



Incidental Choroid Plexus Papilloma in a Fatal Case of Substance Abuse: A Case Report

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Abstract

Original Research

Background: Choroid plexus papilloma (CPP) is a rare, benign, World Health Organization (WHO) Grade I neuroepithelial neoplasm arising from the choroid plexus. It predominantly affects infants and young children, typically presenting with obstructive hydrocephalus; incidental discovery in an adult, particularly at autopsy, is uncommon. We report a case of an incidental lateral ventricular choroid plexus papilloma discovered at forensic autopsy in a young man whose death was attributed to acute cardiac failure in the setting of substance abuse.

Case Presentation: A 19-year-old male was found dead with no antemortem clinical history available. Forensic autopsy revealed cardiomegaly with biventricular hypertrophy, pulmonary edema and hemorrhage, hepatic fatty change, and congestion of the kidneys and spleen. The brain showed cerebral edema, tonsillar herniation, and uncus grooving, together with a well-circumscribed, greyish-white intraventricular mass within a dilated lateral ventricle. Toxicological analysis of urine detected cocaine and benzodiazepines. Histopathological examination of the intraventricular mass demonstrated a benign papillary neoplasm with fibrovascular cores, foci of calcification, and adjacent normal choroid plexus tissue, confirming a diagnosis of choroid plexus papilloma (WHO Grade I). The certified cause of death was acute biventricular heart failure due to hypertensive heart disease in the setting of substance abuse (cocaine and benzodiazepines).

Conclusion: This case highlights the occasional discovery of clinically silent, benign central nervous system neoplasms at forensic autopsy and underscores the importance of complete, systematic post-mortem examination — including routine sectioning and sampling of the brain — even when a plausible cause of death is otherwise apparent. It also illustrates the well-documented cardiotoxic potential of cocaine, which remains the more direct and immediate contributor to death in this case.

Keywords: Choroid plexus papilloma, incidental finding, forensic autopsy, sudden death, substance abuse, cocaine cardiotoxicity.

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INTRODUCTION

Choroid plexus tumours are rare neuroepithelial neoplasms arising from the choroid plexus epithelium, accounting for less than 1% of all intracranial tumours. They are classified by the 2021 World Health Organization (WHO) Classification of Tumors of the Central Nervous System, 5th edition, into choroid plexus papilloma (CPP, Grade I), atypical choroid plexus papilloma (Grade II), and choroid plexus carcinoma (Grade III) [1,9]. CPP is markedly more common in children, with roughly 70% of cases occurring before two years of age, whereas in adults the tumour accounts for approximately 0.5% of all brain tumours and characteristically arises in the fourth ventricle rather than the lateral ventricles typical of paediatric cases [1,2]. Clinically, CPP classically presents with signs of raised intracranial pressure or obstructive hydrocephalus secondary to cerebrospinal fluid overproduction; however, a proportion of adult cases are detected incidentally, without antecedent neurological symptoms, and diagnosis ultimately rests on a combination of clinical, radiological, and histological findings [3,10].

Sudden, unexpected death due to a previously undiagnosed primary intracranial neoplasm is a rare but recognised phenomenon in forensic pathology practice, reported in approximately 0.02%–2.1% of medico-legal autopsy series [4,5]. In most such cases, the tumour is discovered incidentally and is not the direct cause of death, though it may occasionally contribute through mass effect, herniation, or catecholamine-mediated cardiac injury, a mechanism also described with other intraventricular lesions such as third ventricular colloid cysts [4,5,12]. We report a case in which a lateral ventricular choroid plexus papilloma was discovered as an incidental finding during a forensic autopsy of a young man whose death was attributed to acute cardiac failure in the context of cocaine and benzodiazepine use, a combination with well-established cardiotoxic potential [6-8,11].

CASE PRESENTATION

A 19-year-old African male, weighing 105 kg and measuring 187 cm in height, was found dead with no antemortem clinical history available for review. A forensic (medico-legal) autopsy was performed nine days after death.

External Examination

The body was that of a well-nourished young adult male. There was no pallor, jaundice, cyanosis, or pedal oedema. Brownish crusting was noted around the nose, mouth, and ears. No external abrasions or lacerations were identified.

Cardiovascular Findings

The heart was enlarged, weighing 600 g (normal 250–350 g), with biventricular hypertrophy: the right ventricular free wall measured 1.4 cm (normal 0.3–0.5 cm) and the left ventricular free wall measured 2.0 cm (normal 1.3–1.5 cm). The tricuspid, pulmonary, mitral, and aortic valves were structurally unremarkable. The coronary arteries were patent and free of atherosclerotic plaques, and the aorta showed only fatty streaks.

