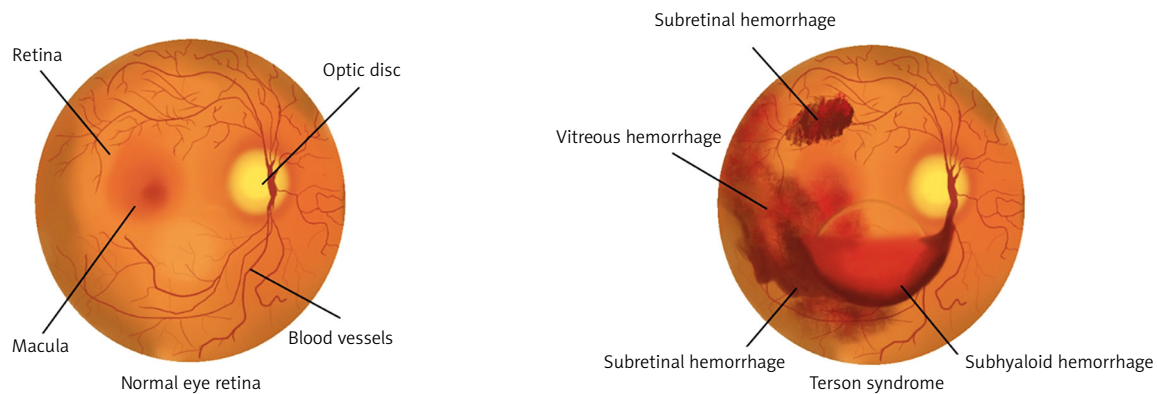


Figure 7. Comparison of fundus images in a healthy patient and a patient with Terson syndrome (own graphics)

high-quality fundal images that are not affected by the postmortem changes of the eyes sclera. As mentioned, after trauma incidents, the ICP increased due to bleeding channeling into the optic nerve sheath. This accumulation of blood causes more congestion in the small optic veins, causing their rupture and intraocular hemorrhages, TS (Figure 7) [42].

In conclusion, although the presence of TS seen during autopsies does not give conclusive information about the cause of death, it broadens the diagnostic scope and expands the techniques available to use during ophthalmoscopic postmortem examinations.

Pediatric infections

RH in infants and young children are considered a strong indication of non-accidental head trauma by numerous medical professionals. However, the selective use of indirect ophthalmoscopy by clinicians in suspected child abuse cases has led to habitual bias and the possibility of misdiagnosing child abuse, as the association between RH and other conditions such as infection, coagulopathy, and accidental trauma may be overlooked. This bias has the potential to cause iatrogenic harm in the form of incorrect diagnoses of child abuse [47].

Causes of RH can include bacterial, viral, fungal, or parasitic infections. Pathogens that cause RH include, but are not limited to *Neisseria meningitidis*, *Streptococcus pneumoniae*, *Staphylococcus* spp., *Escherichia coli*, *Brucella* spp., multi-microbial bacteremia, *Herpesviridae*, *West Nile Virus*, *Candida albicans*, and malaria.

Salvatori *et al.* have reported on 4 cases of young children, all under the age of 3, who had RH due to complications from infections. These RHs were only discovered during autopsy through ophthalmoscopy, as there were no records of clinical fundal examinations. This research suggests that certain ophthalmological signs, which are often associated with abuse, can also appear in cases of fatal pediatric infections. Therefore, it's crucial to maintain a high level of suspicion when investigating child abuse. At the same time,

forensic pathologists need to recognize the possibility of bias in connecting RH solely to non-accidental trauma.

Children with infection and sepsis have fewer than five hemorrhages per fundus as opposed to cranial trauma. The widespread use of indirect ophthalmoscopy can identify the correct cause or conditions that may occur with RH [48].

RH in cerebral malaria (CM)

The diagnostic and prognostic role in CM is played by malarial retinopathy. In pediatric CM, a combination of retinal signs correlates, in fatal cases, with the severity of brain pathology, and has diagnostic and prognostic significance. The diagnosis sometimes remains challenging to establish in fewer imported cases in continental zones.