Respiratory Findings

The lungs were heavy (right 600 g, left 700 g; normal 250–350 g each) with ecchymosis on the parietal pleural surfaces. The right lung showed hemorrhage and congestion on cut section; the left lung showed pulmonary edema. Lung tissue was subcrepitant, and pulmonary vasculature was free of thromboemboli.

Other Systemic Findings

The stomach was hyperaemic with petechial mucosal hemorrhage. The liver (1600 g) showed mild fatty change. Both kidneys were enlarged (200 g and 220 g; normal 120–160 g) with congestion, hemorrhage, and ecchymosis on cut section. The spleen was enlarged (300 g; normal 120–160 g) and congested.

Central Nervous System

The formalin-fixed brain weighed 1600 g (normal 1200–1500 g). There was cerebral oedema with tonsillar herniation and uncal grooving. A well-circumscribed, greyish-white intraventricular mass was identified within a dilated lateral ventricle. The

mass weighed 6 g and measured $3.0 \times 2.0 \times 1.0$ cm, with a solid, greyish-white cut surface. The remainder of the brain, including the cerebral hemispheres, cerebellum, brainstem, and cerebral vasculature, showed no additional structural abnormality.



Figure 1. Coronal section of the fixed brain showing the dilated lateral ventricle at the site of the intraventricular mass.

Toxicological Analysis

Forensic toxicological analysis of urine detected cocaine and benzodiazepines. Gastric content and bile were negative for poisons.

Histopathological Findings

Microscopic examination of the intraventricular mass showed a benign papillary neoplasm composed of numerous branching, finger-like papillary fronds. The papillae were lined by a single layer of uniform

cuboidal to low columnar epithelial cells with bland nuclei and inconspicuous nucleoli. Many papillae contained fibrovascular cores, and some showed cystic dilatation. Foci of calcification were present within the lesion. Adjacent normal choroid plexus

tissue was identified. No cytologic atypia, increased mitotic activity, necrosis, or invasion was seen, consistent with a diagnosis of choroid plexus papilloma, WHO Grade I.

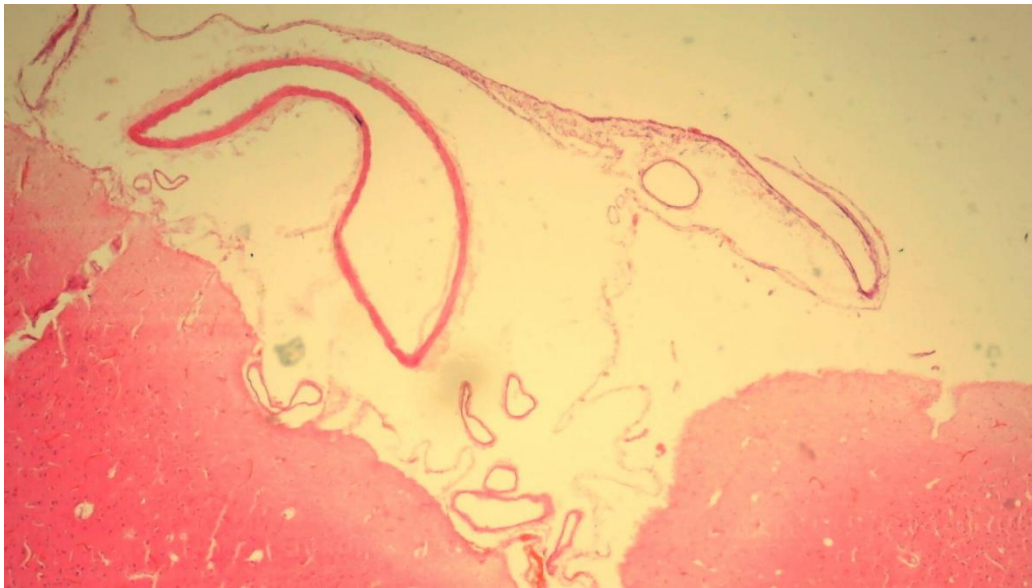


Figure 2. Low-power photomicrograph showing the dilated ventricular lumen with papillary fronds projecting into the cavity (H&E).

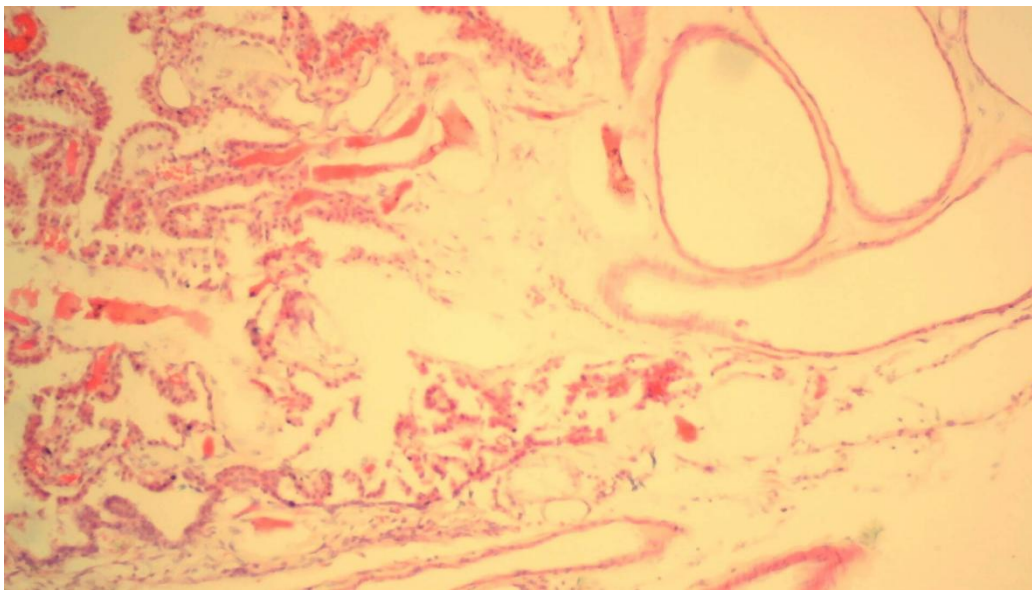


Figure 3. Branching papillary fronds with fibrovascular cores and areas of cystic dilatation (H&E).

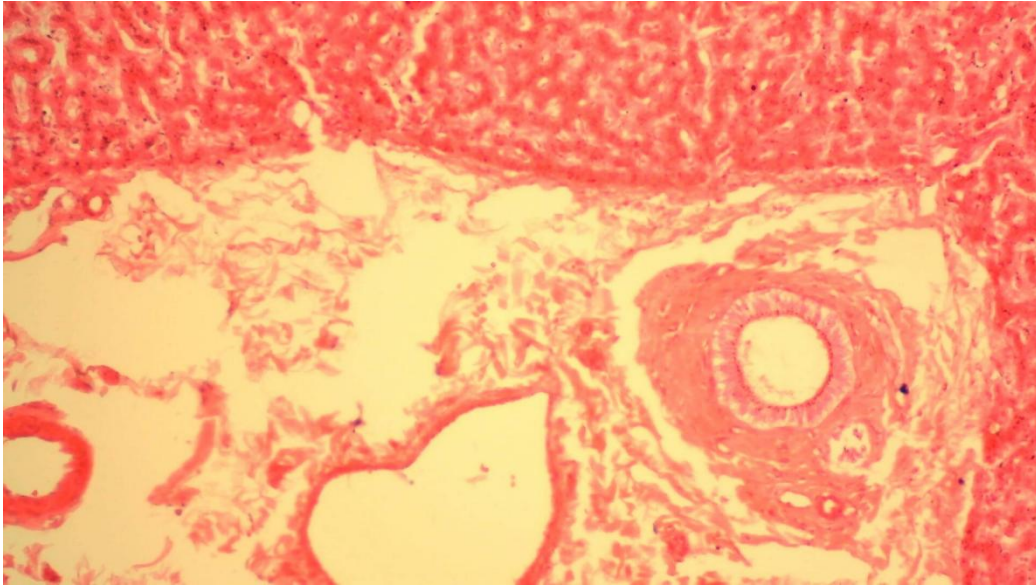


Figure 4. Cross-sectioned papilla demonstrating a fibrovascular core lined by a single layer of cuboidal to low-columnar epithelium (H&E).