Ocular fundus photography using direct and indirect ophthalmoscopy as well as fluorescence angiography have shown that in malarial retinopathy there is vitiligo of the retina, caused by a lack of hemoglobin in infected erythrocytes, as well as that obstruction of the vessels includes hemorrhages, orange to white discoloration of the retinal vessels, and edema of the optic disc [49]. Correlations were also shown between the number of RH and cerebral hemorrhages in patients whose death was due to CM. The above measurements made it possible to confirm the hypothesis that fundus examination is suitable for studying the pathophysiology of CM, but also in its prognosis and diagnosis. A big difference can be observed in terms of thromboses, which are often found in the retinal vessels in the brain in malaria, while they are rare in cases other than CM. RH in CM have been observed in 40–60% of cases. Histopathologically, they are similar to the hemorrhagic ring in the brain in CM. Superficial retinal detachment, subretinal hemorrhage, is also observed, usually in the interior and middle layers of the retina, but more often seen in all layers of the retina. In patients with CM, staining of fluid from the periphery of intrareti-



Figure 8. Incision through the pars plana region by creating a sclerotomy using a number 11 scalpel (own graphics)

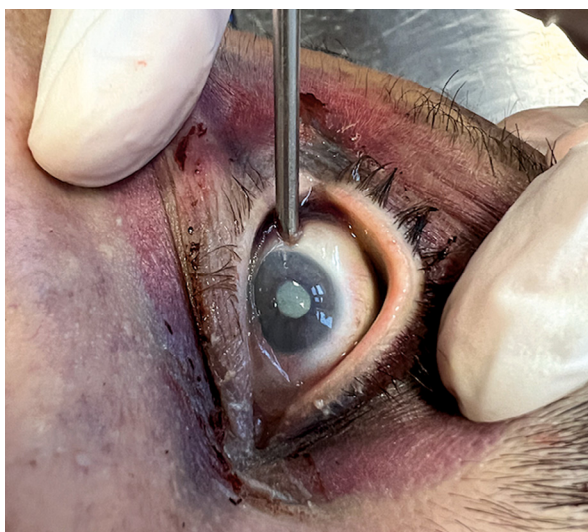


Figure 9. Insertion of the endoscope through the incision (own graphics)

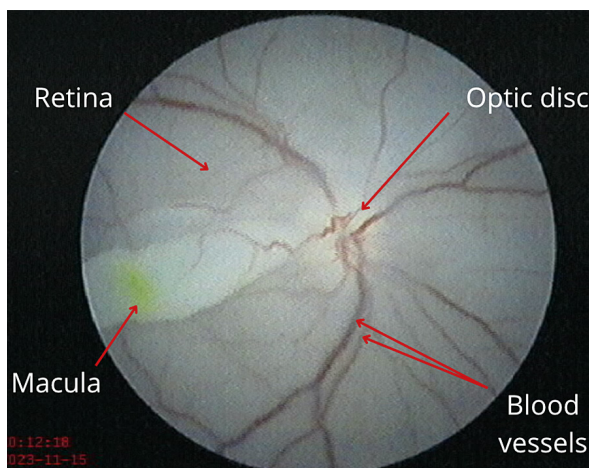


Figure 10. Fundus image with visualization of the optic disc, macula and retina with vessels (own graphics)

nal cystic spaces for fibrinogen and histologic determination of cystoid macular edema (CME) is used. On ophthalmoscopic examination, CME shows the appearance of small cystic intraretinal spaces, due to the accumulation of fluid between the Muller cell and photoreceptor cell, which cover the outer plexiform layers of the macula in the retina. Gliosis, as the only finding, was not different in cases of CM versus non-CM causes, in the retina as well as the optic nerve.

The malar retinopathy described above provides insight into its etiology. The similarity between the retina and the brain hints at the causes of coma as well as death in CM [50].