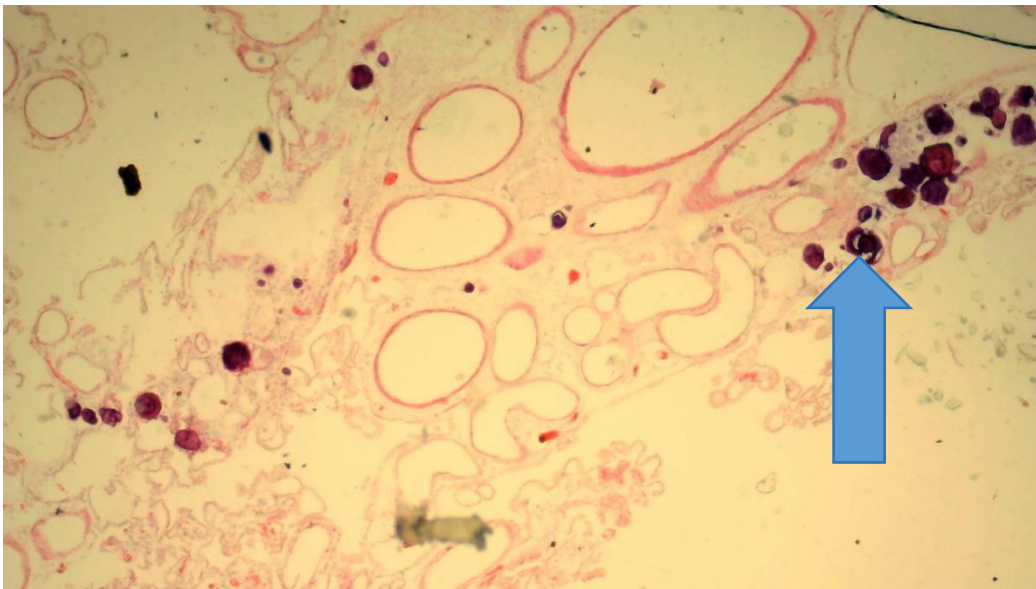


Figure 5. Foci of calcification (arrowed clusters) within the papillary lesion (H&E).

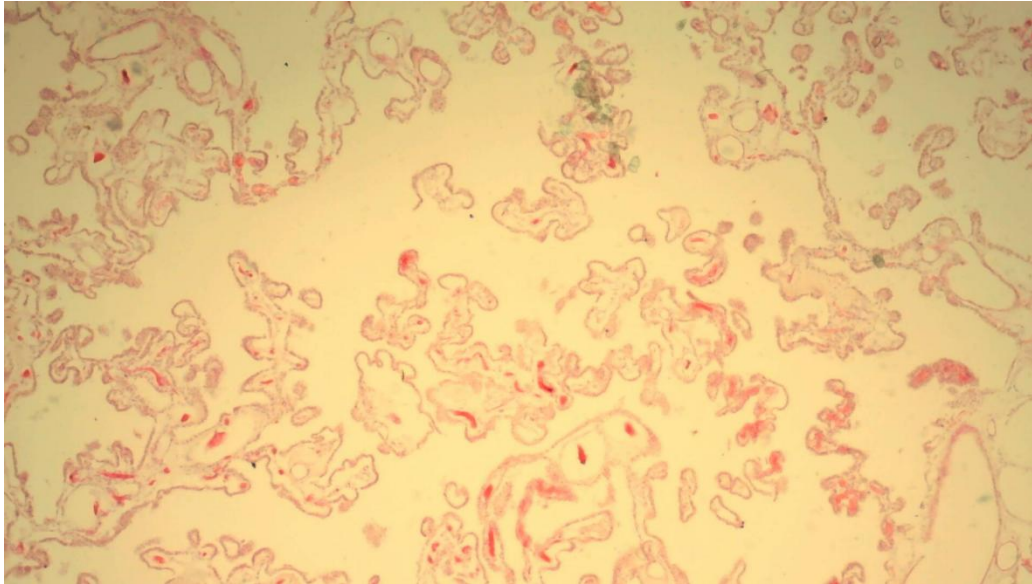


Figure 6. Adjacent normal choroid plexus tissue with typical villous architecture, shown for comparison with the neoplastic component (H&E).

Cause of Death

The cause of death was certified as: 1a. Acute biventricular (heart) failure; 1b. Hypertensive heart disease; 1c. Substance abuse (cocaine and benzodiazepines). The choroid plexus papilloma was considered an incidental finding, unrelated to the immediate cause of death {II}

DISCUSSION

Choroid plexus papilloma is a rare, benign, slow-growing neoplasm that overwhelmingly affects infants and young children, in whom it typically arises in the lateral ventricles and presents with hydrocephalus or raised intracranial pressure [1,2,9]. In adults, CPP is markedly less common and more often located in the fourth ventricle, with clinical onset dominated by signs of intracranial hypertension or cerebellar dysfunction rather than the presentations typical of infancy [2,3]. A recent institutional series of adult choroid plexus tumours found that a minority of cases (approximately one in six) were detected incidentally, without antecedent symptoms [3]. Imaging-based differentiation of CPP from other intraventricular lesions, such as

meningioma or ependymoma, can be challenging and relies on a combination of morphological and perfusion characteristics, underscoring the continued importance of histopathological confirmation, as in this case [10]. The present case is notable in that the tumour was located in the lateral ventricle, the paediatric-typical site, despite occurring in a young adult, and was entirely asymptomatic prior to death, being discovered only at autopsy.

The discovery of a clinically silent central nervous system neoplasm at forensic autopsy, while uncommon, is a well-recognised occurrence. Sudden death directly attributable to an undiagnosed primary intracranial tumor is rare, reported in only 0.02%–2.1% of medico-legal autopsy series, and in most reported instances the tumor is an incidental finding rather than the cause of death [4,5]. In this case, several features argue against the choroid plexus papilloma having played a direct causative role in death: the lesion was small (6 g, 3.0 × 2.0 × 1.0 cm), histologically benign with no evidence of hemorrhage or acute obstruction, and the remainder of the ventricular system was not grossly distorted beyond the lateral ventricle harbouring the mass. The cerebral oedema, tonsillar herniation, and uncal

grooving identified are more plausibly explained as terminal, agonal phenomena secondary to acute cardiac failure and systemic hypoxic-ischaemic insult, rather than as a direct consequence of mass effect from a 6 g papilloma. It is worth noting, by comparison, that other benign intraventricular lesions, most notably third ventricular colloid cysts, have been linked to sudden death through obstructive hydrocephalus or catecholamine-mediated 'neurogenic stunned myocardium' via hypothalamic compression, with histological correlates such as contraction band necrosis in the myocardium [12]. No such mechanism is supported in the present case, given the small size and lateral (rather than third) ventricular location of the tumour, and the absence of any myocardial contraction band necrosis or other histological marker of catecholamine-mediated injury. Nonetheless, some contribution of the lesion to localized ventricular dilatation and CSF dynamics cannot be entirely excluded, and this represents an inherent limitation of a purely post-mortem assessment without antemortem imaging.

The more direct explanation for this death lies in the combination of cardiomegaly with biventricular hypertrophy and the toxicological confirmation of cocaine and benzodiazepine use. Cocaine is well established as a potent cardiotoxin, and cocaine intoxication remains among the most frequent causes of drug-related sudden death identified by medical examiners [6]. In a large prospective forensic series from Spain, cocaine was implicated in around 3% of all sudden deaths, with cardiac causes, including severe left ventricular hypertrophy and coronary atherosclerosis, accounting for the majority of cases; findings that closely mirror the cardiomegaly and biventricular hypertrophy documented in the present case [11]. Its cardiotoxic effects are mediated through two principal mechanisms: sodium channel blockade, producing a local-anaesthetic-like membrane-stabilising effect that slows myocardial conduction and predisposes to arrhythmia, and inhibition of catecholamine reuptake, resulting in increased sympathetic tone, tachycardia, hypertension, and increased myocardial oxygen demand [7,8]. Chronic use is further associated with left ventricular hypertrophy, myocarditis, and dilated cardiomyopathy, which may progress to heart failure

[6,7]. These mechanisms are consistent with the cardiomegaly, biventricular hypertrophy, and pulmonary oedema documented at autopsy in this case, supporting the certified cause of death of acute biventricular heart failure due to hypertensive heart disease in the setting of chronic substance abuse.

From a forensic pathology standpoint, this case reinforces the importance of complete, systematic autopsy examination, including routine sectioning of the brain and, where indicated, targeted histological and immunohistochemical work-up, even when a plausible cause of death is otherwise evident from cardiovascular or toxicological findings [12]. Incidental discovery of unrelated pathology, as in this case, can have implications for accurate death certification, medico-legal documentation, and, in principle, for family counselling regarding incidental findings, even where such findings do not alter the determined cause of death.

CONCLUSION

We report an incidental lateral ventricular choroid plexus papilloma discovered at forensic autopsy in a young man whose death resulted from acute biventricular heart failure related to hypertensive heart disease and substance abuse. This case adds to the limited literature on incidental benign central nervous system tumours identified at medico-legal autopsy and underscores the value of thorough, systematic post-mortem examination in every case, irrespective of the apparent cause of death.

DECLARATIONS

Ethics Approval and Consent to Publish

Written informed consent for publication of this case, including clinical and histopathological images, was obtained from the patient's next of kin. The patient is referred to throughout by non-identifying initials (O.D.) in accordance with journal and institutional requirements for de-identification.

Conflict of Interest

The authors declare no conflict of interest.

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Author Contributions

All authors contributed

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