Retina in AIDS

Oftentimes, eye diseases are commonly noted in individuals with acquired immunodeficiency syndrome (AIDS), particularly during the process of differential diagnosis in suspected cases. Some of the most prominent lesions are spots like “cotton candy” and retinitis caused by cytomegalovirus. Autopsy findings of cytomegalovirus retinitis include ora with serous, hemorrhagic retinal detachments. Part of the posterior vitreous body is indistinct. Isolated Roth stains are present in the uninvolved peripheral retina. Histopathological examination reveals focal acute inflammatory cells. The presence of vortex particles is confirmed by electron microscopy. In some cases, cotton wool spots can be found in both eyes, there are perivascular sheaths of two veins, with one vessel extending to the border of the retinitis pigmentosa. The other, however, extended from the ora to the exposed retina towards the optic nerve [51].

Retinal air embolism (RAE)

RAE is a relatively obscure phenomenon, with limited exposure among clinicians and pathologists to patients or deceased individuals with systemic air embolism (SAE). The symptoms associated with SAE, including RAE, a pallid tongue, skin marbling, and frothy arterial bleeding lack comprehensive understanding. Characteristic indications of RAE encompass intraretinal air columns and bubbles, along with pale silver streaks revealing retinal blood vessels filled with air.

The first case of RAE was described in 1914 by Wever, who drew bubbling, “silvery, shiny rods” of retinal vessels in a man with lung abscesses. The next illustrations depicting the RAE are from 1920, 1957, and 1958. The first was taken after death following sinus irrigation. The next 2 drawings were associated with occipital pneumoencephalography. The 1988 case described was related to a fatal laser bronchoscopic resection of bronchial carcinoma [52].

Bradley *et al.* present a case of an 8-month-old infant. Five hours after death was pronounced, postmortem ophthalmoscopy visualized diffuse RH at the posterior poles and characteristic RAEs. Radiography

visualized subcutaneous emphysema, rib fracture, and intravascular air. An autopsy performed one day after death confirmed cardiac and cerebral intravascular air bubbles. Descriptions of RAE are scarce and lack photographic images of RAE, despite the high diagnostic specificity of SAE. Detailed descriptions depict RAEs as “transparent streaks alternating with dark columns of blood”; “vessels filled with air bubbles”; “pale silver sections”; “innumerable, very fine, shiny lines”; “ (...) as if the arteries had been injected with mercury so that the retina resembled a silver spider web” [52].

In conclusion, RAE remains a mysterious phenomenon, with limited exposure among medical professionals to cases of SAE. The understanding of symptoms associated with SAE, including RAE, is incomplete.

Our experience

We have started examining the fundus of the eye at the Department of Forensic Medicine in Lublin. For the examination we use a rigid endoscope (ShenDa J2900C), with an optical attachment (DE1250 Wireless Endoscope Camera), connecting wirelessly to a tablet (Microsoft Surface Pro) which allows us to save the image. We begin the examination by assessing the external eyeball. We then make an incision through the pars plana region by creating a sclerotomy using a number 11 scalpel (Figures 8–10).

If there is a need to collect vitreous body for laboratory tests, we perform a fundus examination before collecting it. This allows us to avoid artifacts in the form of retinal damage. We have not seen that endoscopic fundus examination affects the results of vitreous body testing for ethanol. After the examination, the equipment is disinfected with a disinfectant solution with ethanol min. 60%. The equipment is then put away to let the excess product evaporate, the endoscope is then wiped with a cloth soaked in water to get rid of any residual ethanol.

During the conduct of the study, we used the assistance of an experienced ophthalmologist in terms of the endoscope insertion methodology. The images obtained were not consulted, this will be the subject of another original paper on the subject.

Conclusions

This article provides a comprehensive overview of the application of postmortem ophthalmoscopy in various forensic contexts. The technique involves the use of a rigid endoscope with a wide-angle view, allowing detailed examination of the ocular fundus after death.

In conclusion, postmortem ophthalmic endoscopy serves as a powerful tool in postmortem examinations. The technique's ability to provide detailed visualization of retinal abnormalities enhances the accuracy of forensic assessments and contributes to

a better understanding of the underlying mechanisms of death in various forensic contexts.

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Ethical approval

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Conflict of interest

The authors declare no conflict of interest.

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